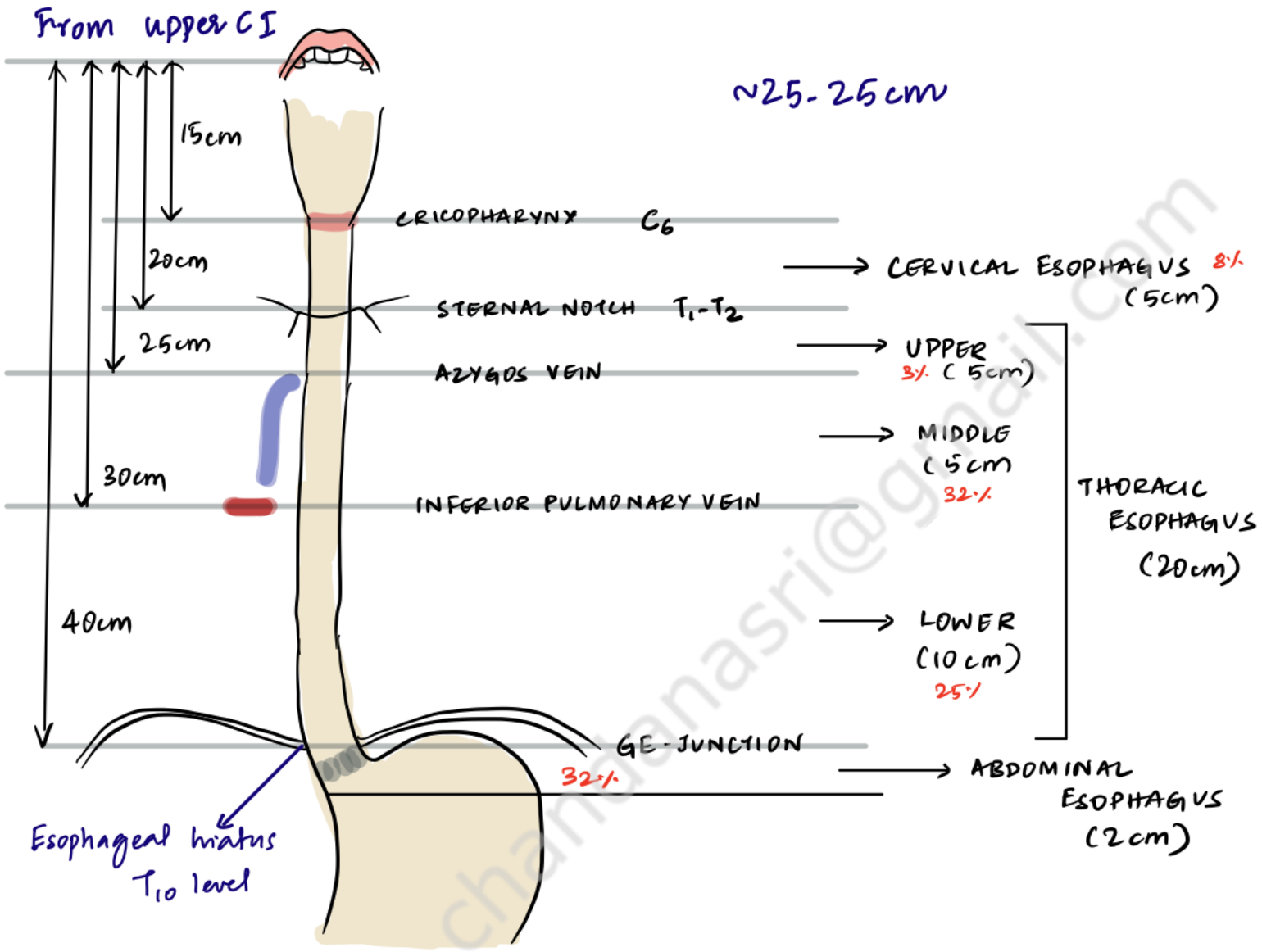
CARCINOMA ESOPHAGUS – SURGICAL ANATOMY



ESOPHAGEAL LYMPHATICS

- Lymphatics situated in SUBMUCOSA of esophagus → dense, interconnected → plexus
  (There are more lymph vessels than blood capillaries in the submucosa)
- LYMPH FLOW → LONGITUDINAL > transverse (6:1) in the submucosal plexus
  - UPPER 2/3rd → CEPHALAD
  - LOWER 1/3rd → CAUDAD
  - → NON-SEGMENTAL LYMPHATIC DRAINAGE (more in the THORACIC ESOPHAGUS)
- Cervical esophagus → lymphatic drainage is more direct & has less submucosal extension → more regionalised lymphatic spread
  - → DEEP CERVICAL PARATRACHEAL
  - UPPER THORACIC → PARATRACHEAL
  - LOWER THORACIC → SUBCARINAL, INF. PULM. LIG. NODES, SUPERIOR GASTRIC NODES

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BLOOD SUPPLY

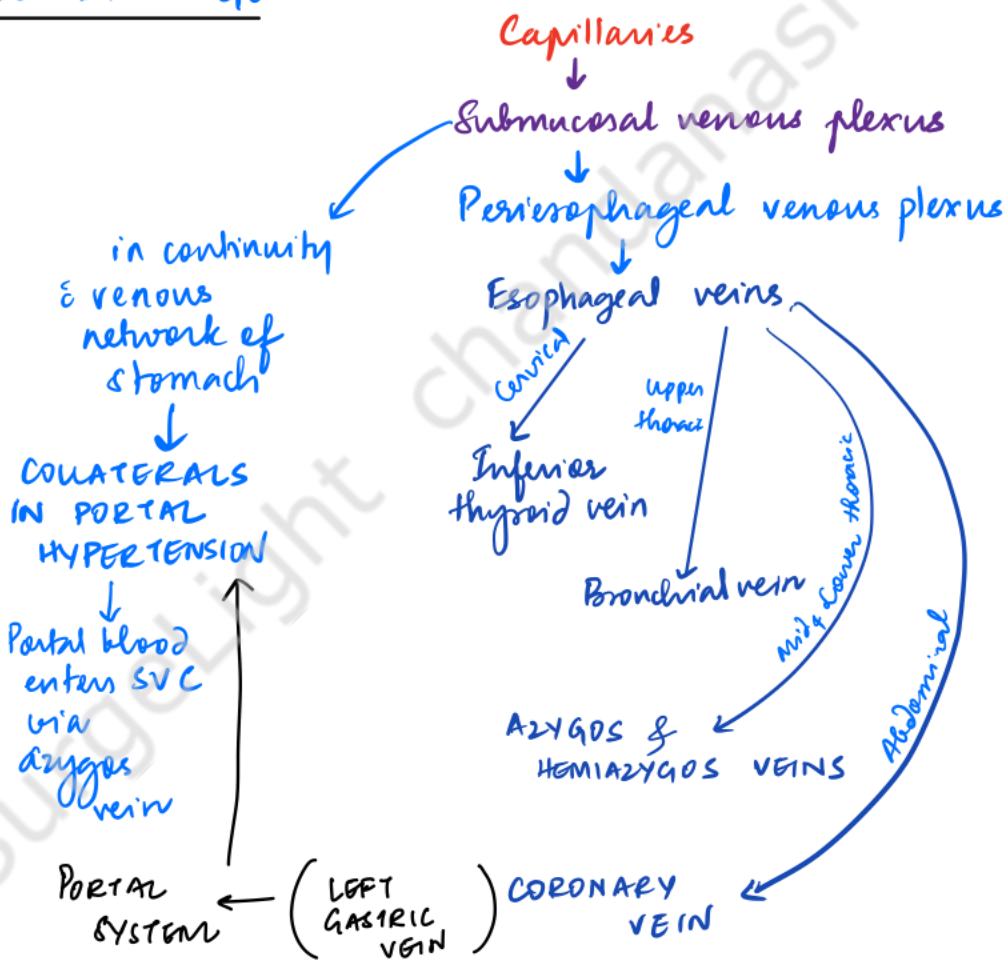
- Upper esophagus → Esophageal branch of inferior thyroid artery
- Thoracic esophagus → Bronchial arteries
  Esophageal branches of aorta
- Abdominal esophagus → Ascending branch of (L) gastric artery
  Inferior phrenic arteries

On entering the wall of the esophagus, they form a longitudinal intramural vascular network in the muscular & submucosal layers

⇓

Esophagus can be mobilized from the level of stomach upto aortic arch, without fear of devascularisation

VENOUS DRAINAGE



NERVE SUPPLY

- Pharyngeal plexus
  RLN → Cricopharyngeal sphincter
  Thoracic segmental nerves

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CARCINOMA ESOPHAGUS

EPIDEMIOLOGY

- 8th m/c cancer
- 6th m/c/c of cancer-death
- Squamous Cell Carcinoma (SCC) 80% > Adenocarcinoma (EAC) ↗ in recent years d/t lifestyle changes obesity 'epidemic'
- SCC - m/c in Esophageal Ca Belt - Henan Province of China, Central Asia, Northern Iran

RISK FACTORS

① SQUAMOUS CELL CARCINOMA

- Smoking
- Alcohol - GENETIC POLYMORPHISM (ALDH-2)
- History of aerodigestive malignancy → field cancerization
- Hot Beverages
- N-nitroso- containing foods
- Betel nut
- Fungal toxin / virus
- Lye corrosive stricture
- H/o Mediastinal radiation
- ✓ Plummer vinson so, Tylosis } Premalignant
- ✓ Achalasia Cardia
- Micronutrient deficiencies - Vit A, C, E, B_1, B_2, Selenium, Mn, Mo, Mg
- Bloom so, Fanconi Anemia

② ADENOCARCINOMA

- BARRETT ESOPHAGUS
- GERD
- Obesity
- H/o mediastinal radiation
- Smoking
- Familial Barrett's Esophagus

PRECURSOR LESIONS

FOR SQUAMOUS CELL CARCINOMA

Squamous Dysplasia

↓

Mild

↓

Moderate

↓

Severe Dysplasia

Diagnosis

- Chromoendoscopy : Lugol's Iodine
  - (N) esophageal squamous epithelium stains BROWN
  - unstained area → abnormal
- Narrow Band Imaging
  - IPCL (Intraepithelial papillary capillary loop)
  - → evidence of early neoplastic neovascularisation

Rx - EMR / ESD / Surgery - based on depth

FOR ADENOCARCINOMA

Barrett's Esophagus

NON DYSPLASTIC INTESTINAL METAPLASIA

↓

LOW GRADE DYSPLASIA

↓

HIGH GRADE DYSPLASIA

↓

ESOPHAGEAL ADENOCARCINOMA

Diagnosis :

- Chromoendoscopy : Acetic Acid
- 4 Quadrant biopsies

Rx - Surveillance for Non Dysplastic
Endoscopic ablative therapy
Surgery

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INVASIVE CANCER

Clinical presentation

- Dysphagia - progressive, Odynophagia
- Regurgitation
- Cough
- Aspiration
- Chest infections
- Hoarseness → tumor infiltration of RLN
- Supraclavicular LN

STAGING

T - T_{is} - HIGH GRADE DYSPLASIA
malignant cells confined to epithelium by BASEMENT MEMBRANE

T_1
- T_{1a} - invades LAMINA PROPRIA, MUSCULARIS MUCOSAE
- T_{1b} - invades SUBMUCOSA

T_2 → invades MUSCULARIS PROPRIA

T_3 → invades ADVENTITIA

T_4
- 4a - Pleura, Pericardium, azygous vein, peritoneum
- 4b - Aorta, vertebral body, airway

N

- N_1 - 1-2 regional nodes
- N_2 - 3-6 regional nodes
- N_3 - ≥7 regional nodes

G - Grade -
- x - can't be assessed
- 1 - well diff
- 2 - mod diff
- 3 - Poorly diff

M M_1 - distant mets (+)

$T_{is}\ N_0\ M_0$ - 0

$T_1\ N_0\ M_0$, $T_1\ N_1\ M_0$ } - I

$T_2, T_3\ N_0, N_1\ M_0$ - II

$T_3\ N_1\ M_0$
$T_{1,2,3}\ N_2\ M_0$ } III

$T_4\ N_{0,1,2}\ M_0$
Any T N_3 M_0 } IV A

Any T Any N M_1 → IV B

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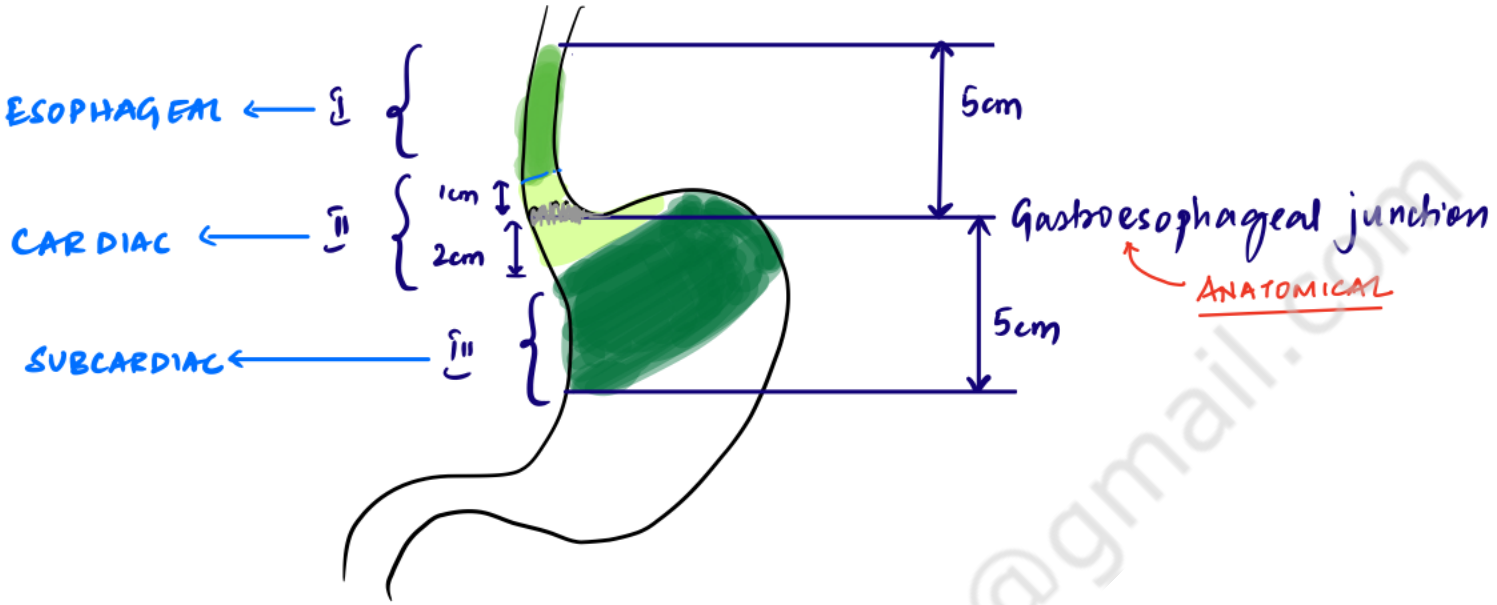
GASTRO-ESOPHAGEAL JUNCTION TUMORS - CLASSIFICATION & STAGING

SIEWERT CLASSIFICATION



Tumors arising from the area upto 5 cm proximal to & 5 cm distal to gastroesophageal junction are termed GEJ tumors - classified as per SIEWERT CLASSIFICATION

AJCC-8 recommendations for staging GEJ tumors

ESOPHAGEAL CANCER - Tumors with epicentre < 2cm into cardia region
GASTRIC CANCER - >2cm into the cardiac region

Siewert I – 1cm proximal to 5cm proximal to GEJ

Siewert II – 1cm proximal to 2cm distal to GEJ

Siewert III – 2cm distal to 5cm distal to GEJ

Siewert I & II → Esophageal adenocarcinoma

Siewert III → Gastric cancer

SurgeLight chandanaraj@gmail.com

STAGING MODALITIES

1. Barium contrast studies - mucosal irregularity
   - shouldering → abrupt step
   - stenotic lumen
   - Dilatation of proximal esophagus
   - tortuosity / angulation / deviation
   - sinus formation
   - fistulation into tracheobronchial tree

2. Bronchoscopy:
   assessment of tumor invasion of bronchial tree
   - widening of carina - nodes
   - extrinsic compression - from posterior tracheal wall
   - Direct tumor infiltration
   - Fistulization

3. CT - M- metastasis - lung, liver, bone etc.
   evaluation of primary - CT < EUS
   T_4 disease
   → obliteration of fat planes, area of contact
   N- suboptimal in detecting mediastinal & abdominal nodal involvement

4. Endoscopic Ultrasound

↑ sensitivity for tumor and nodal evaluation; Guided FNAC of nodes ✓
Limitation
→ in ~1/3rd cases - EUS probe cannot be passed d/t stricture

5. FDG PET

False negatives in small T_1 & T_2
FDG non-avidity in adenocarcinomas of GEJ & proximal stomach
No value in T staging
insufficient spatial resolution to distinguish tumor from juxtatumoral nodes
GOOD specificity for regional nodes

6. THORACOSCOPY & LAPAROSCOPY
   to s/o metastasis before radical surgery

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STAGE-WISE MANAGEMENT OF CARCINOMA ESOPHAGUS

EARLY ESOPHAGEAL CANCER

EARLY SQUAMOUS CELL CANCERS

Japan Esophageal Society classification of T_1

- T_1a
  - M_1: Ep → Carcinoma in situ
  - M_2: LMP → invades lamina propria
  - M_3: MM → invades muscularis mucosae
- T_1b
  - SM_1 → upper 1/3rd submucosa
  - SM_2 → middle 1/3rd submucosa
  - SM_3 → lower 1/3rd submucosa

Management

↓ risk of nodal metastasis
- T_1a — Ep
- T_1a — LMP

In cases i s/o nodal mets
- T_1a-MM
- T_1b-SM_1

→ Endoscopic Submucosal Dissection
> Endoscopic mucosal resection

ESD > EMR

↓

restricted to tumors involving < 2/3rd circumference
(> 2/3rd → EMR → STRICTURE)

SM_2, SM_3 → should be treated as locally advanced Ca Esophagus

EARLY ADENOCARCINOMA & BARRETT'S

Early intramucosal lesions
↓
Endoscopic resection techniques

Submucosal invasion
↓
↑ risk of nodal involvement to 20%.

SURGICAL RESECTION

Vagal sparing minimally invasive esophagectomy
↓
For high grade dysplasia and intramucosal adenocarcinoma

→ Since standard lymphadenectomy is not performed, not suitable for higher risk lesions

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LOCALLY ADVANCED ESOPHAGEAL CANCERS (II, III)

Multimodality approach

SURGICAL RESECTION is the mainstay

PATIENT SELECTION FOR SURGICAL RESECTION

NO STRICT GUIDELINES

DEFER SURGERY I/C/O

1) KPS < 80
2) Poor nutritional status, >10% wt loss
3) Pre-existing cirrhosis & Cardiopulmonary Disease
4) Advanced age >70y

CHOICE OF ESOPHAGEAL SUBSTITUTE → may be osthotopic / Retrosternal / Subcutaneous

1) m/c - gastric conduit → isoperistaltic tube based on greater curvature of stomach & preservation of (R) gastric & (R) gastroepiploics
2) COLONIC INTERPOSITION
3) JEJUNAL CONDUIT - usually after distal esophagectomy + total gastrectomy
   - ROUX-EN-Y CONFIGURATION

CERVICAL ESOPHAGEAL CANCER

Cervical / Cervico-esophageal cancers <5cm from cricopharyngeal sphincter

IN THE SPIRIT OF Laryngeal preservation: DEFINITIVE CHEMORADIATION → ??? SALVAGE SURGERY

- Pharyngolaryngoesophagectomy (PLE) ± ADJUVANT RADIOTHERAPY

APPROACH: Cervical + Abdominal ± thoracic

- Cervical: Enbloc resection of the hypopharyngeal - cervical esophageal tumors + WHOLE LENGTH OF ESOPHAGUS
- Abdominal: GASTRIC TUBE
- thoracic: Not required if transhiatal / MIS dissection can be done

PHARYNGOGASTRIC ANASTOMOSIS + PERMANENT TRACHEOSTOMY

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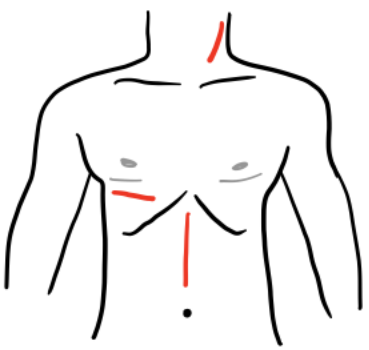
SURGICAL MANAGEMENT OF THORACIC ESOPHAGEAL TUMORS

UPPER THIRD

McKEOWN - 3 PHASE ESOPHAGECTOMY

1. (R) thoracotomy → Esophageal mobilization Lymphadenectomy
2. Midline laparotomy → to mobilize gastric conduit
3. Cervical incision → Cervical anastomosis



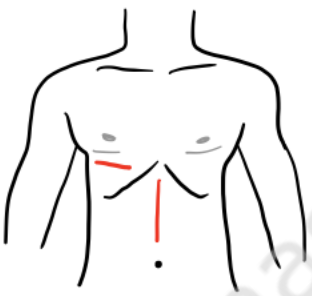
MIS - 3 phase esophagectomy

- Thoracic phase may be done via VATS
- Abdominal phase is done by laparoscopy
- Cervical anastomosis via cervical incision

MIDDLE THIRD

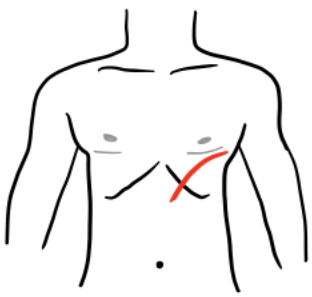
IVOR-LEWIS-TANNER - 2 PHASE

1. Midline laparotomy → to prepare gastric conduit
2. (R) thoracotomy for esophageal mobilization, lymphadenectomy & ANASTOMOSIS (MEDIASTINAL)



ALTERNATIVE APPROACH

- Single (L) thoracotomy / thoracoabdominal approach
- through incision in diaphragm, stomach can be prepared.



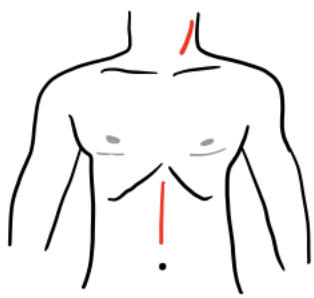
This approach is limited superiorly by arch of aorta & cannot be used for tumors superior to arch of aorta

LOWER THIRD, GEJ

ORRINGER-TRANSHIATAL

1. Midline Laparotomy - Thoracic esophagus is mobilized by blunt & often, blind dissection through enlarged ESOPHAGEAL HIATUS → Stomach is mobilized
2. Cervical incision - Gastric conduit is delivered into neck → Cervical anastomosis

Drawback - does not allow thorough mediastinal lymphadenectomy



MIS - TRANSHIATAL

- Abdominal portion - mobilisation done by minilap / Laparoscopy
- Anastomosis done by cervical incision

MARGINS:

PROXIMAL = 10cm (axial insitu margin)
DISTAL = 5cm → unclear

Aim at negative adventitial margin

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EXTENT OF LYMPHADENECTOMY

→ **3-FIELD LYMPHADENECTOMY-** B/L CERVICAL + COMPLETE MEDIASTINAL + UPPER ABDOMINAL

1. CERVICAL - Boundaries - Superiorly - CRICOID CARTILAGE; Caudally - UPPER MARGIN OF CLAVICLE
   Key nodes to be removed - PARATRACHEAL, PARAESOPHAGEAL

2. MEDIASTINAL
   - STANDARD → Infracarinal lymphadenectomy
   - EXTENDED → Std + superior mediastinal lymphadenectomy around Ⓡ RLN & Ⓡ Paratracheal area
   - COMPLETE → Extended + SUBAORTIC + Ⓛ PARATRACHEAL

3. ABDOMINAL → Superior gastric nodes, Celiac trunk nodes, Common Hepatic nodes

---

→ **2-FIELD** — MEDIASTINAL, ABDOMINAL

HISTOLOGY

- Squamous Cell Carcinoma → LYMPHADENECTOMY HAS TO BE MORE EXTENSIVE (∵ location is more cephalad) → 3 FIELD LYMPHADENECTOMY
- Adenocarcinoma → 2 FIELD LYMPHADENECTOMY

LOCATION OF TUMOR vs need for Cervical LNectomy

Should cervical lymphadenectomy be done or not

overall risk of cervical metastasis 30%

- Upper 1/3rd - 60%
- Middle 1/3rd - 20%
- Lower 1/3rd - 12.5%

→ Weigh morbidity vs risk of mets

RECONSTRUCTION:

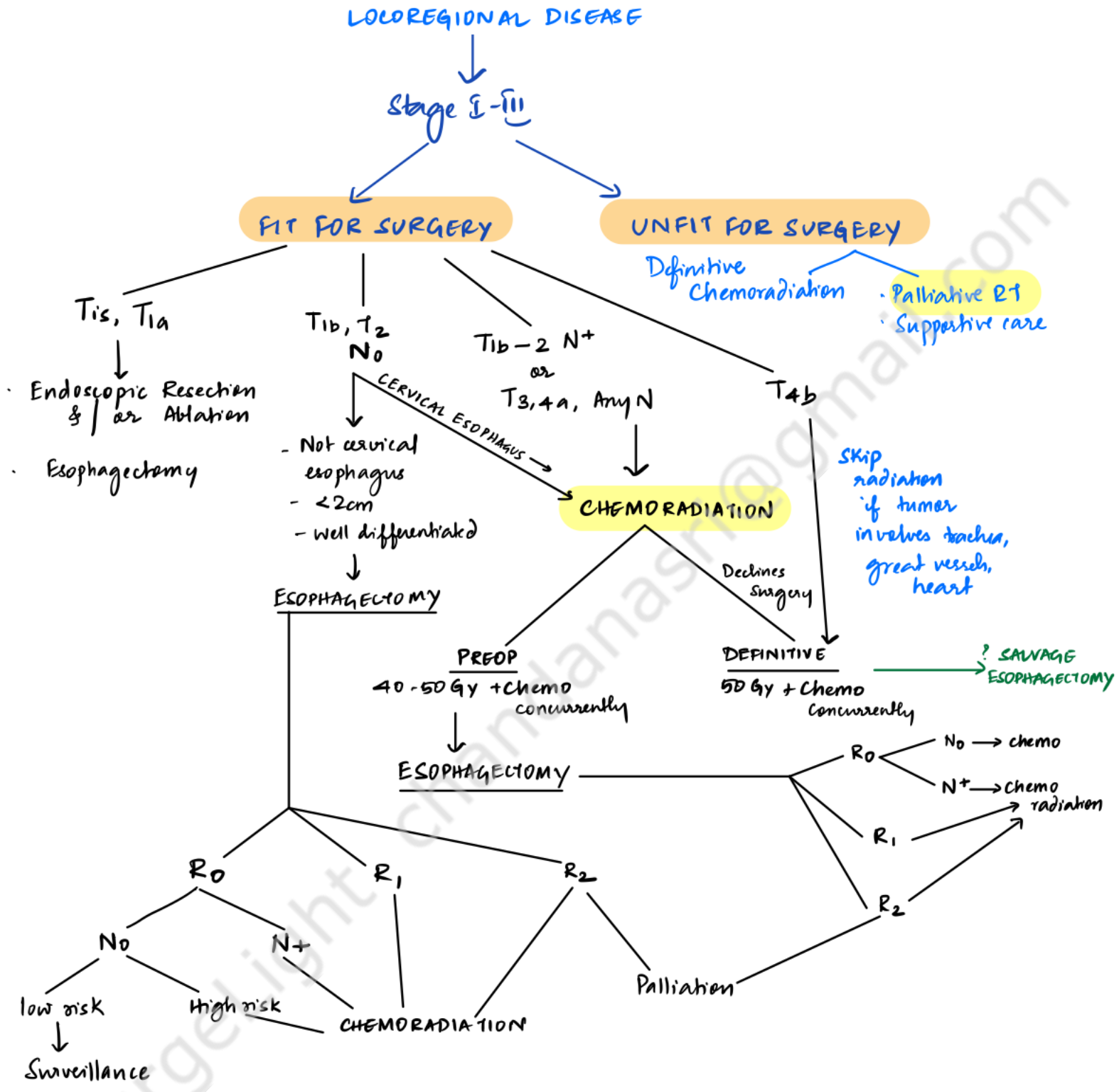
CERVICAL ANASTOMOSIS
- Extensive resection can be done
- Thoracotomy may be avoided
- Less severe reflux
- ↓ complications of anastomotic leak

vs

MEDIASTINAL ANASTOMOSIS
- ↓ incidence of anastomotic leak
- ↓ stricture rate
- ↓ rate of RLN injury

Surgeonchandanasri@gmail.com

MULTIMODALITY TREATMENT STRATEGIES



Regimens

Preop & Adjuvant
- 5FU + Cisplatin / Oxaliplatin
- Carboplatin + Paclitaxel
- Capecitabine + Cisplatin / Oxaliplatin

Metastatic
- Trastuzumab
- Ramucirumab
- Pembrolizumab
- Ipilimumab

Surgeonlight.chandanasri@gmail.com

PALLIATION

1) ENDOSCOPIC - Esophageal stent - SEMS
Laser ablation
Intralesional injection

2) PALLIATIVE - CHEMORADIATION
RT - 45-60Gy over 8 weeks
TARGETTED THERAPY

3) NUTRITION - FEEDING - 2Gr IV Dysphagia
↓
Jejunostomy

4) BLEEDING - EBRT

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Barrett Esophagus

Definition: Change in the mucosa of the esophagus from squamous epithelium to metaplastic columnar epithelium as a result of GERD

PATHOGENESIS:

Exposure of esophageal epithelium to gastric & duodenal fluids

↓

Adaptive metaplasia

↓

columnar cells & Mucous secreting goblet cells

↓

INTESTINAL METAPLASIA

↓

DYSPLASIA

GRADES:

- Non Dysplastic Barrett's Metaplasia
  - progression to esophageal adenocarcinoma 4-6 / 1000 pts
- Low grade Dysplasia
  - progression to esophageal adenocarcinoma 7-8 / 1000 pts
- High grade Dysplasia
  - progression to esophageal adenocarcinoma 14-15 / 1000 pts

- CLASSICAL BARRETT - ≥3cm metaplasia
- Short Segment Barrett - <3cm metaplasia
- Cardiac metaplasia -
  Histological BE without gross endoscopic evidence

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Evaluation:

On endoscopy - tongues of salmon colored mucosa projecting into the distal esophagus from the squamocolumnar junction

Reported as per Prague protocol

C M
- Length of circumferential Barrett's
- Maximum length of total Barrett

ENDOSCOPIC BIOPSY
- Seattle protocol - 4 quadrant biopies every 2cm from SCJ

Narrow band imaging helps visualize Barrett's better than conventional white light

HISTOLOGICAL TYPES OF BARRETT'S

- Intestinal type - Goblet cells
- Cardiac type - Cardiac type mucous glands
- Oxyntocardiac type - Oxyntic + Cardiac glands

TREATMENT

- Rx of Reflux - PPI
- Endoscopic ablation - for Dysplastic Barrett's
  - ✓ RFA
  - ✓ Cryotherapy
- Combining ablation of BE & antireflux surgery
- Endoscopic Mucosal Resection / Endoscopic submucosal dissection → for nodular BE
- Esophagectomy - BE nodules harboring ≥T1b
  - persistent BE & HGD despite multiple ablation attempts
  - Complicated ulcers / strictures / ulcers intractable to conservative management

surgeonlight.chandanasri@gmail.com

CAUSTIC INJURIES OF THE ESOPHAGUS

Damage to the wall of the esophagus, secondary to direct contact with an acid / base

PATHOPHYSIOLOGY

Depends on
- pH ⟶ <2, >12 → more damage
- viscosity
- concentration
- amount ingested
- contact time
- comorbidities

SUBSTANCE

ACID
- Coagulative necrosis
  - ↓
  - PROTECTIVE ESCHAR

ALKALI
- Liquefactive necrosis

(Lye: ↑ conc NaOH / KOH used for cleaning)

INFLAMMATORY RESPONSE

ACUTE NECROTIC PHASE (< 3d)
- Death of cells d/t coagulation of proteins
- Intense inflammatory response
- Vessel thrombosis
- Bacterial & hemorrhagic infiltration of underlying tissue

ULCERATIVE GRANULAR PHASE (3d - 3w)
- Sloughing of superficial necrotic tissue
- Ulceration
- Development of fresh granulation
- Development of new blood vessels
- Infiltration of fibroblasts
- Early collagenous process producing connective tissue

IN THIS PHASE, ESOPHAGUS IS MOST PRONE TO HEMORRHAGE & PERFORATION

ASSESS FOR NECROSIS / DELAYED PERFORATION

CICATRISING & STRICTURING PHASE (3w - 3m)
- Ongoing formation of collagenous connective tissue
- Submucosa & muscularis replaced by dense fibrosis
- Decreasing inflammatory response
- Re-epithelization ± squamous cells

ASSESS FOR STRICTURE
DYSMOTILITY
MALIGNANCY

MANAGEMENT
- Resuscitation
- Assess for necrosis / perforation of esophagus & stomach
- Drain any pleural effusion
- Antibiotics

SurgeLight.chandarsri@gmail.com

EVALUATION

1) History regarding substance
2) Symptoms: Dysphagia
Odynophagia
chest pain
Vomiting
Epigastric pain
3) Examination - Mouth- mucosal injury
Drooling
hoarseness
stridor

IMAGING

- CXR - mediastinitis
- pleural effusion

Esophagogram- controversial

- CT - Grading → in stable pts
  - I - No definite swelling of esophageal wall (<3mm - WNL)
  - II - Edematous wall thickening (>3mm) w/o periesophageal soft tissue infiltration
  - III - Edematous wall thickening
    Periesophageal soft tissue infiltration
    ± well demarcated tissue interface
  - IV - Edematous wall thickening
    Periesophageal soft tissue infiltration
    ± NO demarcated tissue interface
    or
    localised fluid collection around esophagus / aorta

- ENDOSCOPY

Gentle flexible endoscopy

GRADING

0 → (N)

1 → Superficial mucosal edema / erythema

2 → Mucosal & Submucosal ulcerations
- 2a: Superficial erosions exudates
- 2b: Deep discrete circumferential

3 → TRANSMURAL ULCERATIONS
- 3a: Focal necrosis
- 3b: Extensive necrosis

4 → PERFORATION

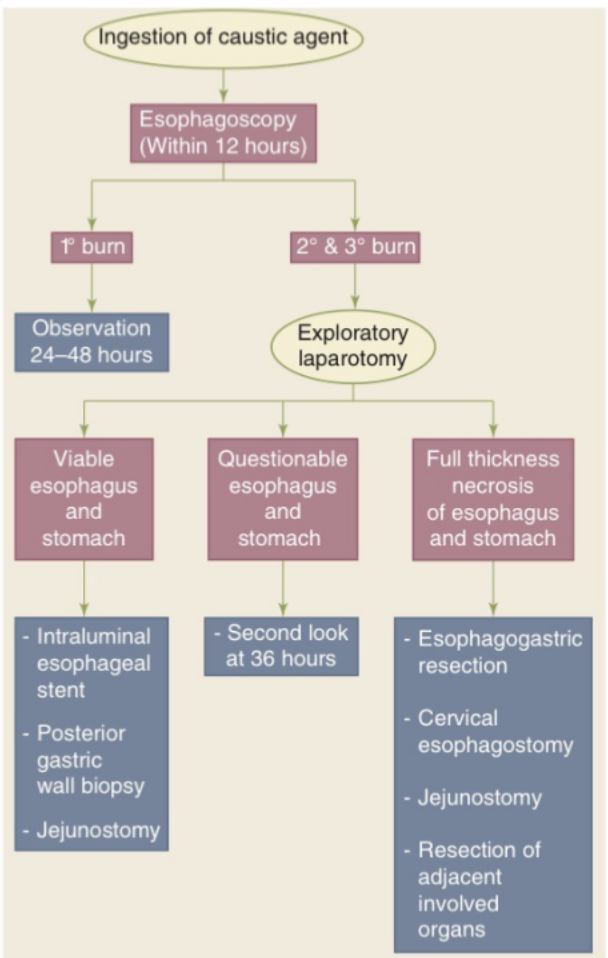

Figure 25-79. Algorithm summarizing the management of acute caustic injury.

Surgeonlight chandanasri@gmail.com

TREATMENT

ACUTE PHASE (<3d)

1) Medical Management
   - Induction of emesis → contraindicated
   - Ingestion of neutralising agents - harmful - exothermic reaction

- Antibiotics
- PPIs
- Steroids – no proven benefits
  – ↑ stricture rates

2) SURGICAL MANAGEMENT

Emergency surgery - Endoscopic 3/4

Damage Control Surgery

Careful assessment of stomach & esophagus
- Removal of necrotic / non-viable tissue
- Hemostasis
- Defer definitive reconstruction

→ Non viable → Esophagectomy & Cervical esophagostomy

NUTRITION
Feeding jejunostomy

INTERMEDIATE PHASE (3d-3w)

Nutritional support - oral / FJ / TPN

Stricture prophylaxis in grade 2-3
? Steroids
? Mitomycin
? Antioxidants

Endoscopy generally avoided in this phase

CHRONIC PHASE

1) Resection / Bypass of strictures → dilatation
   → ↑ risk of malignancy

2) Reconstruction - Gastric conduit (not feasible i/c/o concomitant gastric injury)
   - Colonic interposition
   - Jejunal interposition
   - Pharyngocoloplasty

Long term → Strictures (recurrent), Surveillance for cancer

SurgeLight - chandanasri@gmail.com

ESOPHAGEAL ATRESIA – TRACHEO-ESOPHAGEAL FISTULA

Definition:

Esophageal atresia is a congenital discontinuity resulting in proximal esophageal obstruction

TEF - Abnormal fistulous communication between esophagus & trachea
- can occur alone / in combination

EMBRYOLOGIC BASIS
· During 4th week of gestation
- foregut begins to differentiate into ventral respiratory part and dorsal esophageal part
↓
ventral respiratory part separates from the esophagus by the formation of lateral tracheoesophageal folds which (some say cranial & caudal) fuse in the midline to form TRACHEO-ESOPHAGEAL SEPTUM
↓
Incomplete fusion / abnormal tracheo-esophageal folds → ABNORMAL COMMUNICATION BETWEEN TRACHEA & ESOPHAGUS

· May develop due to disturbances in epithelial proliferation & apoptosis

EPIDEMIOLOGY
1) 1 in 2500–3000 live births
2) M : F : 1·26 : 1
3) Risk of 2nd child c̄ EA/TEF - 2% ⟶ 20% c̄ multiple cases
4) RR in twins 2·26
5) Environmental factors
   - Methimazole in early pregnancy
   - Maternal diabetes
   - Thalidomide exposure
6) Chromosomal anomalies (6-10%)
   - TRISOMY 18
   - TRISOMY 21
7) ASSOCIATIONS

VACTERL -
- Vertebral anomalies - thoracic
- Anorectal anomalies
- Cardiac anomalies
- TE - TEF
- Renal anomalies
- Limb anomalies - Radial dysplasia

- Trisomy 21
- Duodenal / Lower intestinal atresia
- Tracheomalacia

CHARGE -
Coloboma iris, Heart defects, Atresia of choanae, Retarded development, Genital hypoplasia, EA-TEF

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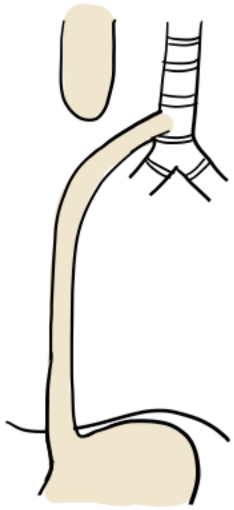
TYPES (A – E) LADD & GROSS
F = Congenital stenosis

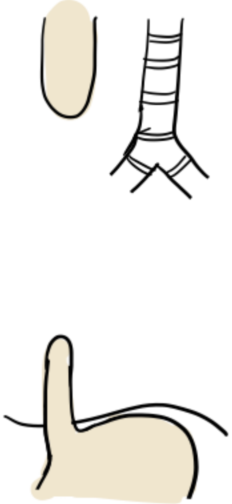
VOGT – I – III c < (II – IIIc = A → D (H fistula not incl); I → aplasia)

EA + Distal TEF



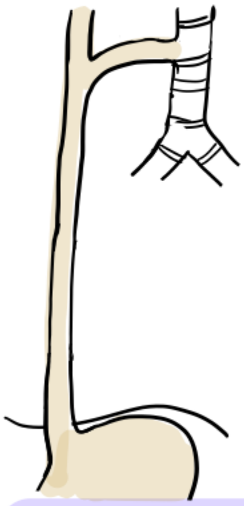
- LADD & GROSS – 'C'
- m/c type (common)
- 85%
- Proximal esophagus
  - dilated
  - thick wall
  - descends into the superior mediastinum upto T_3 or T_4
- Distal esophagus
  - enters trachea posteriorly at carina or 1-2 cm higher
- Distance between esophageal ends is variable

Pure EA



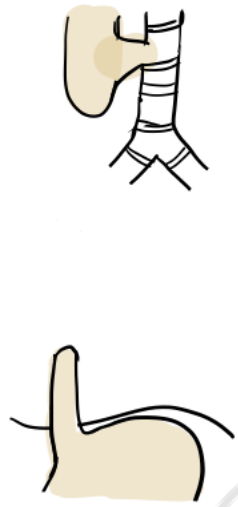
- LADD & GROSS – (A)
- ~7%
- Proximal esophagus
  - ends blindly at the level of azygos vein in posterior mediastinum
- Distal esophagus
  - short stump suspended by fibrous band
- Large distance between esophageal ends

H-type / No EA



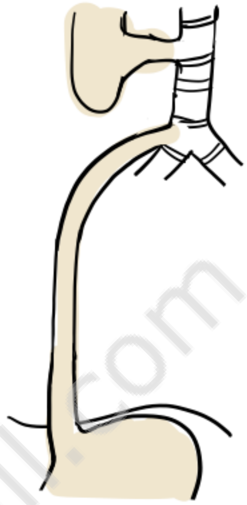
- LADD & GROSS – 'E'
- 4%
- Fistula starts from membranous trachea and runs caudad to esophagus
- Short fistula of variable diameter
- Situated at thoracic inlet / neck

Proximal Fistula



- LADD & GROSS – 'B'
- 2%

Proximal + Distal Fistula



- LADD & GROSS – 'D' (D for Double)
- <1%

EVALUATION

- Antenatal – Polyhydramnios in 2nd half of pregnancy
  - Fluid shifting in the upper pouch
  - Paucity of fluid in stomach & intestine
- Post-natal – drooling of saliva, dyspnea, cyanotic attacks

Air in the bowel below diaphragm ⇒ distal communication; if not – pure atresia / proximal fistula

REPLOGLE tube helps approximate length of upper pouch

Evaluate for other malformations

Used of contrast discouraged – risk of aspiration

surgelight.chandanasri@gmail.com

Management

- Immediate oro/naso-esophageal insertion of Replogle tube
  → continuous/ intermittent aspiration of saliva to prevent aspiration
- Nurse in propped up position
- Intubation and ventilation i/c/o severe respiratory distress
  severe pneumonia
  severe associated malformations

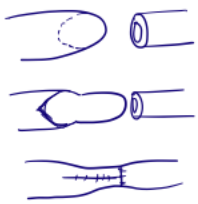
ET-tube must go beyond fistulous opening

SURGERY - After proper resuscitation

Open extrapleural approach is prefered ; Thoracoscopic repair may also be done

(R) Dorso-lateral thoracotomy (i/c/o (N) aortic arch ; (L) thoracotomy for (R) arch)
division of azygous vein
Proximal & distal ends dissected & mobilized
Fistulous tract divided and defect repaired

Attempt Esophago-esophagostomy
↓
Lengthening procedures- circular/spiral myotomy
mucosal-muscular flap



in case of long gap

- proximal pouch may be tagged to prevertebral fascia
- Cervical esophagostomy + gastrostomy
- gastric transposition
- Colonic interposition

Waterston Prognostic Classification

	Birth weight			Survival
I	>1.5 kg		No Cardiac malformation	97%
II	<1.5 kg	(or)	Cardiac malformation	60%
III	<1.5kg	+	Cardiac malformation	22%

Complications

- Aspiration - Pulmonary distress
- Anastomotic leak - mediastinitis

} early

Esophagotracheal fistula
Anastomotic strictures
Tracheomalacia

} Late

Surgeonlightanandanasri@gmail.com

ESOPHAGEAL MOTILITY DISORDERS

CHICAGO CLASSIFICATION OF ESOPHAGEAL MOTILITY DISORDERS

ACHALASIA & ESOPHAGOGASTRIC JUNCTION OUTFLOW OBSTRUCTION

1) TYPE - I / CLASSIC ACHALASIA - impaired LES relaxation, Absent peristalsis, (N) esophageal Pressure

2) TYPE - II / ESOPHAGEAL COMPRESSION
impaired LES relax^n, (N) Peristalsis,
↑ ESOPHAGEAL PRESSURE

3) TYPE - III / SPASTIC ACHALASIA
Impaired LES relaxation
Absent peristalsis
Distal esophageal spastic contractions

4) ESOPHAGOGASTRIC JUNCTION OUTFLOW OBSTRUCTION / ACHALASIA IN EVOLUTION

DISORDERS OF PERISTALSIS

MAJOR
1) ABSENT CONTRACTILITY
2) DIFFUSE ESOPHAGEAL SPASM
3) HYPERCONTRACTILE / JACKHAMMER ESOPHAGUS

MINOR
1) INNEFECTIVE MOTILITY
2) FRAGMENTED PERISTALSIS

MOTILITY DISORDERS (ESOPHAGUS)

PRIMARY
1. ACHALASIA
2. DES
3. HYPERCONTRACTILE ESOPHAGUS
4. HYPERTENSIVE LES
5. INEFFECTIVE ESOPHAGEAL MOTILITY

SECONDARY
- underlying collagen vascular / Neuromuscular dz
  - Scleroderma / SLE
  - Dermatomyositis
  - Chagas Ds
  - Myaesthenia
- Endocrine / Metastatic

• MOTILITY DISORDERS OF PHARYNY / UPPER ESOPHAGUS → TRANSIT DYSPHAGIA

- Disorders of pharyngeal phase of swallowing - DISCO-ORDINATION
- Causes 1) Inadequate oropharyngeal bolus transport
  2) Inability to ↑ Pharyngeal pressure
  3) Inability to elevate larynx
  4) Discoordination between PHARYNGEAL CONTRACTION & CRICOPHARYNGEAL RELAXATION
  5) ↓ compliance of pharyngo-esophageal segment

CONGENITAL

ACQUIRED
- CVA
- Brain stem tumors
- Polio
- Multiple sclerosis
- Pseudobulbar palsy

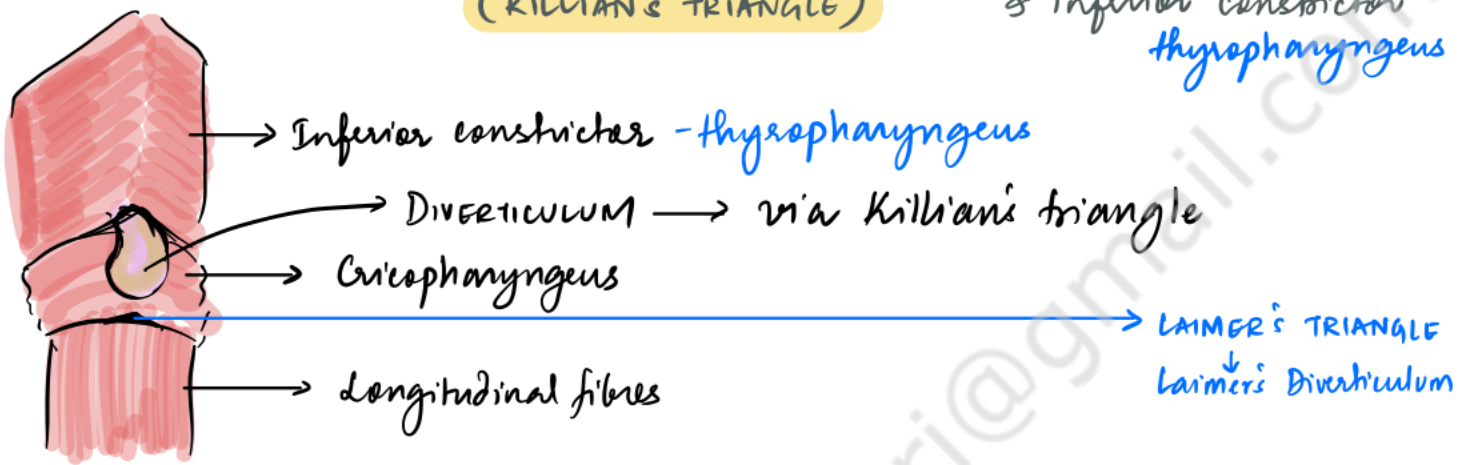
OFTEN ASSOCIATED ∃ ZENKER'S DIVERTICULUM
(Outcome of Cricopharyngeal dysfunction)

Sunbright.chandanasri@gmail.com

ZENKER'S DIVERTICULUM

- Pulsion diverticulum → result of ↑ intraluminal pressure
  ↓
  Herniation of mucosa & submucosa only (FALSE DIVERTICULUM)
  through muscular layer
  ↓
  in the gap at the transition of cricopharyngeus & inferior constrictor thyropharyngeus
  (KILLIAN'S TRIANGLE)

- m/c on left side
- D/t UES dysfunction



CLINICAL FEATURES

1) Cervical Dysphagia / Choking / Odynophagia
2) Halitosis
3) Regurgitation
4) BOYCE SIGN - Neck mass gurgling on palpation

INVESTIGATIONS

1) Barium esophagogram - size, location, neck, approach
2) UGI scopy - s/o other causes
3) Manometry - characterising underlying motility disorder

MANAGEMENT

- CRICOPHARYNGEAL MYOTOMY (CPM) ± DIVERTICULECTOMY ± DIVERTICULOPEXY (to prevertebral fascia) ± DIVERTICULAR INVERSION
- DIVERTICULECTOMY / PEXY / INVERSION ALONE
  TRANSCERVICAL / TRANSORAL ENDOSCOPIC
- DIVERTICULOTOMY + CPM VIA TRANSORAL ENDOSCOPIC ROUTE
  (Division of septum b/w pharynx & diverticulum)
  → Cautery
  CO_2 Laser
  Linear stapler
  Harmonic scalpel

SurgeLight chandanasri@gmail.com

ACHALASIA CARDIA

"failure to relax" (syn. Cardiospasm)

EPIDEMIOLOGY - 1 in 1,00,000, best understood motility disorder

PATHOPHYSIOLOGY

functional loss of Myenteric plexus ganglion cells of distal esophagus & LES

- ? Autoimmune
- ? Chagas
- ? Idiopathic

↓ Loss of inhibitory signals
↓ unopposed excitatatory signals
↓ INABILITY OF LES TO RELAX
↓ ESOPHAGEAL DILATATION
↓ LOSS OF PROGRESSIVE PERISTALSIS

CLINICAL FEATURES

- CLASSICAL TRIAD : DYSPHAGIA : solids → liquids (MAINGOT) / liquids → solids (SABISTON)
  REGURGITATION : of undigested, foulsmelling food
  WEIGHT LOSS
- Chest pain
- Epigastric pain / Heart burn
- Respiratory symptoms - d/t chronic aspiration
  ↓ d/t failure of clearance of food & liquid from esophagus
  ↓ Pneumonia
  Lung abscess
  Bronchiectasis

EVALUATION

1) Upper GI endoscopy : as a part of dysphagia evaluation -

IN ACHALASIA - Mucosal biopsy must be taken to r/o malignancy - 8% risk of SCC

'PSEUDOACHALASIA'
Inflammatory ring
GERD
Eosinophilic Esophagitis
Esophageal Ca

ACHALASIA - dilated tortuous esophagus, residue in esophagus, difficulty to negotiate LES

2) Contrast Esophagogram
- Dilated esophagus & distal narrowing (BIRD BEAK)
- Delayed emptying
- Massive dilatation, tortuousity - SIGMOID/MEGA ESOPHAGUS

I <4cm
II 4-6cm
III >6cm
} dilatation
IV - Sigmoid

SurgeEightchandanasri@gmail.com

3) ESOPHAGEAL MANOMETRY - GOLD STANDARD

5 classic signs of Achalasia < 2 LES, 3 Body

1) Hypertensive LES
   [>35 mm Hg pressure; Integrated Relaxation Pressure >15 mm Hg]
2) Failure to relax i deglutition
3) Body of esophagus: Pressure >> Baseline (Pressurisation) - d/t incomplete air evacuation
4) Simultaneous mirrored contractions i no e/o progressive peristalsis
5) Low amplitude waveforms → Lack of muscle tone

3 manometrical types
- Type I - ⊖ LES relaxation, ⊖ peristalsis, Ⓝ esophageal pressure
- Type II - ⊖ LES relaxation, ⊖ peristalsis, ↑ esophageal pressure
- Type III - ⊖ LES relaxation, ⊖ peristalsis, Distal esophageal spastic contractions

Respond better to therapy → Type II

→ Other investigations
- Endoscopic ultrasound
- Timed barium esophagogram to document contrast bolus retention

MANAGEMENT STRATEGIES

MEDICAL
1) Least invasive, but also
2) Least effective

- Oral CCBs
  Nifedipine 10-30mg
  30-45min before meals
- Oral Nitrates
  Isosorbide dinitrate
  5-10mg
  10-15min before meals
- PDE-5 inhibitors
  Sildenafil

BOTULINUM TOXIN
Under endoscopic guidance,
↓
Botox injected into LES
↓
Blockade of A-Ch release
↓
Relaxation

Effects usually last < 6 months

PNEUMATIC DILATATION
Non compliant cylindrical balloon (3, 3.5, 4cm)
↓
dilate LES & tear its muscle fibres (15-60s)
↓
Effects last upto 2 years

RISK OF ESOPHAGEAL PERFORATION

SURGICAL
- Transthoracic - Division of circular & longitudinal fibres - myotomy - not done now
- TRANSABDOMINAL / HELLER'S MYOTOMY ± FUNDOPLICATION (OPEN, LAP)
  - Gastroesophageal lig divided
  - Esophagus identified
  - Both longitudinal & circular fibres divided - 4-6cm proximal (along esophagus) 2cm distal (onto body of stomach)
- POEM - Per-oral endoscopic myotomy
  small cut in esophageal mucosa 14cm proximal to GEJ
  ↓
  Submucosal plane entered
  tunnel along esophagus & body of stomach
  ↓
  MYOTOMY
  ↓
  Mucosal closure

Surgeonlight.chandanasri@gmail.com

DIFFUSE ESOPHAGEAL SPASM

- un-co-ordinated & simultaneous contractions of (N) AMPLITUDE within the esophagus (m|c-distal)
  → cannot propel food effectively into stomach ↓
  - DYSPHAGIA
  - REGURGITATION
  - CHEST PAIN

CORKSCREW PATTERN ON CONTRAST

HRM - (N) Relaxation of LES
≥ 20% premature contractions

Pathophysiology - impairment of inhibitory innervation
- dysfunction of endogenous NO synthesis in esophagus

℞ - CCBs
Nitrates
TCAs

No role of pneumatic dilatation

HRM directed Botox
POEM

HYPERCONTRACTILE ESOPHAGUS

- Normal, sequential contractions of ABNORMALLY HIGH AMPLITUDE & DURATION
  ↓
  Hypertensive peristalsis

Chest pain > Dysphagia

NUTCRACKER | JACKHAMMER | CUPERSQUEEZER

ROSARY BEAD PATTERN ON CONTRAST

HRM
> 180-200 mmHg contractions
± poorly relaxing LES

May overlap : Achalasia Cardia

℞ - CCBs
Nitrates
TCAs
POEM

SurgeLight chandanasri@gmail.com

GASTRO-ESOPHAGEAL REFLUX DISEASE AND HIATUS HERNIA

Definition: retrograde flow of gastric contents into esophagus, leading to symptoms/ complications

PATHOPHYSIOLOGY: Failure of endogenous antireflux mechanisms

ENDOGENOUS ANTIREFLUX MECHANISMS:
1) Lower Esophageal Sphincter (LES) mechanism
2) Spontaneous esophageal clearance
3) Efficiently functioning gastric reservoir

1) LES mechanism: LES is made of (4) anatomic structures

(1) INTRINSIC MUSCULATURE OF DISTAL ESOPHAGUS
- It is in a state of TONIC CONTRACTION
- Relaxes within 500 milli seconds of initiating of a swallows to allow passage of food into stomach
- Returns to the state of TONIC CONTRACTION

(2) SLING FIBRES OF GASTRIC CARDIA
- oriented diagonally from cardia-fundus junction to LC of stomach
- same anatomic depth as esophageal circular fibres
- contribute to LES tone

(3) DIAPHRAGMATIC CRURA – Pinch valve mechanism
- Inspiration → ↓ intrathoracic pressure w.r.t. intra-abdominal pressure
→ prevent reflux via ↓ in AP diameter of crural opening
↓
↑ LES pressure

(4) INTRA-ABDOMINAL LOCATION OF GEJ
- ↑ IAP → transmitted to GEJ → ↑ pressure at LES → prevents reflux

DETERMINANTS OF LES ANTIREFLUX FUNCTION
- RESTING PRESSURE
- LENGTH OF LES
- INTRA-ABDOMINAL LENGTH OF ESOPHAGUS

GERD develops when INTRAGASTRIC PRESSURE exceeds the high-pressure zone of distal esophagus

HYPOTENSIVE LES → Low resting pressure of LES
- Hiatus hernia

SPONTANEOUS LES RELAXATION → Relaxation of the LES in the absence of esophageal peristalsis

DEFECTIVE LES – Resting pressure < 6 mmHg
Overall sphincter length < 2cm
Intra-abdominal length < 1cm → m/c cause of GERD

2) Spontaneous clearance mechanism – to restore (N) esophageal pH
- ↑ swallowing frequency
- Secondary peristalsis

} Impaired in ↓ ESOPHAGEAL BODY FUNCTION

3) Gastric reservoir – Gastric distension → unfolding of LES → REFLUX

SurgeLight chandanasri@gmail.com

CLINICAL PRESENTATION

1) Heartburn - m/c - 80%.
- epigastric / retrosternal caustic / stinging sensation
NOT A/i PRESSURE SENSATION
DOES NOT RADIATE TO BACK

2) Regurgitation - 54%.
'Water brash'
- movement of gastric contents into oropharynx (acid/bile) (not undigested food)
- made worse by bending

3) Abdominal pain (30%)
4) Dysphagia (20%) - for solids
5) EXTRA-ESOPHAGEAL SYMPTOMS

DE-MEESTER SCORING
- Scores 3 symptoms
1) HEART BURN
2) REGURGITATION
3) DYSPHAGIA
0 - None
1 - Mild
2 - Moderate
3 - Severe

Factors predisposing to GERD
1) Obesity
2) Alcohol
3) Smoking
4) Stress
5) Infancy
6) Pregnancy

Laryngeal
- Hoarseness, Dysphonia
- Throat clearing, Throat pain
- Globus
- choking
- Laryngospasm
- Contact Ulcers
- Laryngeal/ tracheal stenosis

Pulmonary
- Cough
- Shortness of Breath
- Wheeze
- ? Association c IPF

MECHANISMS:
- Proximal esophageal reflux → micro-aspiration of gastroduodenal contents
↓
DIRECT CAUSTIC INJURY TO LARYNX
LOWER RESPIRATORY TRACT
- Reflux → Vagal nerve reflex → BRONCHOSPASM & COUGH
(common vagal innervation of trachea & esophagus)

Antireflux surgery is associated with improvement in respiratory symptoms in ~ 70-90% cases

PHYSICAL EXAMINATION should be done to r/o other causes for symptoms

COMPLICATIONS OF GERD:
1) Erosive Esophagitis
2) Stricture
3) Barrett's Metaplasia - 40x ↑ risk of adenocarcinoma
short seg <3cm
long segment - ≥3cm

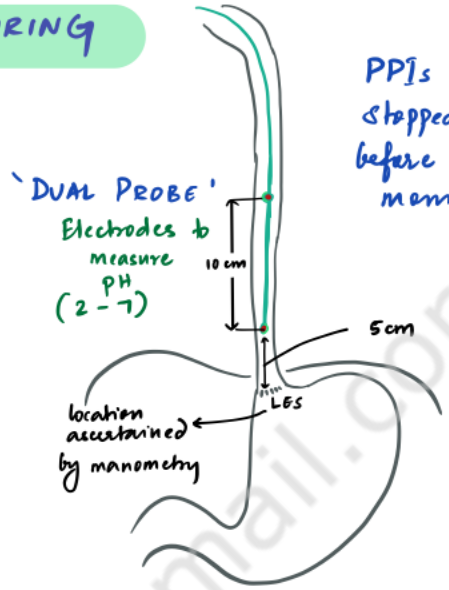
surgeryright.chandanasri@gmail.com

EVALUATION

1) AMBULATORY pH AND IMPEDANCE MONITORING

AMBULATORY pH

- to quantify distal esophageal acid exposure
- GOLD STD



PPIs must be stopped 1 week before 24h pH monitoring

Parameters measured over 24h

1) total number of reflux episodes (pH < 4)
2) longest duration of reflux
3) Number of episodes lasting > 5min
4) % time spent in reflux in · upright · supine

COMPOSITE SCORE (WEIGHTED) → DEMEESTER SCORE

↓

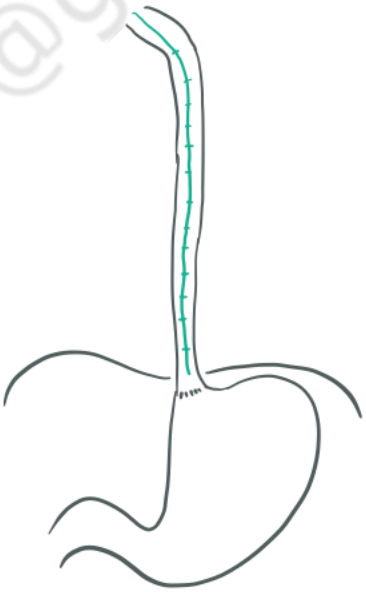
≥ 14.7 → abnormal

ESOPHAGEAL IMPEDANCE MONITORING

- identifies episodes of non-acid reflux
- electrodes are placed at 1cm intervals to detect changes in resistance to flow of current (IMPEDANCE)

↑ in the presence of air

↓ in the presence of liquid / bolus (irrespective of pH)



pH-impedance catheters

↓

can differentiate between swallow & reflux

2) ESOPHAGEAL MANOMETRY

- To assess - function of esophageal body, LES

HRM - High resolution manometry

↓

32-channel flexible catheter & pressure-sensing devices at 1cm intervals

study - 15 min
10 swallows

COLOUR-CONTOUR PLOT & time in x-axis & esophageal length in y-axis

↳ PRESSURE REPRESENTED BY COLOR SCALE (see fig 42-6 on pg 1048 - Sabiston 20e)

measure
- LES resting pressure
- Length of LES

detect dysmotility

3) ESOPHAGO-GASTRO-DUODENOSCOPY

↓

Assessment of GEJ flap valve (Hill's classification - Fig 23-3 in Maingot 13e)

- Barrett esophagus
- Esophagitis - ulcers, erosions
- Strictures
- Hiatus hernia

4) BARIUM ESOPHAGOGRAM (Positional reflux)

- Hiatus hernia, diverticulae, tumors, strictures, dysmotility etc

SurgeLight chandanasri@gmail.com

MANAGEMENT OF GERD

Medical Management
- 8 week course of PPI after ruling out other issues
- Lifestyle advice

SURGERY
- INDICATIONS
  - Objectively proven reflux disease
  - Symptoms despite adequate medical management
  - Younger pt unwilling for lifelong drugs
  - Structurally abnormal LES
  - Severe endoscopic esophagitis ē structurally defective LES
  - Stricture d/t reflux
  - Barrett's esophagus

PROCEDURES

Principle - creation of a new antireflux valve at GE Junction while preserving the ability to swallow normally

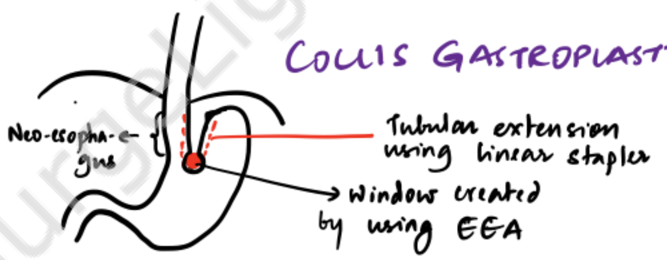
- Create a flap valve - which results in ↑ LES pressure
- Place an adequate length of the distal esophagus in the positive - pressure environment of the abdomen
- The reconstructed cardia should relax on deglutition
  - only fundus of the stomach should be used to buttress sphincter (because it is known to relax in concert ē LES)
  - fundal wrap should only be limited to lower esophagus
  - Vagus should be preserved.
- The fundoplication should not exceed the the peristaltic power of esophagus

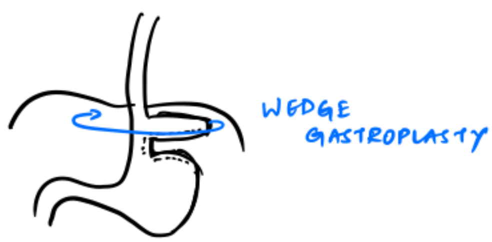
PROCEDURE - FUNDOPLICATION

Key steps:

1) HIATAL DISSECTION & PRESERVATION OF BOTH VAGI
2) CIRCUMFERENTIAL ESOPHAGEAL MOBILISATION & FUNDAL MOBILISATION (± ligation of short gastrics)

COLLIS GASTROPLASTY





- Collis Gastroplasty can be done if enough length of intra-abd esophagus cannot be obtained

3) HIATAL CLOSURE - usually posterior to the esophagus
4) CREATION OF A SHORT & FLOPPY FUNDOPLICATION OVER AN ESOPHAGEAL DILATOR
   - ~2cm of floppy fundoplication
     - OPEN
     - LAP

SurgeLight chandana@gmail.com

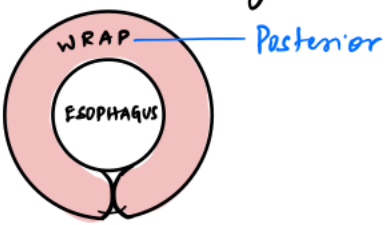
TYPES OF FUNDOPLICATION

COMPLETE (360°)

1) NISSEN'S FUNDOPLICATION

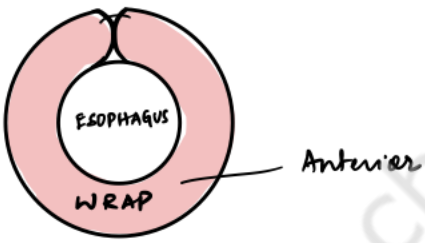
Total posterior fundoplication

- Hiatal dissection
- Esophageal mobilisation circumferentially ~ 3cm of intra-abd esophagus



2) ROSETTI-HALL FUNDOPLICATION

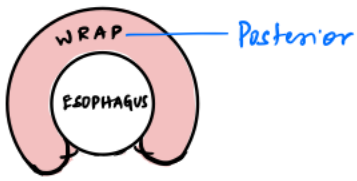
360° - anterior wrap



PARTIAL

POSTERIOR

1) TOUPET FUNDOPLICATION (270°)

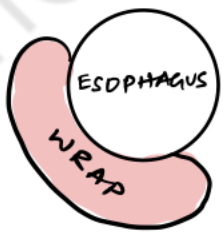


ANTERIOR

1) 90°
2) 120° (DOR)
3) 180°

ANTEROLATERAL

WATSON'S - 120°



OPERATIVE COMPLICATIONS

1) Pneumothorax - pleural violation
2) Gastric & esophageal injuries
3) Splenic & Liver injuries

Outcomes following fundoplication

- > 90% → improvement
- Temporary dysphagia
- Temporary inability to belch, ↑ flatulence (GAS BLOAT SO)
- Failure

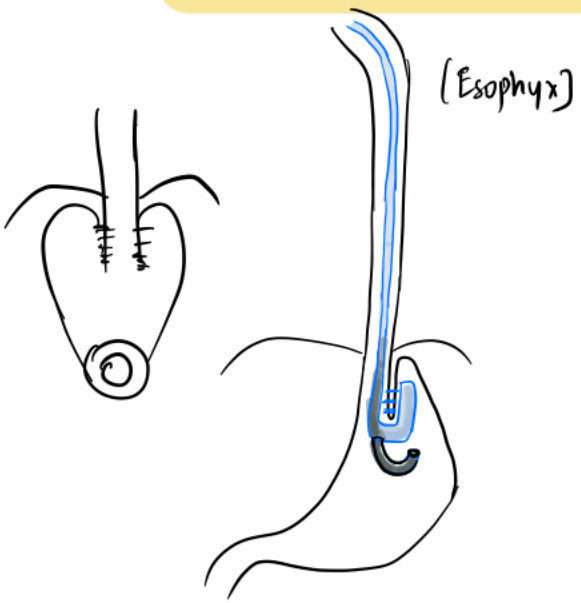
SurgeLight chandanasri@gmail.com

NOVEL THERAPIES FOR GERD

AUGMENTATION OF LES

- RADIOFREQUENCY ENERGY → tissue reaction → Augmentation
- INJECTION OF INERT BIOPOLYMERS
- ENDOLUMINAL GASTROPLICATION
- IMPLANTATION OF MAGNETIC SPHINCTER AUGMENTATION DEVICE (MSAD)

TRANSORAL INCISIONLESS FUNDOPLICATION



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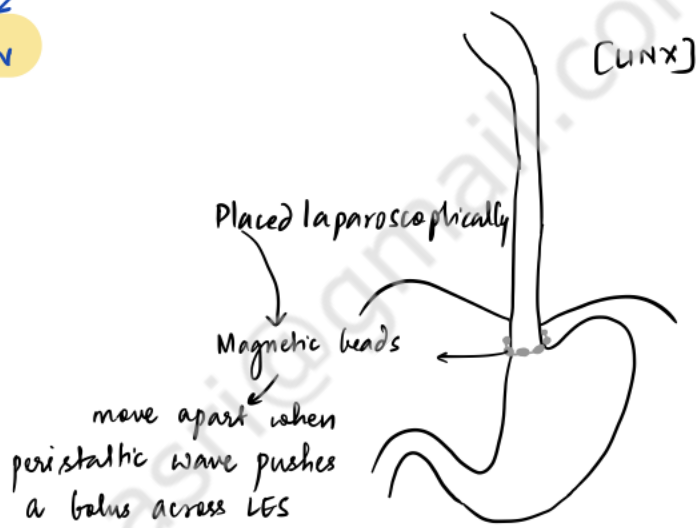


Similar: PLACEMENT OF ANGELCHIK PROSTHESIS- GEL BAND AROUND GEJ

Other procedures (OLDER PROCEDURES)

1) BELSEY MARK IV :

Through (L) posterolateral thoracotomy & transpleural approach - esophagus is identified and delivered
↓
Hiatal dissection
↓
Circumferential incision of phreno-esophageal membrane
↓
Mobilisation of fundus - ligating BELSEY'S ARTERY (communication between (L) gastric A & inferior phrenic A)
↓
PARTIAL FUNDOPLICATION & / OVR GASTROPLASTY (240°)
FUNDOPLICATION IS PLICATED TO THE ABDOMINAL ASPECT OF DIAPHRAGM
HIATUS IS CLOSED
THORACOTOMY CLOSED

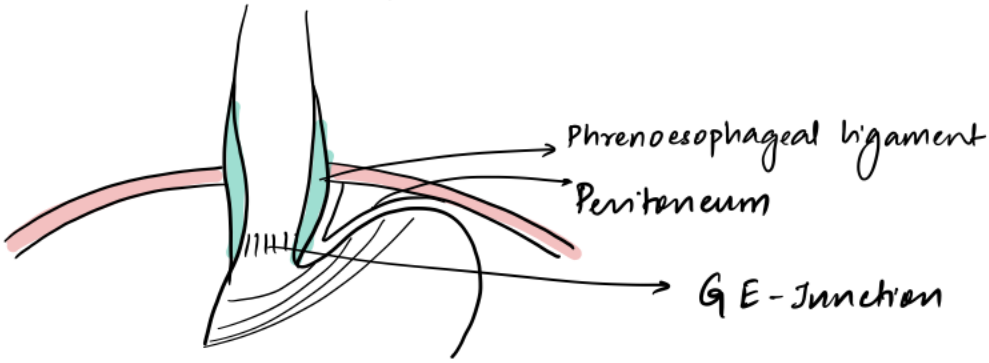
2) HILL'S OPERATION: fixing GEJ to median arcuate ligament

3) THAL PATCH – for esophageal stricture

4) ALLISON – transthoracic repair of sliding hernia & GERD – not done

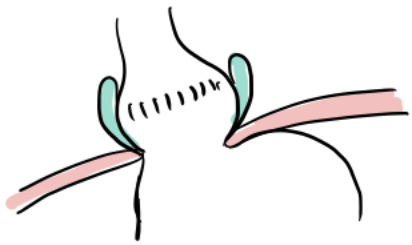
Surgeonlightchandanasingh@gmail.com

PARAESOPHAGEAL HERNIA



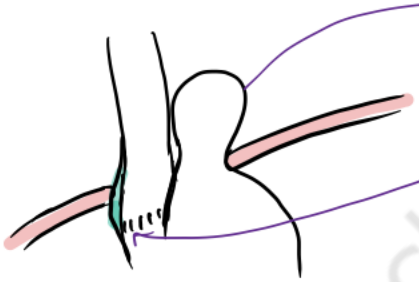
HIATUS HERNIA

TYPE-1 : SLIDING HERNIA – GEJ migrates cephalad into posterior mediastinum



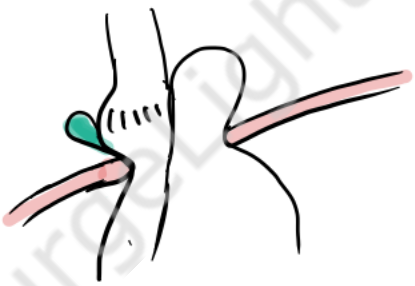
- Due to laxity of phreno-esophageal membrane
- M/c a/i GERD
- Saint's triad [Hiatus Hernia, GERD, Gallstones]

TYPE-2 : ROLLING HERNIA – PARAESOPHAGEAL HERNIA



- Gastric fundus migrates into posterior mediastinum via hiatal defect
- GEJ is anchored intra-abdominally

TYPE-3 : SLIDING + ROLLING HERNIA



- Both GEJ & gastric fundus are located in posterior mediastinum

TYPE-4 : Any visceral structure (colon/spleen/pancreas/small bowel) migrates cephalad to the esophageal hiatus into the mediastinum

→ True Paraesophageal hernias

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PRESENTATION

Reflux
Heart burn
Dysphagia
Post prandial bloating
Nausea
Vomiting
Respiratory Compromise

EVALUATION → Similar to GERD

COMPLICATIONS

1) CHRONIC ANEMIA
↓
d/t Cameron's erosions / ulcers in gastric folds
↓
2° to repetitive movement through the hiatus
↓
where the stomach slides over hiatal crura
↓
gastric mucosal ischemia
↓
Resolves after PEH repair

2) GASTRIC VOLVULUS

Abnormal rotation of stomach >180° along one of 2 axes

BORCHADT's TRIAD → 70% of adult acute organoaxial volvulus
1) Epigastric pain
2) Non-productive retching
3) Inability to pass gastric tube

- INCARCERATION
- CLOSED LOOP OBSTRUCTION
- STRANGULATION

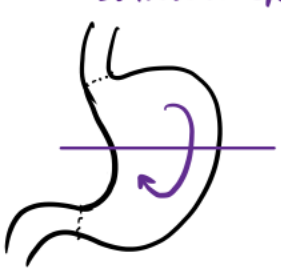
Can be passed in mesoaxial volvulus as GEJ is open

IMAGINARY LINE CONNECTING GEJ & PYLORUS

ORGANO-AXIAL (Long axis)
- m/c - 2/3rd cases
- A/i PEH
- usually complete
- ↑ risk of strangulation

Mixed

IMAGINARY LINE BETWEEN GC & LC



MESO-AXIAL (Short axis)
- <1/3rd cases
- Not a/i PEH
- Usually partial
- ↓ risk of strangulation

SurgeLight chandanasr@gmail.com

BASED ON ETIOLOGY

GASTRIC VOLVULUS

Primary

- 30% cases
- Due to laxity / disruption of the stomach's ligamentous attachments
- Occurs spontaneously without any other intra-abdominal / diaphragmatic pathology
- usually meso-axial
- presents in children
- Chronic / intermittent
- may be a/w congenital asplenia / wandering spleen

Secondary

70% - m/c

2° to another anatomical abnormality

↓

Diaphragmatic defects

↓

Predispose the rotated stomach to lie in the thoracic cavity

Adults

Paraesophageal hernia

children

- Diaphragmatic eventeration
- CDH

usually organo-axial

↓

INTRATHORACIC 'UPSIDE DOWN' stomach

can also occur due to abdominal bands, adhesions & gastric tumors, after sleeve gastrectomy, LGB, adult LDLT (in such cases, it is intra-abdominal)

MANAGEMENT

Surgical emergency - Goals - Reduction, Fixation, repair of predisposing factors

- OPOLZER OPERATION - Partial Gastrectomy, GJ, Fundoantral gastrogastrostomy
- TANNER GASTROPEXY i Colonic displacement
- GREY'S GHIMENTON GASTROPEXY
- Open anterior gastropexy

SurgeLight chandanasri@gmail.com

SURGERY - for Para-esophageal hernia

Principles of Repair

1) Identify hernia (sac) — 2 layers: Peritoneum, Phrenoesophageal ligament
2) Do not make any attempt to reduce contents immediately
3) Dissect the sac off the mediastinum all around
   ↓
   Reduction of contents
4) Repair of hiatal defect c/ ± mesh — Biological / Prosthetic
5) Correction of esophageal foreshortening by Colli's gastroplasty
6) Antireflux procedure - Nissen / Toupet
7) ± Gastropexy

SurgeLight chandanasri@gmail.com

ESOPHAGEAL PERFORATION

CAUSES

IATROGENIC
- During diagnostic/therapeutic procedures
- m/c cause

SPONTANEOUS → BOERHAAVE SYNDROME
- 15% cases
- highest mortality (d/t Dx, Rx delay)
- occurs into (L) pleural cavity or
- just above GEJ
- associated c GERD & sliding hernias

Pathogenesis → Acute ↑ in intra-abdominal pressure against a closed glottis (VOMITING)

FOREIGN BODY → 14%

TRAUMA → 10%

MALLORY-WEISS → mucosal tear → bleeding
→ Occurs in rolling hernias – longitudinal fissures in mucosa of herniated stomach & GEJ

EVALUATION

→ Pain
→ h/o forceful vomiting
→ CXR – Mediastinal emphysema ≥ 1 hr
Mediastinal widening 2° to edema
Cervical emphysema – air along erector spinae
Pneumothorax
→ Contrast esophagogram → extravasation

Mx – Conservative
- Sengstaken Blakemore tube
- Endoscopic epinephrine injection
↓
Fails
↓
Sx
↓
Oversewing

Management

- contained perf – conservative – TPN, Abx, PPI
- Good outcomes c 1° closure within 24 hr
- (L) thoracotomy → identify → trim, close, reinforce c pleural/fundal patch
- >24h – edema/friable
mobilize – end cervical esophagostomy → Reconstruction later
feeding jejunostomy

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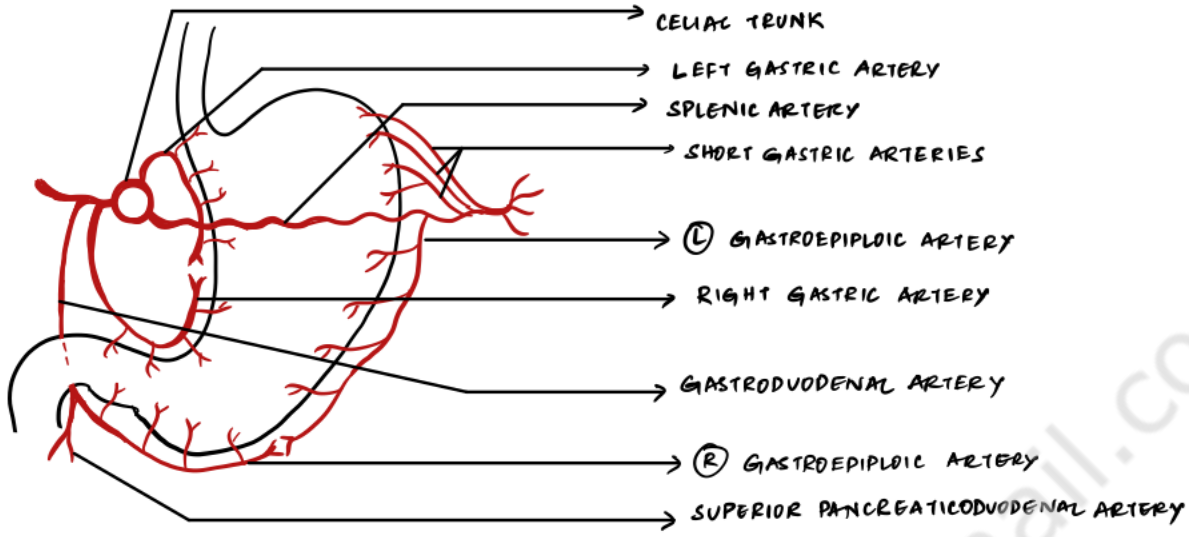
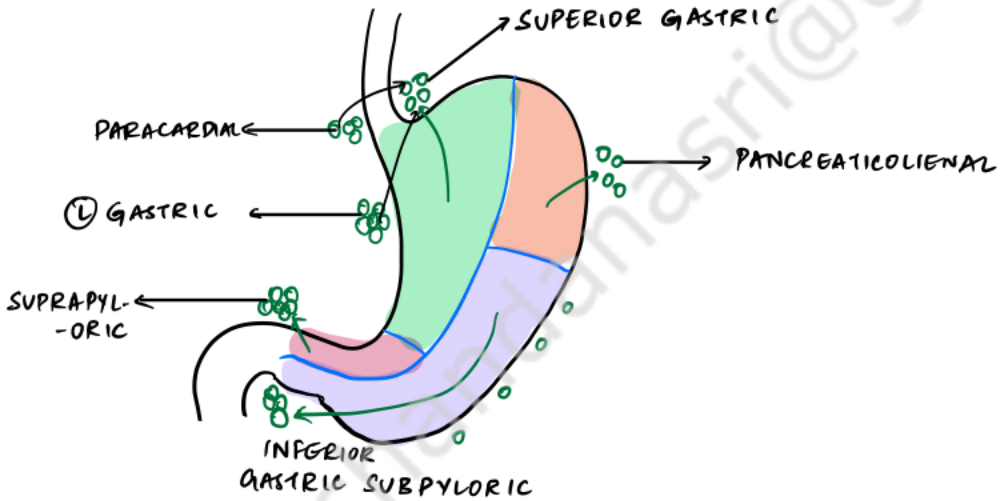
SURGICAL ANATOMY OF STOMACH

BLOOD SUPPLY



LYMPHATIC DRAINAGE



LYMPHNODE STATIONS

1. (R) Cardiac
2. (L) Cardiac
3. Lesser Curvature
4. Greater Curvature
   - 4sa - Short Gastric
   - 4sb - (L) Gastroepiploic
   - 4d - (R) Gastroepiploic
5. Suprapyloric
6. Infrapyloric
7. (L) Gastric artery
8. Common hepatic artery
   - (a) Anterior
   - (b) Posterior
9. Celiac artery
10. Splenic hilum
11. Splenic Artery
    - 11p - proximal
    - 11d distal
12. Hepatoduodenal
    - a - Left
    - b - Posterior
13. Retropancreatic
14. Root of mesentery
15. Middle Colic artery
16. Para-aortic
110. Para-esophageal
111. Diaphragmatic

Extent of lymphadenectomy for various tumor locations

	D_1	D_2
Upper $1/3^{rd}$	1, 2, 3, 4	5, 6, 7, 8, 9, 10, 11, 110
Middle $1/3^{rd}$	1, 3, 4, 5, 6	2, 7, 8, 9, 10, 11
Lower $1/3^{rd}$	3, 4, 5, 6	1, 7, 8, 9

surgelight chandanasri@gmail.com

CARCINOMA STOMACH

EPIDEMIOLOGY

- 3rd leading cause of cancer death worldwide
- >65y
- Asia > Europe, North America

RISK FACTORS

1. Helicobacter pylori
2. Dietary - salt, smoked foods, poorly preserved foods
   nitrates, nitrites, secondary amines
   Vitamin A, C, Calcium, antioxidants → protective
   LACK OF REFRIGERATION
3. Smoking
4. Alcohol
5. Radiation exposure
6. Previous gastric surgery - Gastric remnant cancer (>10y postop, Bile reflux)
7. Coal and rubber workers
8. Epstein Barr virus
9. Family Hx; HEREDITARY GASTRIC CANCER SYNDROMES

   suggestive features: GC in ≥2 1° / 2° relatives
   Cancers in multiple generations
   Signet cell histology
   Early age of onset (<45y)

   1) Hereditary diffuse gastric cancer - CDH-1 mutation
      ↓
      encodes E-cadherin
      a/i premenopausal lobular breast cancer
      role of prophylactic total gastrectomy in CDH-1 mutation carriers
   2) Lynch Syndrome
   3) Li fraumeni Syndrome
   4) Familial adenomatous polyposis
   5) Peutz Jegher's Syndrome
   6) Juvenile polyposis syndrome
   7) MUTYH associated polyposis
   8) HBOC syndrome
10. Pernicious anemia
11. ? Type A blood
12. Precursor lesions
    1) Adenoma
    2) Atrophic Gastritis
    3) Menetrier's disease
    4) Dysplasia
    5) Intestinal Metaplasia

sushrut.chandanasri@gmail.com

PREMALIGNANT LESIONS OF STOMACH

① ADENOMA / ADENOMATOUS GASTRIC POLYPS

- Risk of carcinoma ~10-20%.

↑ risk if POLYP is
- proportional to size of polyp
- >2cm
- sessile

Mucosal atypia is frequent

progression from dysplasia to carcinoma in situ has been observed

GASTRIC POLYPS

FUNDIC GLAND POLYPS	HYPERPLASTIC POLYPS	ADENOMATOUS POLYPS	HAMARTOMAS
Commonly seen in pt on CHRONIC PPI Typically multiple Seen in proximal stomach - Suspect polyposis S° if s/i gastroduodenal adenoma in young patient - High grade dysplasia possible i polyposis S° - Stop PPI if >20 Fundic polyps >1cm fundic polyp - Total Polypectomy if >1cm - ulcerated - antral location	- occur in the setting of ATROPHIC GASTRITIS d/t - H. pylori Autoimmune Bile gastritis - Upto 20% have focus of dysplasia - Upto 2% have cancer (>2cm) - Remove all hyperplastic polyps >0.5cm Repeat EGD 6 monthly - Periodic EGD surveillance required for extensive or advanced atrophic gastritis	- Solitary - Sessile - Distal Stomach - Associated with Atrophic gastritis Gastric metaplasia / Dysplasia - Definite cancer risk - IN POLYP - in stomach ℞ - REMOVE ALL ADENOMATOUS POLYPS + DO REMOTE gastric biopsies ↓ follow up i EGD surveillance	in Peutz Jeghers S° PTEN Hamartom S° Pt i PJ syndrome are at increased risk of gastric cancer (30% Lifetime risk) ↓ No evidence that removal of hamartomas decreases risk ↓ But, carcinoma arising from hamartoma has been described

② ATROPHIC GASTRITIS

Loss of gastric glands along i chief cells & parietal cells

m/c cause - Helicobacter pylori (Corporal distribution; PUD - antrum)

Others - Autoimmune (pernicious anemia)
- Chemical irritation (Bile reflux)

CORREA (Chronic atrophic gastritis)
- Autoimmune — Proximal stomach
- Hypersecretory — Distal stomach
- Environmental - Junction of oxyntic & antral mucosa

ATROPHIC GASTRITIS MAY PROGRESS TO 1) Dysplastic Atrophic Gastritis
2) Metaplastic Atrophic Gastritis

OLGA / OLGIM } Staging & severity of atrophic gastritis } → Based on histology of 5 gastric biopsies ← GC, LC, Corpus, Incisura

Surgeonlightanand@gmail.com

③ MENETRIER'S DISEASE / HYPERTROPHIC GASTRITIS

- Hypoproteinemic Hypertrophic gastropathy
- Rare, acquired, premalignant condition

Massive gastric folds in fundus and corpus of stomach

(Mucosa - Cobblestone / Cerebriform appearance)

↓

HPE - Foveolar hyperplasia / expansion of surface mucous cells
Absent parietal cells

- Excessive mucus production → Protein loss
  - Quantified by chromium 24 labelled albumin test
  - ? Cytomegalovirus (children)
  - ? H. pylori
- Decreased acid production → Hypochlorhydria / Achlorhydria
  - 24hr pH monitoring

↑ TGF-α in gastric mucosa

Management

Biopsy to rule out gastric carcinoma / lymphoma

Rx - anticholinergics, Octreotide, Anti-H. pylori } Inconsistent results

Total Gastrectomy < in carcinoma
if protein loss is massive despite Rx

④ INTESTINAL METAPLASIA

Gastric carcinoma occurs in the areas of intestinal metaplasia
→ risk is proportional to the extent of metaplasia

SUBTYPES

- Complete - Glands are lined by goblet cells & intestinal absorptive cells
- Incomplete - Resembles colonic epithelium
  - irregular mucin droplets
  - Absence of brush border

Type I : Mature
II : Different levels of dedifferentiation
III : Marked

usually associated with H. pylori
Rx - Anti-H. pylori

⑤ DYSPLASIA

- universal precursor of gastric cancer

Mild dysplasia - endoscopic biopsy surveillance
H. pylori eradication

SEVERE DYSPLASIA < Localised - Endoscopic Mucosal Resection
Widespread / Multifocal - Gastric resection

SurgeLight chandanasri@gmail.com

CLINICAL FEATURES

- Non-specific symptoms :
  - " Anemia
  - Asthenia
  - Anorexia "
  - Vague upper abdominal discomfort

  | Dyspepsia
  | GERD

- F/s/o GOO
  - Vomiting, UGP
  - Succusion splash
- Abdominal mass
- Ascites
- Troisier sign, Blumer's shelf, Sister Mary Joseph nodule
- Krukenberg tumor

EVALUATION

- CBC
- BMP
- LFT, Albumin & Prealbumin

} Laboratory Workup

- UPPER GI ENDOSCOPY

Screening Endoscopy

- FAP - Screening EGD - commence at 25y
  repeat ½-4 yearly
  based on SPIGELMAN STAGE of Duodenal polyposis
- Endoscopic screening is done in high prevalence countries like
  - Japan
  - South Korea
  - Hereditary Gastric Cancer Syndromes

Diagnostic Endoscopy

- Identifying high risk & malignant lesions
  [aided by CONFOCAL LASER ENDOMICROSCOPY (CLE)
  Flexible Spectral Color Enhancement endoscopy
- Tissue collection for HPE & molecular profiling (HER2Neu, PD 1)
- Planning extent of resection & systemic therapy (as in LINITIS PLASTICA)
- EUS - staging (T & N)

- Principles of Endoscopic biopsy - Multiple biopsy specimens (6-8) from different sites of the lesion
  Single biopsy sensitivity ~70% ; 7 biopsies - 98%.
- Contrast Enhanced Cross-sectional Imaging :
  - CT c IV + Oral contrast
  - PET CT - helps detect occult disease
    - Lacks accuracy in mucinous / diffuse disease - LINITIS PLASTICA
- Diagnostic Laparoscopy & Peritoneal Cytology
  To avoid non-therapeutic laparotomies - Ascites
  Peritoneal deposits
  Linitis plastica

SurgeLight chandanasri@gmail.com

STAGING OF CA STOMACH – TNM staging

T_{is} – Carcinoma in situ, no lamina propria invasion, high grade dysplasia

T_1 – T_{1a} – invades lamina propria, muscularis mucosae
T_{1b} – invades submucosa

T_2 – invades muscularis propria

T_3 – penetrates subserosal tissue

T_4 – T_{4a} – invades serosa
T_{4b} – invades adjacent organs

N_1 – 1-2 regional nodes

N_2 – 3-6 regional nodes

N_3 – 3a – 7-15 regional nodes
3b – >16 regional nodes

M_0 – No distant metastasis
M_1 – Distant metastasis

G_1 – Well differentiated
G_2 – Moderately diff
G_3 – Poorly diff / undiff

0	T_{is}	N_0	M_0
I	T_1, T_2	N_0	M_0
II A	T_1, T_2	$N_1, N_2 N_3$	M_0
II B	T_3, T_{4a}	N_0	M_0
III	T_3, T_{4a}	N_1, N_2, N_3	M_0
IV A	T_{4b}	Any N	M_0
IV B	Any T	Any N	M_1

Surgeelight chandanasri@gmail.com

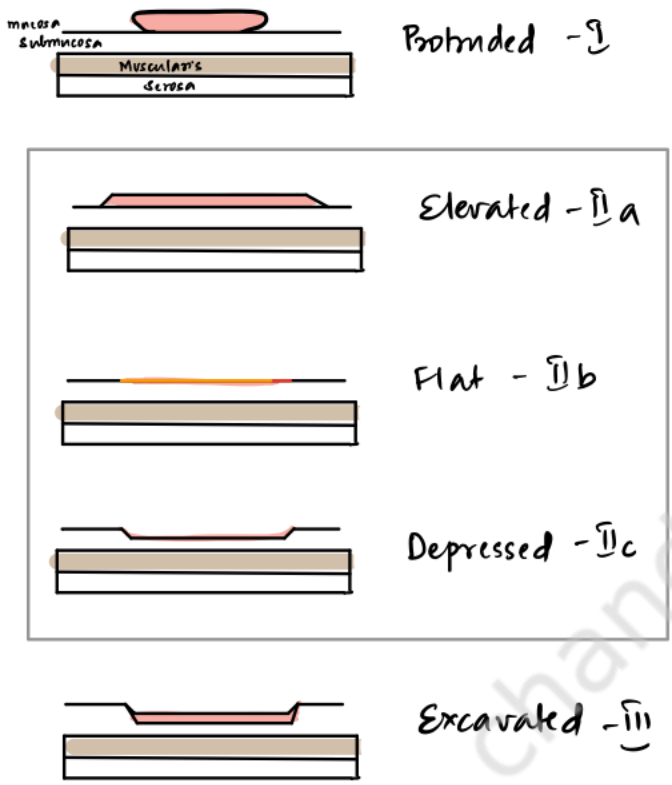
CLASSIFICATION

① BASED ON DEPTH OF INVASION

EARLY GASTRIC CANCER
Involvement of mucosa ± submucosa
(T1a) (T1b)

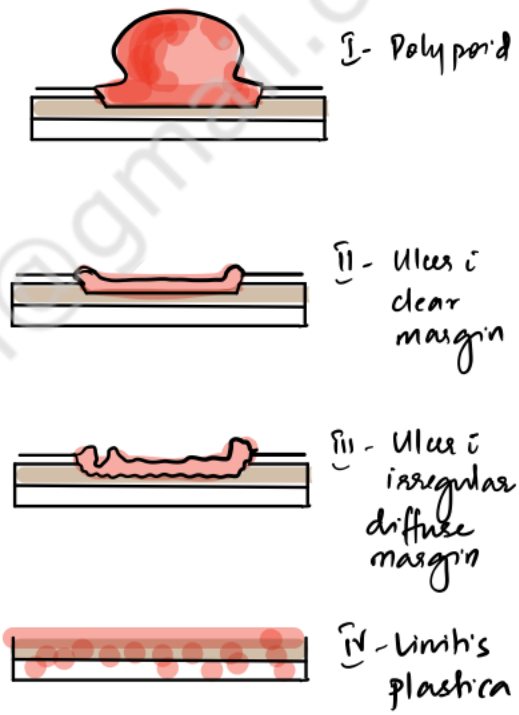
ADVANCED GASTRIC CANCER
Involvement of muscularis ± serosa

JAPANESE CLASSIFICATION



- Protruded - I
- Elevated - IIa
- Flat - IIb
- Depressed - IIc
- Excavated - III

BORMANN CLASSIFICATION



- I - Polypoid
- II - Ulcer ; clear margin
- III - Ulcer ; irregular diffuse margin
- IV - Linitis plastica

② PATHOLOGICAL CLASSIFICATION (Based on Histopathology)

Lauren's Classification

INTESTINAL
- Environmental - H.pylori
- Gastric atrophy, Intestinal Metaplasia } Premalignant
- Men > Women
- ↑ Age
- Gland formation
- Hematogenous spread
- Microsatellite instability
- APC gene mutations
- p53, p16 inactivation

Other / Indeterminate

DIFFUSE
- Familial
- Blood type 'A'
- Women > Men
- Younger age
- Poorly differentiated, Signet
- Transmural / lymphatic spread
- ↓ E-cadherin
- p53, p16 } inactivation

SurgeLight chandanasri@gmail.com

③ WHO Classification

- ADENOCARCINOMA
  - Papillary
  - Tubular
  - Mucinous
  - } Intestinal type
  - Signet cell & other poorly cohesive Ca } Diffuse type
  - Mixed → Other / Indeterminate
- ADENOSQUAMOUS CELL CARCINOMA
- SQUAMOUS CELL CARCINOMA
- UNDIFFERENTIATED
- UNCLASSIFIED

④ MING's CLASSIFICATION

- Expanding → Favorable
- Infiltrative → Unfavorable

SITE OF OCCURENCE

- Gastroesophageal junction & Cardia → now-most common ~60% → especially in the developed world
- Body (~15%)
- Pylorus (~20%)

Rest of the world – Pylorus & antrum (Distal stomach) is the most common site

MOLECULAR PATHOLOGY

- c-met, K-sam, c-erbB2 } proto-oncogenes over expressed in gastric cancers
- P16, P53 } tumor suppressor genes inactivated in gastric cancer

E-Cadherin – cell adhesion molecule → ↓/⊖ in diffuse-type gastric cancer (50%)

- Microsatellite instability (20-30%)

SurgeLight chandanasri@gmail.com

SPREAD

Diffuse type - submucosal & subserosal lymphatics

Intestinal type - hematogenous spread

Direct spread - penetrates muscularis & serosa
↓
adjacent organs
- pancreas
- colon
- liver

Lymphatic spread

Embolic
↓
Embolus of malignant cells in the collecting lymphatics
→ m/c

Infiltration/ Permeation
↓
Growth of a colony of tumor cells along the course of the lymphatic vessel

Spread to (L) Supraclavicular node (Virchow node) - Troisier's sign

Hematogenous spread

liver > lung > Bone

TRANSPERITONEAL SPREAD

PERITONEAL DEPOSITS
ASCITES
BLUMER'S SHELF
KRUKENBERG TUMOR
SISTER MARY JOSEPH NODULE

Paraneoplastic Syndromes associated with Gastric Cancer

1) Acanthosis nigricans (55% of malignancy-associated AN is seen in Ca stomach)
2) Polymyositis, Dermatomyositis
3) Cerebellar ataxia
4) Leser Trelat Sign

Surgery Light chandanasr@gmail.com

MANAGEMENT

- Endoscopic resection of Early Gastric Cancer
  - Tumor limited to the mucosa (or limited Submucosal extension ≤0.5mm)
  - No lymphovascular invasion
  - Tumor smaller than 2cm
  - No ulceration
  - Well / moderately differentiated histology
- EMR / ESD

- CURATIVE SURGERY
  (T1b – T3 tumors)

Criteria of Unresectability for cure:
- Locoregionally advanced: Infiltration of root of mesentery
  Invasion / Encasement of major vascular structures (excluding splenic vessels)
- Distant metastasis / peritoneal seeding / para-aortic nodes

Distal / Total / Subtotal gastrectomy ± Roux-en-Y reconstruction [4-6cm proximal, 2cm distal margin]
± D_2 dissection ≥15 lymphnodes should be removed for proper staging
consider FJ

NEOADJUVANT CHEMOTHERAPY

- May be given to improve the chances of R_0 resection in T_{2+}, N_+

  Eg: in proximal gastric cancers
  Linitis plastica
  Borderline performance status

Regimens

Fluoropyrimidine (5FU / Capecitabine) + Platin (Cisplatin / Oxaliplatin)

Fluoropyrimidine (5FU / Capecitabine) + Docetaxel + Cisplatin

ECF – Epirubicin + Cisplatin + Capecitabine / Fluorouracil

RT – 45 – 50.4 Gy (1.8 Gy/d)

SurgeLight.chandanasingh@gmail.com

Palliative Surgery

- Palliative gastrectomy - bleeding tumors
- GJ- Obstruction
- FJ- feeding
  - Devine's pyloric exclusion

SurgeLight chandanasri@gmail.com

PEPTIC ULCER DISEASE

Definition - erosions in the gastric / duodenal mucosa that extend through the muscularis mucosae → into submucosa / deeper

- **PHYSIOLOGY OF GASTRIC ACID SECRETION**

- Hydrochloric acid is secreted by gastric parietal (oxyntic) cells in response to: (→ Total acid production ∝ parietal cell mass)
  - D-cells - Somatostatin (⊖)
  - Acetylcholine (vagus) (⊖)
  - Gastrin (G cells) (⊖)
  - Histamine (⊕) (Enterochromaffin-like cells)
- Parietal cell proton pump - H^+/K^+ ATPase

ACID SECRETION occurs in 3 phases:

1) Cephalic : thought, sight, smell, taste of food → NTS, DMN, DVC → VAGUS → Acetylcholine → ↑ Acid Secretion — 30%

2) Gastric : when food reaches stomach → amino acids, small peptides → G-cells → Gastrin → ↑ Acid secretion; proximal gastric distension → Vagovagal reflex → ↑ Acid secretion; ANTRAL DISTENSION → ↑ Acid secretion — 60%

3) Intestinal : luminal chyme → proximal small bowel mucosa → (ECL-Histamine) hormone mediated ↑ Acid secretion — 10%

INTERDIGESTIAL ACID SECRETION → 10% OF MAX. GASTRIC OUTPUT

PATHOGENESIS OF PUD

↑ AGGRESSIVE FACTORS

- ↑ HCl production (Gastrinoma, Retained antrum, Prolonged fasting)
- ↑ pepsins
- Ethanol ingestion
- Smoking
- Duodenal bile reflux (Roux limb)
- Ischemia · Hypoxia
- NSAIDs, Steroids (Methamphetamine, Cocaine)
- H. pylori infection
- Zollinger Ellison Syndrome
- STRESS - Cushing ulcer, Curling ulcer (ICU)
- Cocaine - juxtapyloric

↓ DEFENSIVE FACTORS

- Mucosal bicarbonate secretion
- Mucus production
- Blood flow
- Growth factors
- Cell renewal
- Endogenous prostaglandins
- Somatostatin / Gastrin balance

injury to gastroduodenal mucosal barrier → MUCOSAL DAMAGE → ULCERATION

DYSREGULATION

95% of non-NSAID ulcers have antral gastritis

surgeonlight chandanasri@gmail.com

Helicobacter pylori infection

80-95% Duodenal ulcers
75% Gastric ulcers
} → a/c H. pylori infection

H. pylori

- spiral / helical gram-negative bacillus - 4-6 flagella
- resides in gastric-type epithelium within / beneath the mucosal layer (stomach, heterotopic gastric mucosa - Meckel's, Barrett's, Gastric metaplasia in duodenum) rectum
- produces urease

UREA → AMMONIA + BICARBONATE

Alkaline microenvironment in gastric milieu

- Acute Pangastritis (noncrossive)
- Chronic antral gastritis (10%) → PUD
- Corpus dominant gastritis (90%) → Ca
- Hyperplastic gastric polyps
- ITP

MECHANISMS FOR H. PYLORI INDUCED G.I INJURY

1. Production of toxic products that cause local tissue injury
   - AMMONIA
   - CYTOTOXINS - vacuolating cytotoxin A, cag A, HSP 60, sialic acid
   - MUCINASE - degrades mucus & glycoproteins
   - PHOSPHOLIPASE - damages epithelial & mucous cells
   - PAF (Platelet Activating Factor) → mucosal injury → thrombosis in microcirculation
2. Induction of local mucosal immune response
3. Increased Gastrin levels : H. pylori mediated destruction of antral 'D' cells
   ↓ Somatostatin levels ← antral alkalinization
   ↑ GASTRIN → ↑ HCl
   <SOMATOSTATIN/GASTRIN DYSREGULATION>
4. Gastric Metaplasia in the duodenum : Acid hypersecretion → ↓ duodenal pH → gastric metaplasia in duodenum as a protective response
   - Duodenal colonisation by H. Pylori
     ↓ Duodenal bicarbonate
     Duodenitis
     ↓
     Duodenal ulcer

INDICATIONS FOR H. PYLORI TESTING

1. Peptic ulcer
2. Gastritis
3. Significant dyspepsia
4. MALT Lymphoma
5. Early Gastric cancer

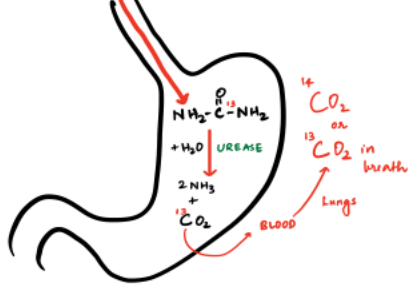
INVASIVE TESTS FOR H. PYLORI

1. Histological evaluation of gastric biopsies
2. Rapid urease test of fresh biopsy specimen

NON-INVASIVE METHODS OF H. PYLORI TESTING

1. Urea breath test - Sensitivity & specificity > 90% (+ve only in active infection)
2. Serology → +ve even after eradication
3. Detection of Stool antigen

^{13}C urea or ^{14}C urea



SergeLight.chandana@gmail.com

℞ Anti H. Pylori Regimens

- Clarithromycin 500mg BD + Amoxicillin 1g BD / Metronidazole 500mg TID + PPI
- Metronidazole 500mg BD + Amoxicillin 1g BD + PPI
- Sequential: PPI x 14 d ⟶ Amoxicillin 1g BD x 7d ⟶ clarithromycin + Metronidazole x 7d
- Bismuth Quadruple Therapy (Second line): PPI + Bismuth subsalicylate 300mg QID + Tetracycline 500mg QID + Metronidazol 250mg QID

NSAIDs

Systemic inhibitors of cyclo-oxygenase enzymes

↓ Prostaglandins

↓ Bicarbonate & mucin secretion

↓ Blood flow to mucosa

Gastric & Duodenal Ulcer

Acid → important but non-causative role in ulcer formation
- 70% of pt i duodenal ulcer have acid production in (N) range
- Acts as co-factor - exacerbates underlying ulcer damage attenuates healing

Stress Ulcer

Severe Physiological stress ←
- Head injury (Cushing ulcer)
- Burns >35% TBSA (Curling ulcer)
- Hypotension
- AKI, Liver failure
- Coagulopathy
- Sepsis
- Major surgery
- ↑ dose steroids

↓ Splanchnic hypoperfusion

↑ acid secretion ↓ Reperfusion injury

↓ Breakdown of Gastroduodenal mucosal barrier

Management: Prophylactic H_2 blockers / PPIs
- EGD
- Selective Vasopressin infusion
- Operative intervention as last resort

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PEPTIC ULCER LOCATION

DUODENAL > GASTRIC > MARGINAL / ANASTOMOTIC

Japan (Gastric > Duodenal)

MARGINAL ULCERS

- Peptic ulcers which characteristically occur on the jejunal side of the gastrojejunostomy following
  - DISTAL GASTRECTOMY
  - GASTRIC BYPASS (BARIATRIC)
  - SIMPLE GJ

- Risk of marginal ulceration depends on
  - ACID/PEPTIC load delivered into the jejunum (unaccustomed to any acid)
  - luminal jejunal buffering (absent in Roux GJ)

RISK FACTORS

1. ROUX GASTROJEJUNOSTOMY
2. LARGE GASTRIC POUCH AFTER DISTAL GASTRECTOMY
3. ROUX-EN-Y GASTRIC BYPASS
4. RETAINED / EXCLUDED ANTRUM
5. INCOMPLETE / INADEQUATE VAGOTOMY

- Ischemia
- Permanent suture material

Complications

- Perforation
- Obstruction
- Bleeding
- Non-healing
- Malignancy

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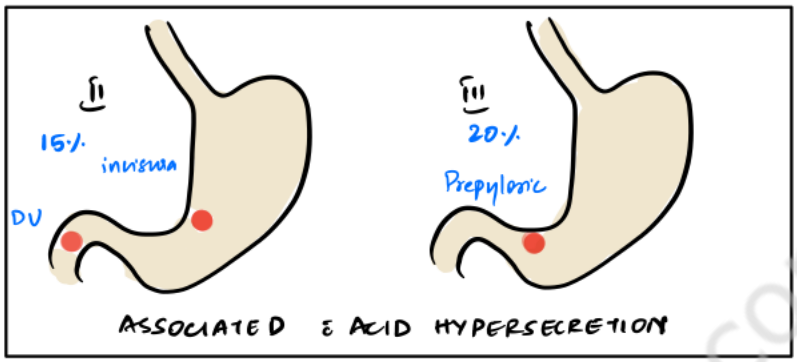
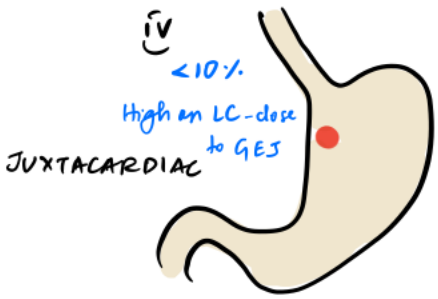
GASTRIC ULCER

Modified Johnson Classification



60%. ← LC incisura
↓
most occur within 1.5 cm of the transition between fundic & antral mucosa



NSAID induced ulcer
ANY WHERE IN THE STOMACH
Greater curvature ulcers < 5%.

- Peak incidence in 55-65y

- H. pylori
- NSAID
- Chronic alcohol intake
- Smoking
- Long-term corticosteroids
- Higher risk of Malignancy

CLINICAL FEATURES

1) PAIN : non-radiating, burning/stabbing/gnawing quality, epigastric → RADIATION TO BACK → posterior perforation
commonly worsened by eating
less likely to awaken patient at night

2) Gastric Dysmotility - Delayed Gastric emptying
Abnormal pyloric sphincter function
Prolonged high amplitude gastric contractions
Duodenogastric reflux
Alterations in gastric migrating motor complex

~10% gastric ulcers are MALIGNANT / associated c Malignancy

COMPLICATIONS

- Hemorrhage - 35-40%
- M/c complication → PERFORATION
- obstruction
- spontaneous gastrocolic fistulas

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EVALUATION

- Routine
- Serum Gastrin levels

1. Flexible upper GI endoscopy
   - Visual diagnosis - ulcer, stricture
   - sampling of tissue to evaluate H. Pylori infection → H. Pylori testing
     - Silver stain
     - Giemsa stain
     - Genta stain
   - malignancy → 10% risk → aggressive biopsy / brushings

ALL GASTRIC ULCERS → MULTIPLE BIOPSIES FROM PERIMETER OF THE LESION

BENIGN GASTRIC ULCER
- Converging mucosal folds
- Regular margins
- Granulation tissue on floor
- Overhanging edge
- LC > GC

MALIGNANT GASTRIC ULCER
- Effaced mucosal folds
- Irregular margins
- Necrotic tissue on floor
- Everted edge
- GC - ↑ risk

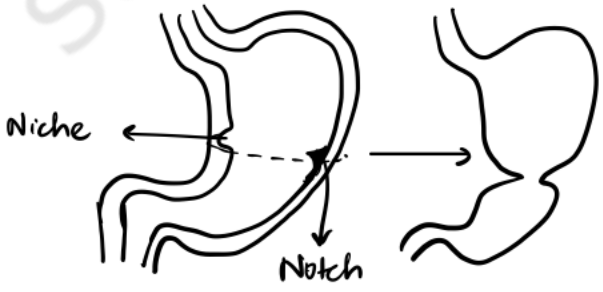
2. Upper GI radiography series
   - Barium meal → demonstration of Barium in ulcer crater (round / oval, edema ±)
     - DOUBLE CONTRAST → greater accuracy
     - location, depth & extent of deformation

BENIGN GASTRIC ULCER - BARIUM STUDY

- Ulcer crater projects beyond lumen of ulcer overhanging mucosa at the margins
- Margins - regular & rounded
- Mucosal folds preserved → converge towards ulcer

CICATRICAL GASTRIC ULCERS

HOURGLASS



WOMEN

TEAPOT

cicatrization & shortening of LC

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DUODENAL ULCER

- m/c occurs in first part of duodenum (>95%)
- well demarcated break in mucosa which may extend into the muscularis propria
- most duodenal ulcers are <1cm
- Benign

Clinical features:
Pain - midepigastric abdominal pain - well localised
generally tolerable, frequently RELIEVED BY FOOD
Episodic (PERIODICITY) (constant pain → deeper penetration)
Worse during emotional stress
Referral to back → penetration into pancreas

GIANT DUODENAL ULCER - full thickness peptic ulcer ≥ 2cm in diameter usually involving a large portion of the duodenal bulb
↓ association c H-pylori, ↑ association c NSAIDs
- PERSISTENT PAIN
Perforation - hazardous d/t extensive tissue loss → simple patch → post op leak
℞ - PARTIAL GASTRECTOMY c B II / Thal patch / Conversion into pyloroplasty

COMPLICATIONS
- m/c → Bleeding → almost always managed non-operatively
- Perforation - m/c indication for surgery in duodenal ulcer
Most perforations arise from ulcers in the anterior aspect of duodenal cap (92%)
- 10% are associated with concurrent bleeding ulcer in posterior wall - KISSING ULCER
- Obstruction - 3rd m/c complication

(Intractability - rare in anti-H.pylori & Acid suppressant era)

DIAGNOSIS

Upper GI endoscopy - ulcer - site, size, depth, number
bleeding
tissue for H. pylori
Other pathologies

Upper Gastrointestinal Radiography

surgeonlightchandanasri@gmail.com

MANAGEMENT

1) Medical Management : Antacids, Sucralfate
Acid suppression - PPIs, H_2 blockers
H. pylori eradication
elimination of risk factors
optimize → Non-healing → r/o malignancy by multiple endoscopic biopsies

2) SURGICAL MANAGEMENT :

Surgical approach for NON-HEALING but otherwise UNCOMPLICATED gastric ulcers depends on - type of ulcer
- acid levels

TYPE-1 gastric ulcers (LC, incisura): malignancy is a concern
excision of non-healing ulcers - recommended

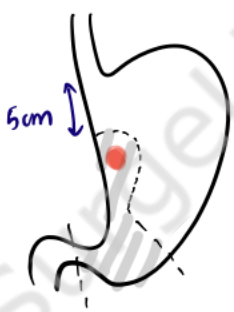
DISTAL GASTRECTOMY ± VAGOTOMY (questionable) / WEDGE RESECTION OF ULCER ALONE

Type-2 & 3 gastric ulcers → associated ± ↑ secretion of acid

DISTAL GASTRECTOMY + TRUNCAL VAGOTOMY
(HSV alone - poor outcomes)

Type-4 (near GEJ - cardia)
→ difficult to manage
ulcer excision ✓ → try to do it without gastrectomy (morbidity of small gastric remnant)

PAUCHET PROCEDURE



The ulcer is included in the distal gastrectomy

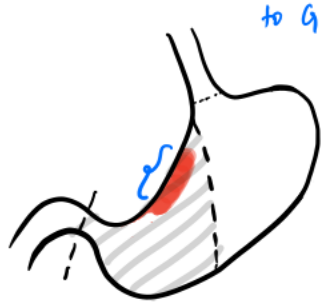
KELLING MADLENER PROCEDURE

- Ulcer itself is not resected

Vagotomy + Antrectomy to reduce acid

CSENDES PROCEDURE

For ulcers located 2cm - 5cm distal to GEJ



Vertical extension of the resection into include lesser curvature

Type-5 - Definitive antisecretory surgery

(TRUNCAL VAGOTOMY + ANTRECTOMY) should be considered if Medical management fails / NSAIDs cannot be stopped

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COMPLICATED GASTRIC ULCER DISEASE

① BLEEDING ULCER - source is usually ⓛ Gastric Artery

Resuscitation
↓
Endoscopic investigation → FORREST CLASSIFICATION
↓
ENDOSCOPIC CONTROL OF BLEEDING
↓
EPINEPHRINE + THERMO-COAGULATION OR CLIPPING

FORREST CLASSIFICATION
- Ia - Spurting, Ib - Oozing } ACTIVE BLEED
- IIa - Visible vessel
- IIb - Clot
- IIc - Hematin in ulcer base
- III - Clean ulcer

FAILED ENDOSCOPIC CONTROL
↓
ENDO-VASCULAR EMBOLIZATION → SURGERY
- Oversew
- ulcer biopsy
± VAGOTOMY

② PERFORATED ULCER

Type I perforated GU
- Stable → DG + Billroth I
- Unstable → Biopsy + Graham patch closure

Type II & III → Patch Closure ± Truncal vagotomy (TV + Drainage)

③ GIANT GASTRIC ULCERS

Diameter ≥ 2cm
found in LC
Higher incidence of malignancy than smaller ulcers (6-30%)
usually penetrate into surrounding structures (even when benign)
↓
MEDICAL THERAPY → 80% heal
↓
Failure to heal → Gastrectomy + Vagotomy (II & III)

④ OBSTRUCTION - Gastrectomy / Gastrojejunostomy

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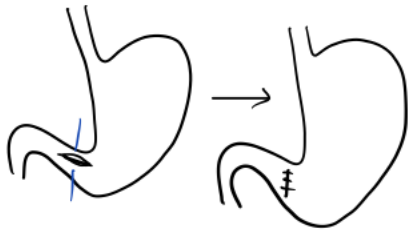
SURGICAL STRATEGIES IN PEPTIC ULCER DISEASE

① VAGOTOMY — TV / SV / HSV
- to eliminate direct cholinergic stimulation of gastric acid secretion

② DRAINAGE PROCEDURES - as adjuncts to vagotomy

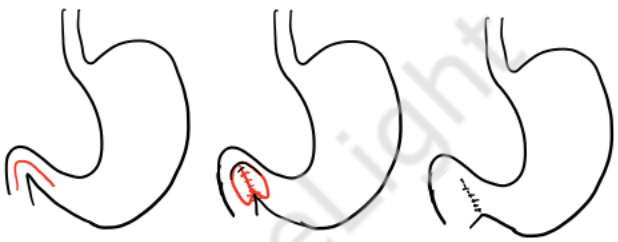
PYLOROPLASTIES

① HEINEKE-MIKULICZ PYLOROPLASTY



- Kocherization
- 2 traction sutures on anterior surface of pylorus at 12 & 6 'o' clock (include pyloric vein of Mayo)
- Full thickness horizontal incision over anterior surface starting 2-3 cm proximal to pylorus – extending through pylorus - 2-3 cm over anterior duodenum
- Using traction sutures to pull the horizontal incision into diamond (◇) shaped wound → sutured longitudinally

② FINNEY PYLOROPLASTY



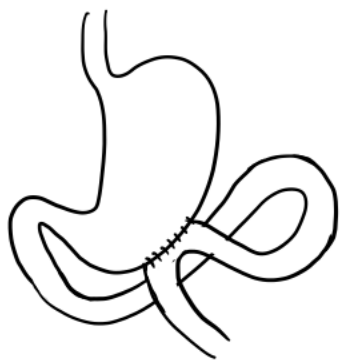
③ JABOULAY GASTRODUODENOSTOMY

GASTROJEJUNOSTOMIES

- Gastrojejunostomy is indicated when there is duodenal obstruction and the duodenal bulb is so scarred, inflammed and edematous that pyloroplasty is not safe
- Drainage procedure of choice when vagotomy is performed by lap.

Anastomosis between proximal jejunum & most dependent portion of greater curvature

ANTECOLIC | RETROCOLIC



Complications

1) Marginal ulceration
2) Afferent / Efferent loop obstruction
3) Internal hernia
4) Intussusception

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③ GASTRIC RESECTION PROCEDURES

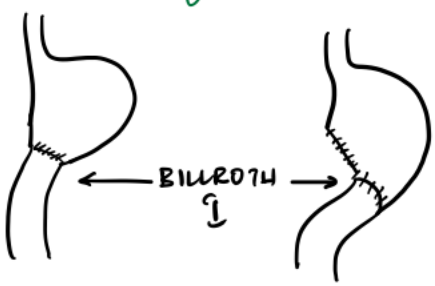
ANTRECTOMY (→ 40% Distal Gastrectomy) (to ↓ Gastric phase) + Vagotomy to abolish acid secretion + Reconstruction

PARTIAL GASTRECTOMY + BILLROTH I (B-I)

- Gastric remnant anastomosed to the duodenum

Advantages of B-I

1) Restores (N) GI continuity
2) Leaves specialised duodenal mucosa next to gastric mucosa
3) Avoids problems a/i afferent & efferent limbs
4) Easier endoscopic examination of bowel & ERCP
5) ↓ incidence of Ca in remnant stomach (long follow up >15y)

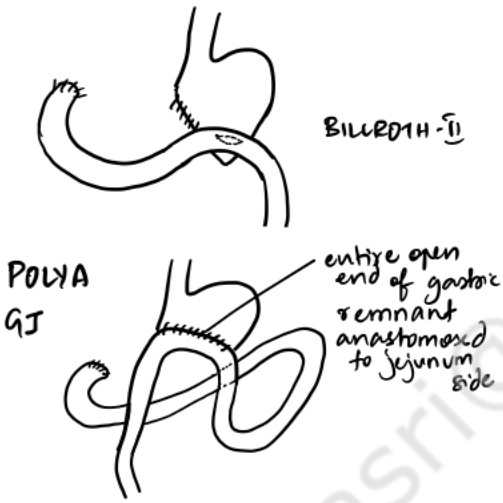


Limitations

- Cannot be done in extensively scarred duodenum
- Tension in the anastomosis if the gastric remnant & duodenum cannot be brought together easily
- Bile reflux

PARTIAL GASTRECTOMY + BILLROTH II (B-II)

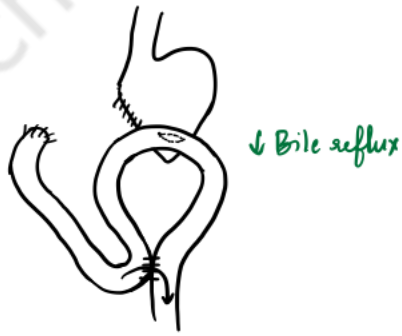
- Gastric remnant anastomosed to a loop of jejunum after closing duodenal stump



Should be coupled ± acid suppression surgery to prevent marginal ulceration

Biliary reflux as in B-I

BRAUN - JEJUNO-JEJUNOSTOMY

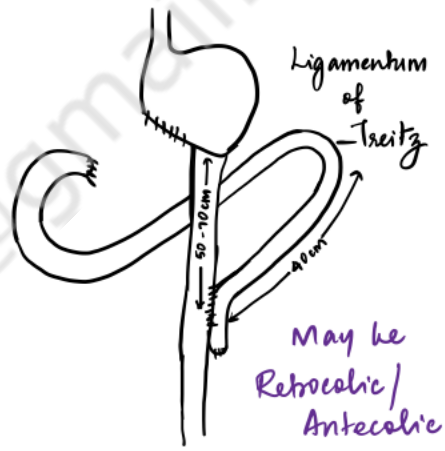


An entero enterostomy downstream from GJ will divert the biliary secretions from the duodenum into the jejunum

PARTIAL GASTRECTOMY ± ROUX EN Y

Jejunum divided 40cm distal to ligament of treitz

50-70cm Roux-limb created & side to side jejunojejunostomy fashioned



May be Retrocolic / Antecolic

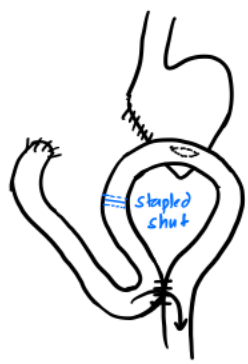
ADVANTAGE

↓ Bile reflux

DISADVANTAGE:

- More ulcerogenic d/t complete lack of duodenal contents in the vicinity of the gastrojejunostomy
- Extra anastomosis

UNCUT ROUX-EN-Y



Complications: POST GASTRECTOMY SYNDROMES (See notes)

Surgeon right chandrasrigo@gmail.com

MANAGEMENT OF COMPLICATED PEPTIC ULCER DISEASE

COMPLICATION	TYPE OF PEPTIC ULCER: DUODENAL	GASTRIC	MARGINAL
Perforation	PATCH PATCH + HSV PATCH + TV/D TV + Antrectomy	Biopsy and patch Excise ulcer / close defect Distal gastrectomy if ulcer	Patch Resection of GJ
Bleeding	Oversew alone Oversew + TV/D TV + A	Biopsy & oversew Distal gastrectomy if ulcer	Oversew alone Resection of GJ
Obstruction	HSV + GJ TV + GJ TV + A	HSV + GJ / ulcer biopsy TV + GJ / ulcer biopsy TV + A	Resection of GJ
Non healing	HSV + GJ TV + A	Wedge Resection Distal Gastrectomy + Roux	Resection of GJ

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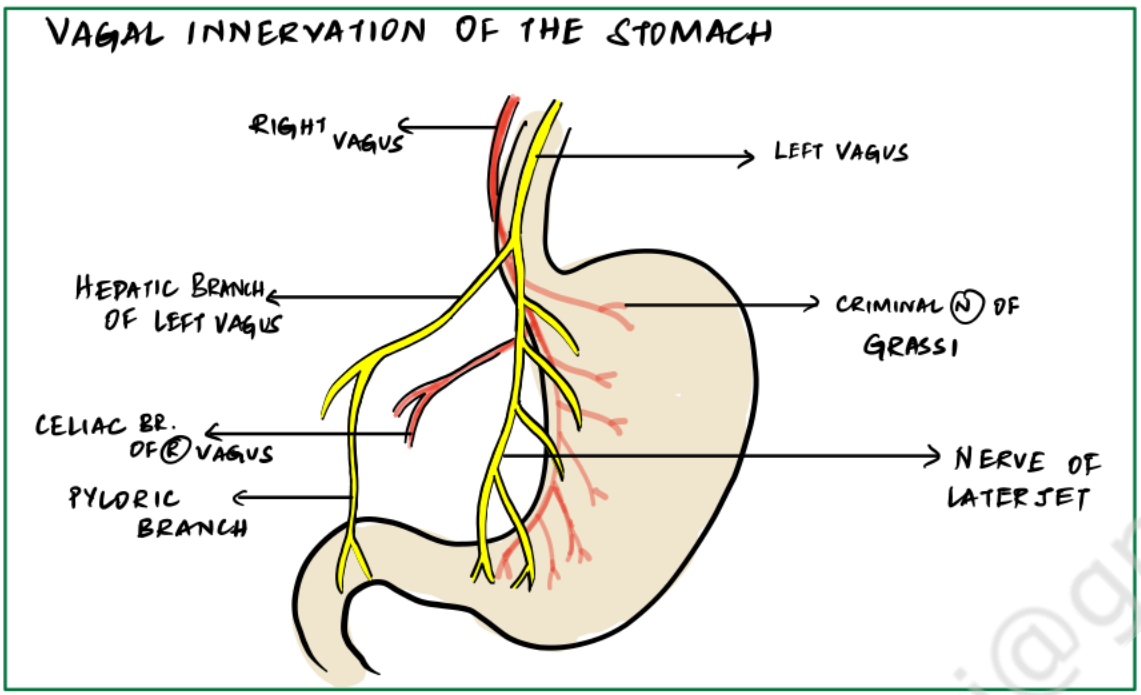
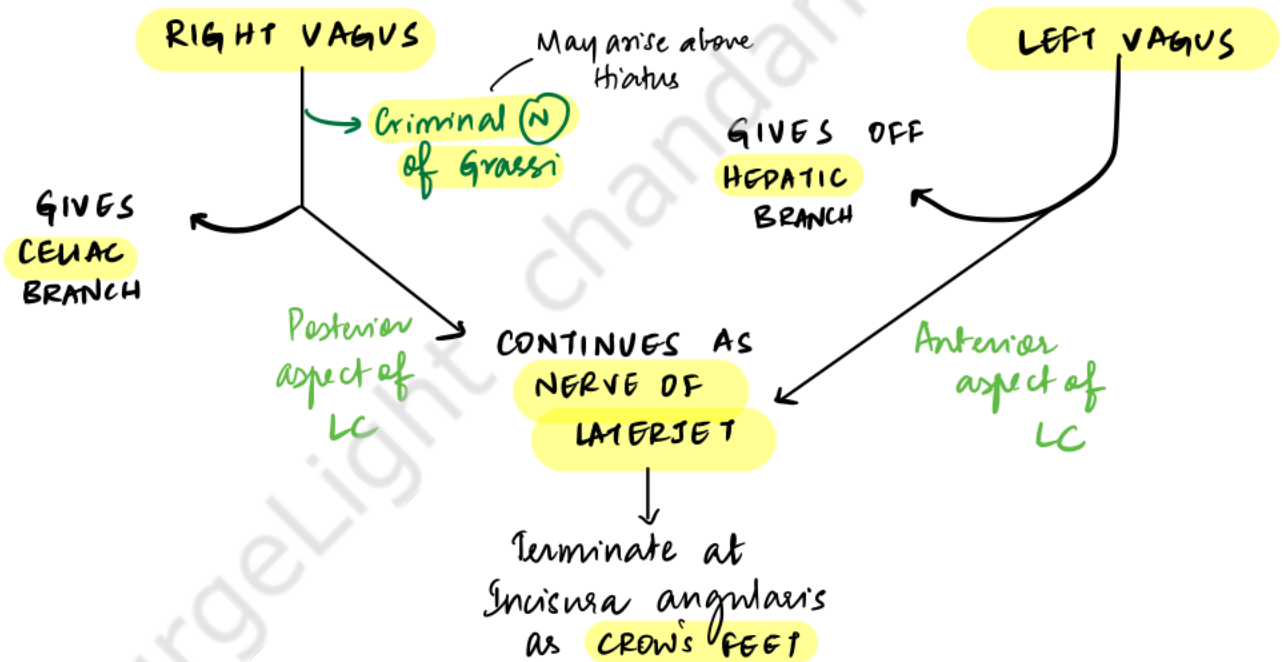
VAGUS & VAGOTOMIES



- Vagus nerves arise from vagal nucleus in the floor of the 4th ventricle
- traverse the neck in the carotid sheath
- enter mediastinum
- give off RLN
- give esophageal branches in the thorax
- enter esophageal hiatus

'LARP' (L) → Anterior
(R) → Posterior



- 90% of vagal nerve fibres are sensory - carry stimuli from GIT to brain
- 10%. efferents from dorsal nucleus of medulla → synapse in myenteric & submucosal plexuses

CRIMINAL NERVE OF GRASSI - first branch of the posterior trunk
- innervates gastric fundus
- failure to divide during acid-reducing surgery ⇒ Recurrent ulcers

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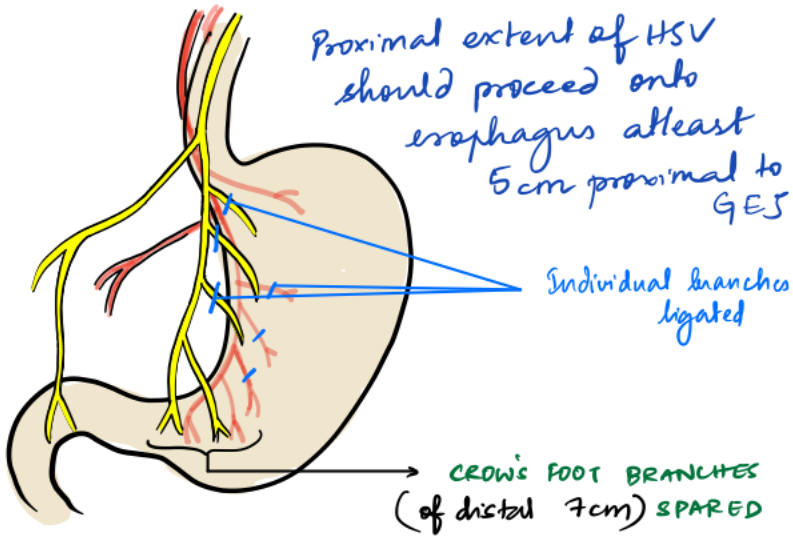
VAGOTOMIES

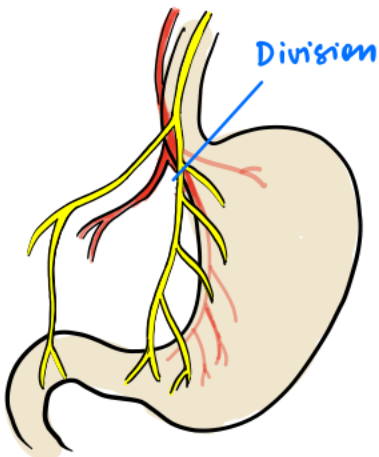
1) PARIETAL CELL / HIGHLY SELECTIVE VAGOTOMY



- Vagal innervation of the proximal 2/3rds of stomach is divided ↓ (location of parietal cells)
- Antropyloric innervation is spared to prevent pylorospasm due to unopposed sympathetic activity
- Innervation of the rest of the abdominal viscera is preserved

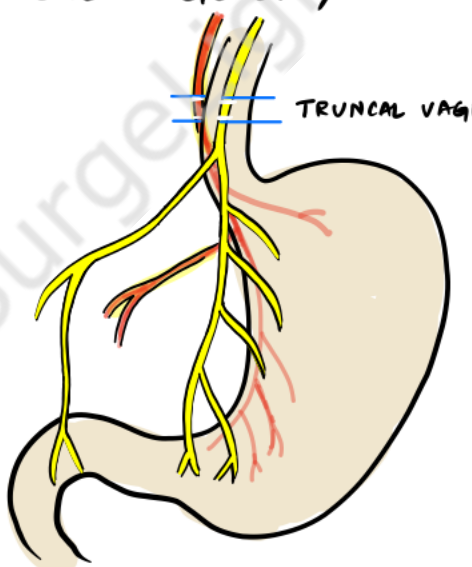
- NOT SUITABLE FOR - type II (Gastric + Duodenal ulcer) & type III (Prepyloric) ulcer disease
  (∵ Hypergastrinemia d/t gastric outlet obstruction & persistent antral stasis)

2) SELECTIVE VAGOTOMY



- Division of anterior and posterior vagi distal to hepatic and celiac branches
- Requires drainage procedure
- Can be combined with antrectomy

3) TRUNCAL VAGOTOMY



- Anterior and posterior vagal trunks are divided proximal to the hepatic and celiac branches
- Coupled with a DRAINAGE PROCEDURE
  (∵ antropyloric mechanism is denervated)
  - Pyloroplasty (Heincke Mickulicz)
    or
  - Gastrojejunostomy (preferred i/c/o cicatrised duodenum)
- GOOD RESULTS in complicated PUD

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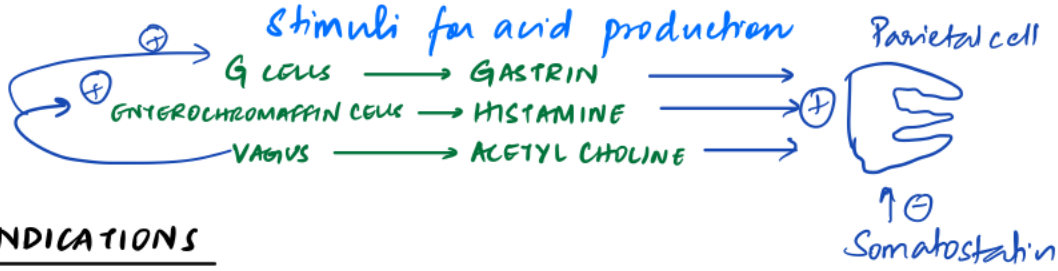
PHYSIOLOGY

- Gastric acid is secreted by PARIETAL CELLS mainly located in the body of the stomach



INDICATIONS

Can be coupled i other surgeries for complicated PUD to ↓ recurrence

Bleeding duodenal ulcer
Bleeding gastric ulcer } If stable, if contamination ↓
GOO
Intractability
Recurrence

COMPLICATIONS

1. Delayed Gastric Emptying - due to loss of vagally mediated receptive relaxation (∴ DRAINAGE PROCEDURES ARE DONE)
2. Postvagotomy diarrhea - intestinal dysmotility, bile acid malabsorption
3. Postvagotomy hypergastrinemia - Parietal cell denervation → ↓acid → G cell stimulation & hypergastrinemia
4. DUMPING S° - if antrectomy / drainage procedure is done

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Gastrocolic Fistula

- Between stomach & colon (transverse colon - m/c)
- **Etiology**

Benign	Malignant	Iatrogenic
• Crohn's disease affecting transverse colon • Peptic ulcer disease • Foreign body • Pancreatitis	Ca stomach Ca Colon Lymphoma Recurrence at ileocolic anastomosis	• PEG → colon gets pinched between stomach & anterior abdominal wall - Post surgical

- **Presentation**

Pain abdomen
Abdominal distension
Diarrhea
Vomiting - feculent vomiting
Feculent breath
Anemia
Dehydration
Malnutrition

Evaluation

Upper GI Barium studies / Barium enema - demonstrates communication
Endoscopy / Colonoscopy → can miss Dx
CT ± IV & oral contrast

Rx - Surgical resection of fistula - En bloc resection + Roux en Y GJ
Gastroscopic fibrin sealant

SurgeLight chandanasri@gmail.com

BARIATRIC AND METABOLIC SURGERY

INDICATIONS FOR BARIATRIC SURGERY

- BMI >40 Kg/m² OR >35 Kg/m² + Medical comorbidity worsened by obesity
- Failed dietary therapy
- Psychiatrically stable (no alcohol dependence / illegal drug use)
- Motivated, Knowledgeable

METABOLIC SYNDROME (≥3/5)

CRITERIA	DEFINITION
Abdominal obesity	Waist circumference < M → >40 in (102 cm); F → >35 in (88 cm)
Hypertriglyceridemia	≥150 mg/dL
Low HDL	M: <40 mg/dL ; F: <50 mg/dL
High Blood pressure	≥130/85 mmHg
High fasting glucose	≥110 mg/dL

BMI CLASSIFICATION (BMI in Kg/m²)

			INTERNATIONAL	ASIAN
NORMAL			18.5 - 25	18.5 - 23
OVERWEIGHT			≥25	≥23
	PREOBESE		25 - 30	23 - 27.5
	OBESE		>30	>27.5
		I	30 - 35	27.5 - 32.5
		II	35 - 40	32.5 - 37.5
		III	>40	>37.5

Mechanisms of effects of Bariatric Surgery

① WEIGHT LOSS DEPENDENT

Caloric restriction → long term, sustained weight loss
↓
Improvements in
- Glucose metabolism
- Lipid metabolism
- Hypertension

② WEIGHT LOSS INDEPENDENT

Bypass of the duodenum → ↑ exposure of nutrients to INCRETIN SECRETING cells in small & large intestine
↓
↑ GLP-1 secretion
↓
Improved glucose metabolism

↓
Alterations in gut microbiome

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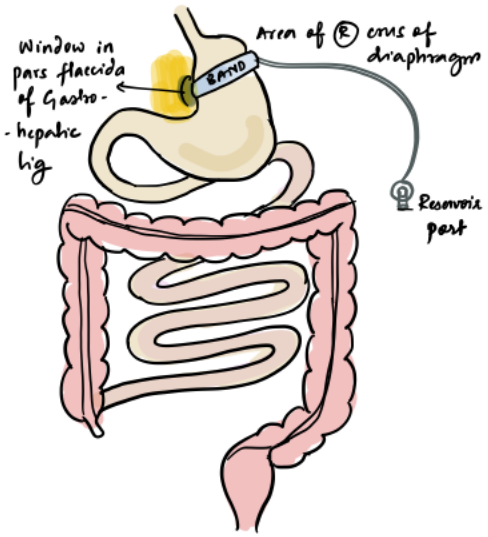
TYPES OF BARIATRIC PROCEDURES BASED ON MECHANISM OF ACTION

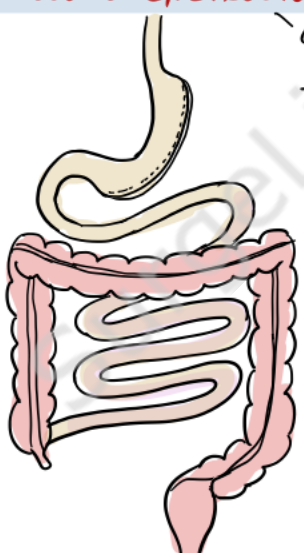
RESTRICTIVE

① LAPAROSCOPIC ADJUSTABLE GASTRIC BANDING (LAGB)



- Placement of an inflatable silicone ring around the proximal stomach
- Band is placed through a window in the pars flaccida of gastrohepatic ligament
- Stomach imbricated over band
- Band is attached to a reservoir system that allows adjustment of band tightness.
- Reservoir system is accessed through a subcutaneous port

② SLEEVE GASTRECTOMY

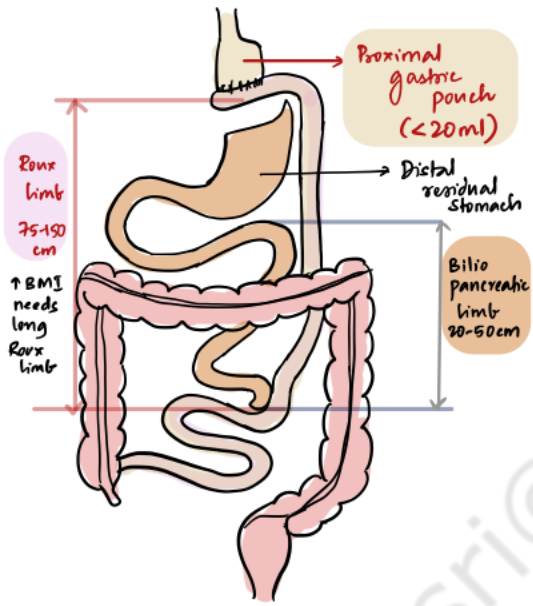


GC devascularised and a sleeve resected

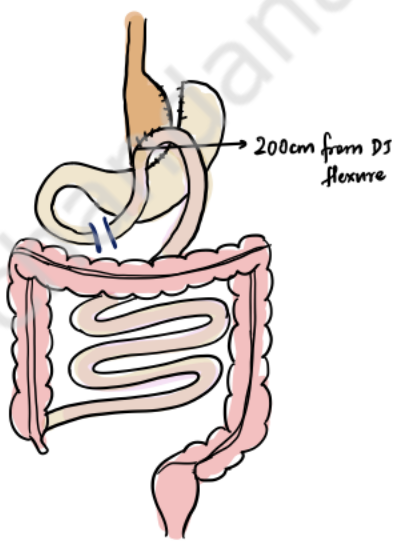
VERTICAL BANDED GASTROPLASTY - no longer done

LARGELY RESTRICTIVE
MODERATELY MALABSORPTIVE

① ROUX-EN-Y GASTRIC BYPASS



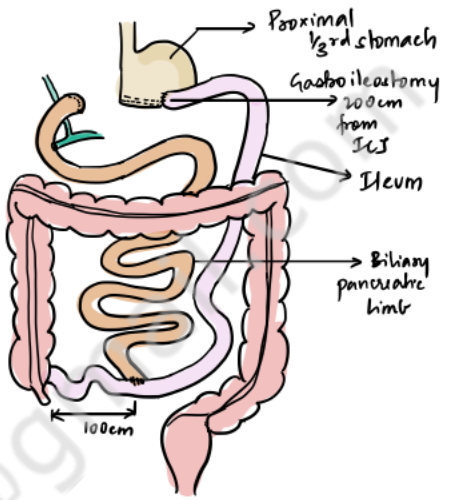
② MINI GASTRIC BYPASS



- small gastric pouch - early satiety
  - caloric restriction
- Bypass of duodenum - incretin stimulation
  ↓
  GLP-1 secretion
  ↓
  Improved glucose metabolism
- Alteration of bile acid levels
  ↓
  Improvement in cholesterol metabolism

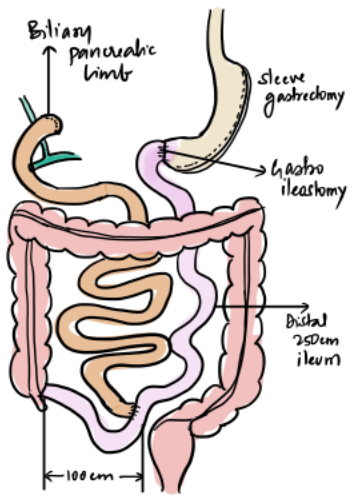
LARGELY MALABSORPTIVE
MILDLY RESTRICTIVE

① BILIOPANCREATIC DIVERSION

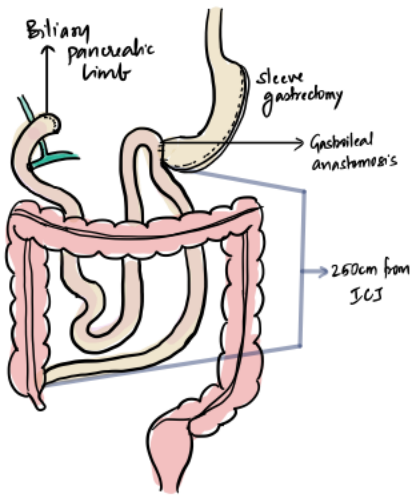


- Technically difficult
- Nutritional & metabolic complications ↑

② DUODENAL SWITCH



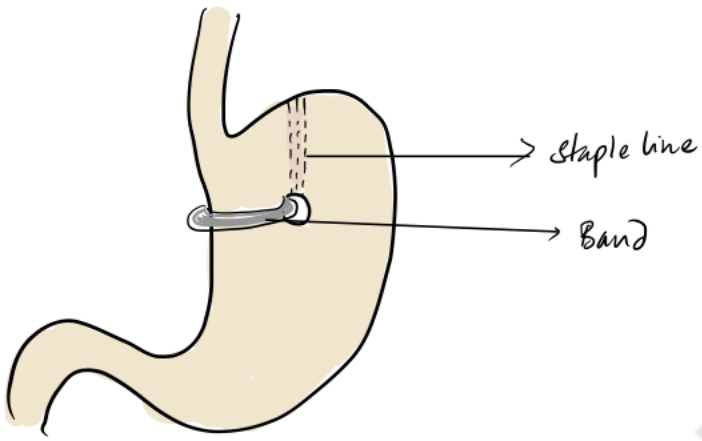
③ SADI-S - Single Anastomosis Duodeno-ileal bypass : Sleeve / Loop duodenal switch



OLDER PROCEDURES

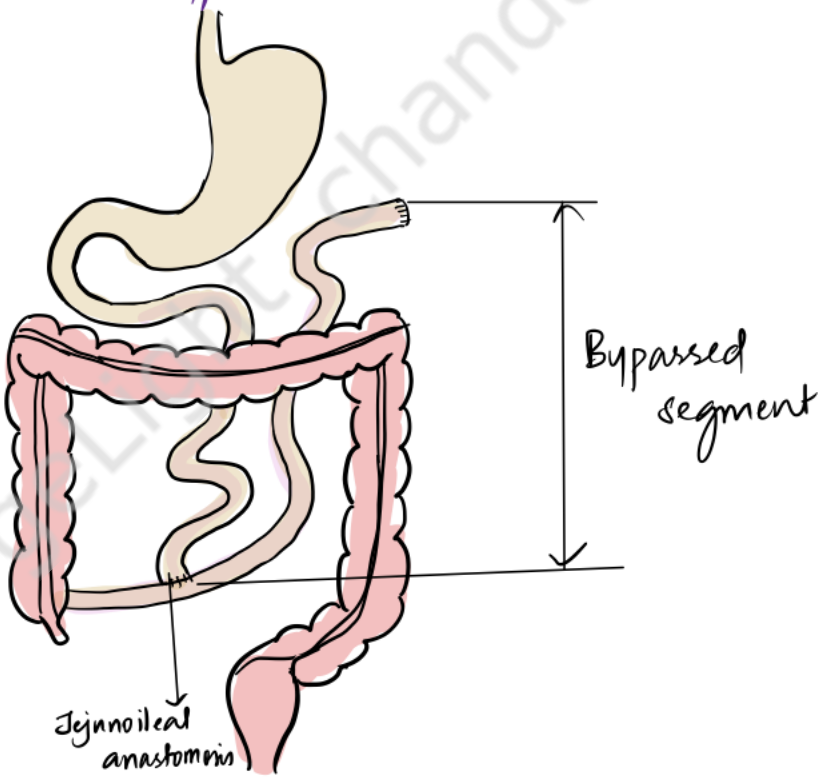
① Purely restrictive

Vertical Banded Gastroplasty



② Purely Malabsorptive

Jejunoileal bypass



SurgeLight.chandanasri@gmail.com

RECENT ADVANCES

ENDOSCOPIC INTERVENTIONS

Endoluminal Restrictive procedures

1) ENDOCINCH - Endoscopic plication of gastric wall to reduce gastric lumen

2) TOGA - Transoral Gastroplasty

Endoscopic device is used to suction in the gastric mucosa to form a restrictive pouch

3) POSE - Primary Obesity Surgery Endoluminal
   - Full thickness gastric wall plication

Space occupying Devices

1) Intragastric Balloon - Radiopaque

Malabsorptive procedures

1) Endobarrier - Duodenojejunal bypass - impermeable sleeve placed just beyond hepatopancreatic duct - extending into proximal jejunum
   - → Barrier to mucosal absorption of nutrients
   - → Delays mixing of food & hepatobiliary-pancreatic secretions
   - → faster food transit to jejunum
   - → can be removed endoscopically

Surgeblight chandanasri@gmail.com

GASTROINTESTINAL STROMAL TUMORS

GISTs are gastrointestinal mesenchymal neoplasms which arise from the Interstitial cells of Cajal - components of the GI autonomic nervous system that serve as 'PACE MAKERS' (propagation of intrinsic slow wave gut peristalsis)

Interstitial cells of Cajal can also be found in GENITOURINARY SYSTEM, PORTAL VEIN, PANCREA

GISTs can be found anywhere in the GI tract from ESOPHAGUS TO INTERNAL ANAL SPHINCTER

LOCATIONS OF GIST

1) Stomach - 56%
2) Small Bowel - 32%
3) Colon & Rectum - 6%
4) Esophagus - < 1%
5) Others - Mesentery
Pelvis
Pancreas
Liver
Omentum
Genitourinary tract

PATHOLOGY

HISTOLOGICAL TYPES

SPINDLE SHAPED	EPITHELOID	MIXED
70%	20%	10%

Rarely - may contain myxoid stroma, NE features, signet ring variant, lymphocytic infiltrate

- Receptor Tyrosine Kinase mutations
  - C-KIT mutations - 85%
    - → CD 117
    - CD 34 (70%)
  - PDGF-receptor α mutations - 35%

(C KIT & PDGFRA mutations are mutually exclusive)

- Wild type GIST (WT) - BRAF V600E
  DOG-1
- SDH gene

EPIDEMIOLOGY

- ~ 7-15 cases per million population
- Patients older than 50-60y
- Almost equal M:F, i slight male preponderance
- MOST CASES ARE SPORADIC
- ASSOCIATIONS
  - CARNEY'S TRIAD - Gastric GISTs
    Pulmonary Chondromas
    Extra adrenal Paraganglionomas
    + COMPLEX
    - Esophageal leiomyoma
    - Adrenal cortical adenoma
  - CARNEY-STRATAKIS SYNDROME
    Gastric GIST
  - Hereditary / familial KIT mutations
    Skin hyperpigmentation
    Diffuse hyperplasia of intestinal myenteric plexus
  - Familial PDGFRA mutations - large hands, small intestinal polyps, fibroids, lipomas
  - NF-1
  - Von Hippel Lindau disease

SurgeEight.chandanasri@gmail.com

Clinical Presentation

- Often asymptomatic & discovered incidentally on imaging
  - due to submucosal location, exophytic growth & tendency to displace rather than invade adjacent structures.
- Symptoms depend on location & size of tumor
- Mild pain / bloating / dyspepsia
- palpable tumors ± pressure symptoms
- Acute GI blood loss / chronic anemia - mucosal ulceration
- Peritonitis - due to tumor perforation

EVALUATION

Imaging - CT / MRI

Hypervascular, enhancing masses
Heterogenous - due to hemorrhage / necrosis / cystic degeneration

Endoscopy - submucosal mass ± luminal bulge
Ulceration

EUS - can help differentiate between submucosal mass & impingement by masses from surrounding organs (pancreatic mass / pseudocyst)
- helps guided biopsy / FNAC
- high risk features: IRREGULAR BORDERS
  CYSTIC SPACES
  ECHOGENIC FOCI
  INTERNAL HETEROGENEITY

Chest CT - for staging workup

18-FDG-PET - to characterize masses that are ambiguous on CT
- to monitor response to tyrosine kinase inhibitor therapy
- to detect emergence of drug-resistant clones

Pre-operative biopsy is NOT routinely necessary for a primary, resectable neoplasm suspicious of GIST (Risk of dissemination & bleeding)

INDICATIONS - DDx includes non-operatively treated lymphoma
Pre-op Dx required for targetted NACT
Suspected metastatic disease - before palliative therapy

ENDOSCOPIC BIOPSIES are preferrable to percutaneous biopsies

Surgeonlight chandanasri@gmail.com

STAGING & PROGNOSTICATION

AJCC 8th e

Gastric & Omental GIST

T_x - can't be assessed
T_0 - No 1° tumor
T_1 - ≤ 2cm
T_2 - 2-5 cm
T_3 - 5-10 cm
T_4 - >10 cm

N_0
N_1 - Regional nodes +

M_1 - Distant mets

Grade: Low - ≤ 5 mitoses / sq mm
High - > 5 mitoses / sq mm (50 hpf)

I A	T_1, T_2	N_0	M_0	Low Grade
I B	T_3	N_0	M_0	Low Grade
II	T_1, T_2	N_0	M_0	High grade
	T_4	N_0	M_0	Low grade
III A	T_3	N_0	M_0	High grade
III B	T_4	N_0	M_0	High grade
IV	N_1 / M_1	with any grade / any T		

Other - Small Intestinal, Esophageal, Colorectal, Mesenteric, Peritoneal

T, N, M, Grade staging is same

STAGE GROUPING

I	T_1, T_2	N_0	M_0	Low Grade
II	T_3	N_0	M_0	low Grade
III A	T_1	N_0	M_0	High Grade
	T_4	N_0	M_0	Low Grade
III B	$T_{2,3,4}$	N_0	M_0	High Grade
IV	N_1 / M_1	with any grade / any T		

Risk Stratification Systems

1) NIH, Modified NIH - Tumor size, Mitotic Count (+ location, rupture)
2) AFIP - Tumor size, mitotic count, location
3) MSKCC Nomogram - —11—
4) Joensuu et al - Tumor size, Mitotic count, location, rupture

INDICATORS OF POOR PROGNOSIS

1. Larger tumor size
2. High mitotic index
3. Small bowel & Colorectal GISTs
4. High Ki67
5. Aneuploidy
6. Telomerase expression
7. KIT < exon 9 mutations / exon 11 deletions
8. Margin positivity

Spotlight chandanasri@gmail.com

ASSESSMENT OF MALIGNANT POTENTIAL

BENIGN	PROBABLY BENIGN (<3% progressive)	UNCERTAIN	LOW-MODERATE MALIGNANT POTENTIAL (12-15% progressive)	HIGH MALIGNANT POTENTIAL (49-86% progressive)
<2cm & <5 mitoses/50 hpf	2-5 cm & <5 mitoses/50 hpf	<2 cm & >5 mitoses/50hpf	>10 cm & <5 mitoses/50 hpf 2-5 cm & >5 mitoses/50hpf	5-10 cm & >5 mitoses/50hpf >10 cm & >5 mitoses/50hpf

MANAGEMENT

ACTIVE SURVEILLANCE

- Gastric GISTs <2cm

Stratified as low risk based on clinical, pathological & molecular features

↓

EGD ± EUS every 6-12 m

↓

Intervene if

Growth to >2cm
Irregular extraluminal bodies
- Ulceration
- Heterogenous foci
- Cystic spaces
- Development of symptoms

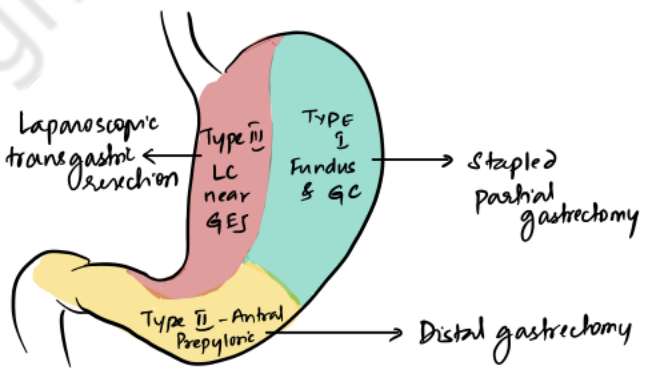
SURGICAL RESECTION

Standard of care for 1°, resectable, localized GISTs

Indications:
- GISTs ≥2cm
- Symptomatic tumors
- All GISTs of non-gastric origin

Goal- R0 resection
Prevention of capsule rupture, spillage & organ preservation when feasible

TYPES OF GASTRIC GIST



SYSTEMIC THERAPY

Imatinib mesylate

NEOADJUVANT CHEMOTHERAPY

- INDICATIONS
  - Locally advanced
  - Large tumors
  - Anatomically difficult sites

4-12 months

ADJUVANT CHEMOTHERAPY

- INDICATIONS
  - >3cm size
  - High risk clinicopathological factors

DEFINITIVE Rx FOR METASTATIC GIST

Other drugs
- Sunitinib (CKIT, PDGFR, BRAF)
- Regorafenib

GIST is generally radioresistant

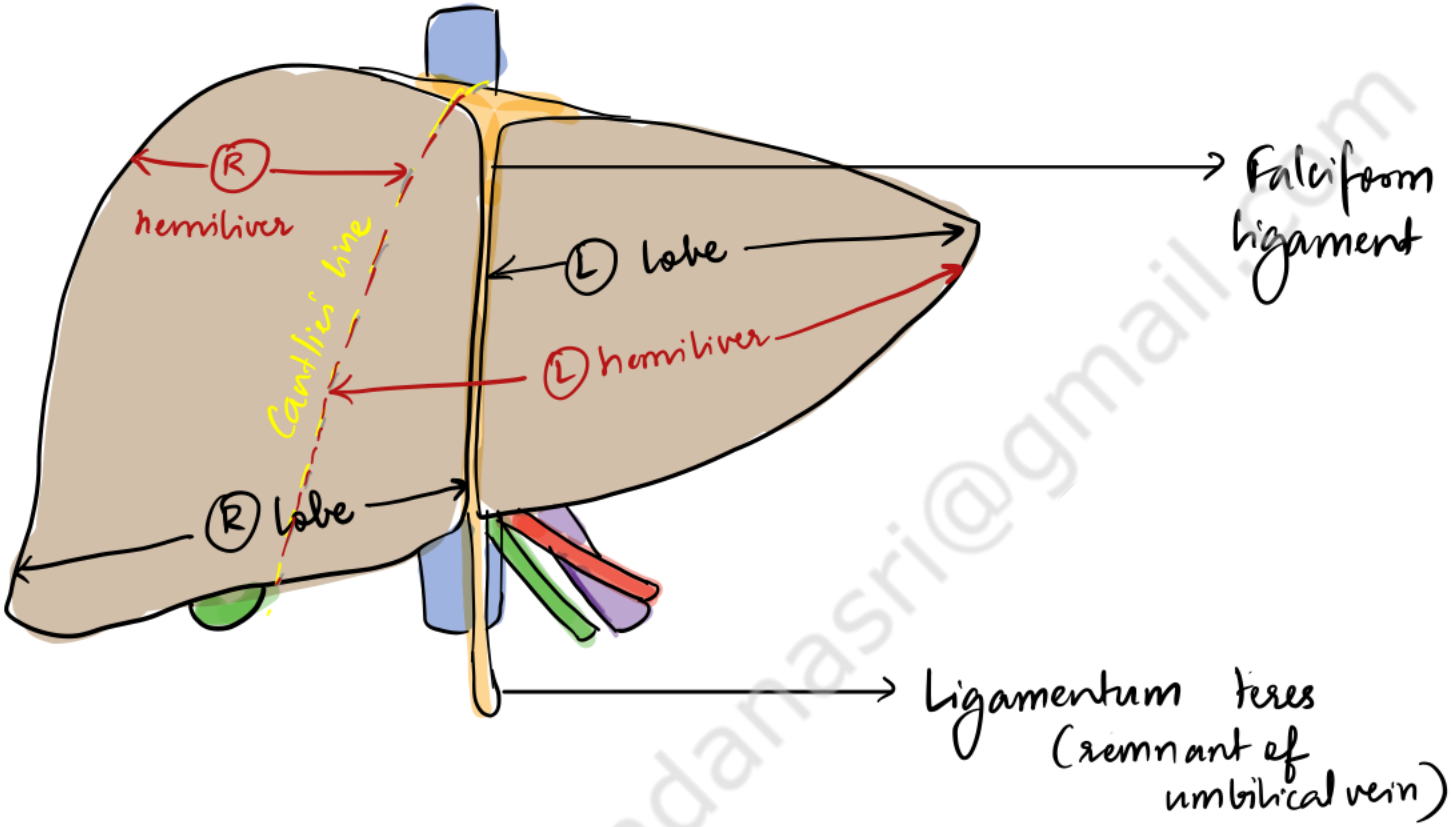
SurgeLight chandanasri@gmail.com

Liver

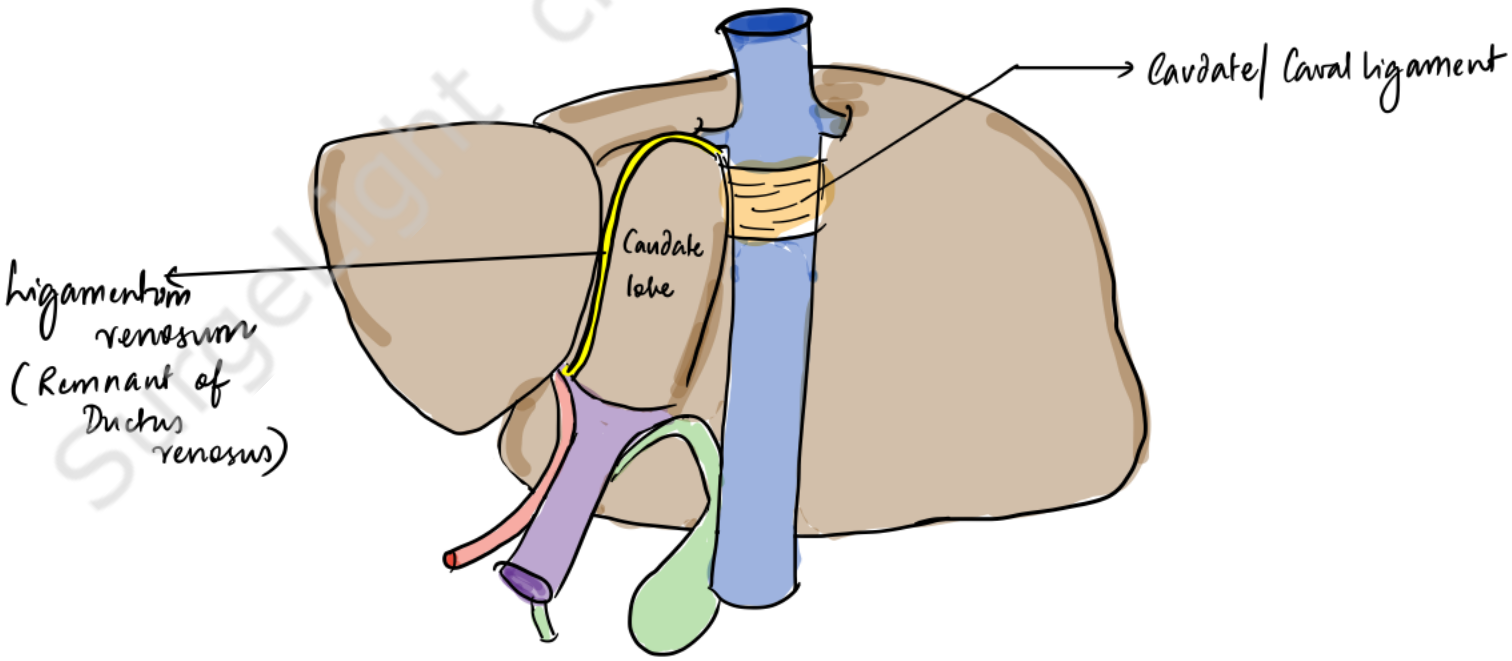
ANATOMY

SURFACE ANATOMY

① ANTERIOR VIEW



② POSTERIOR VIEW



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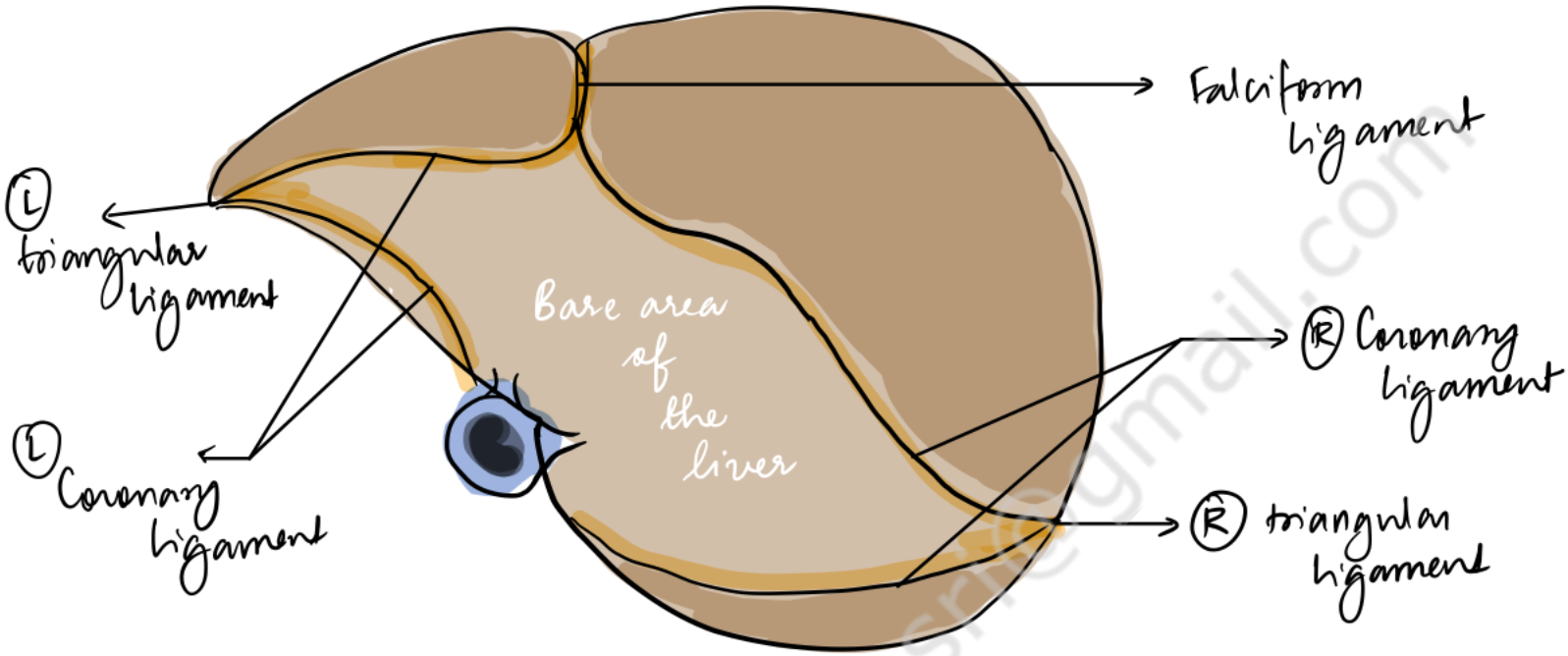
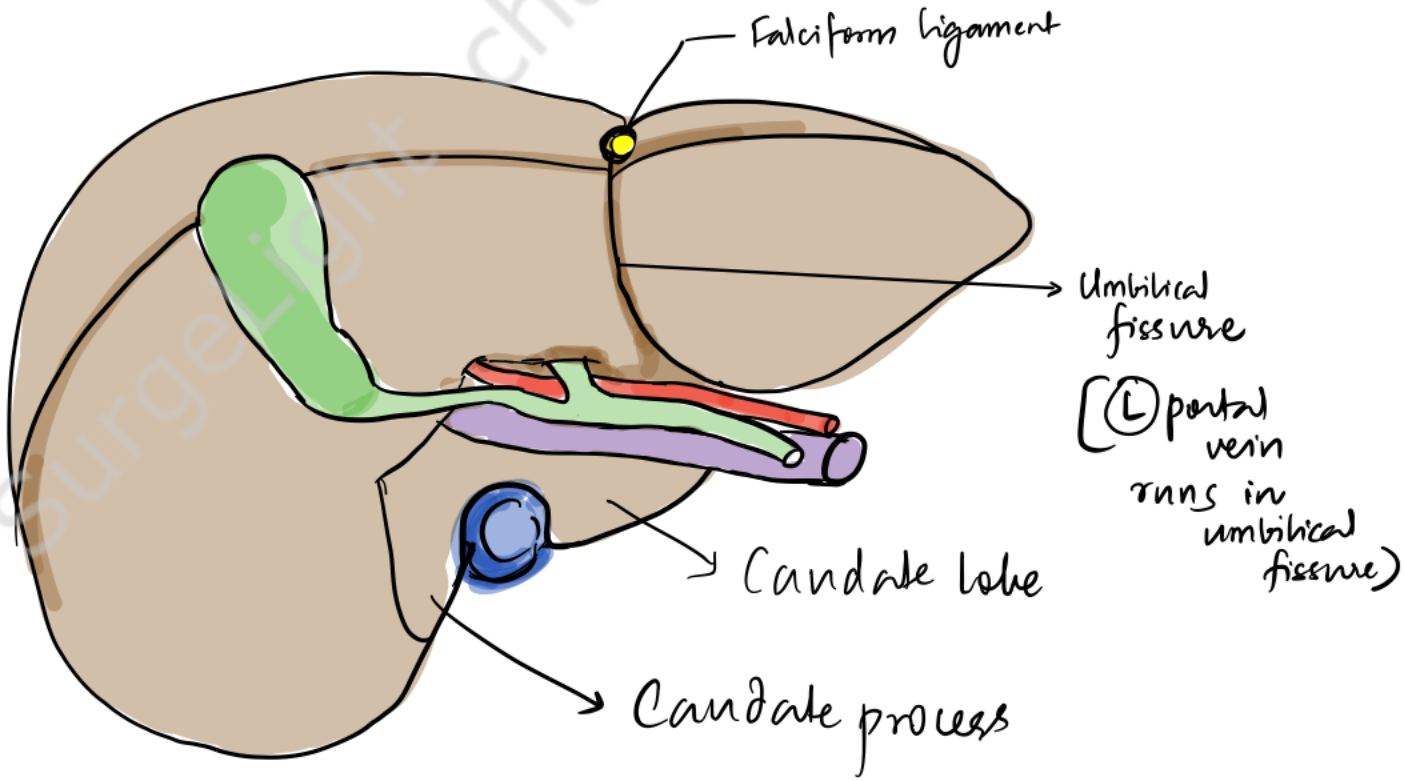
③ SUPERIOR VIEW



④ INFERIOR VIEW



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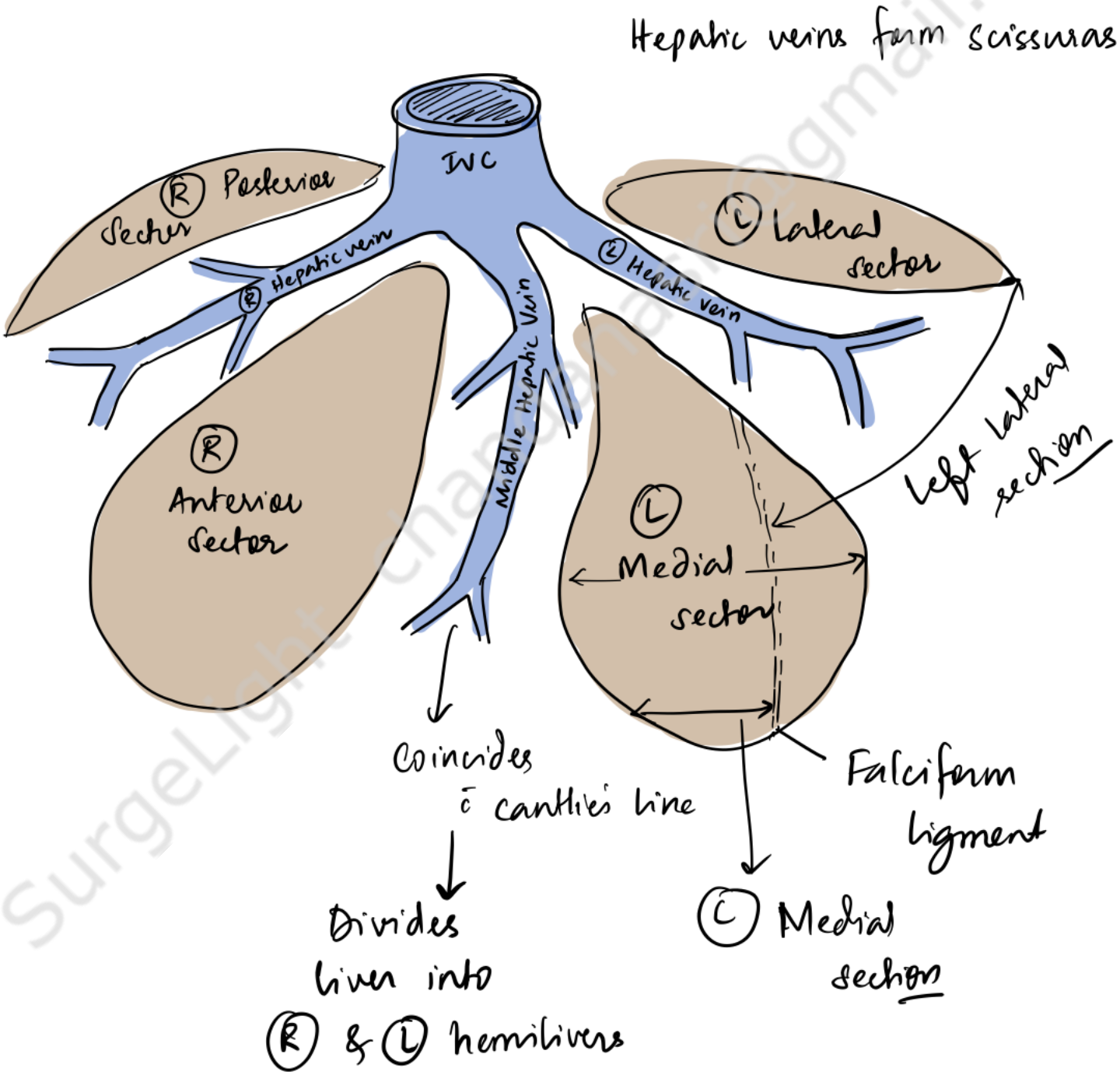
SEGMENTAL ANATOMY

Sectors
Sections
Segments

HEPATIC VEINS → Sectors - Brisbane 2000



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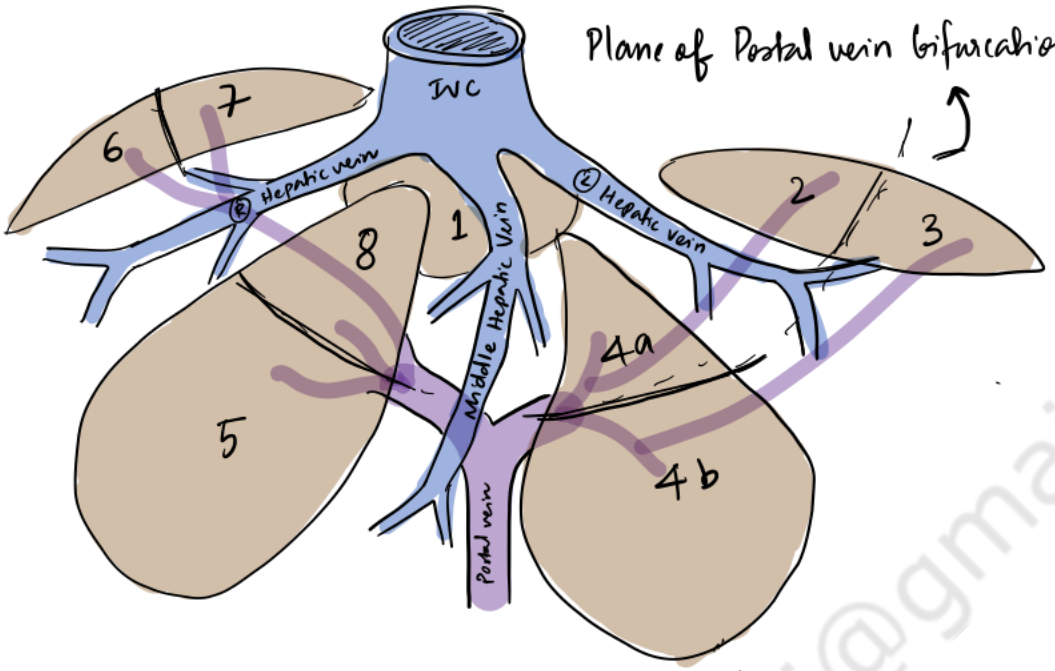
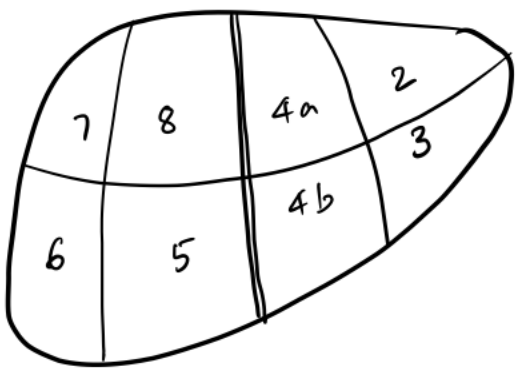
PORTAL VEIN - SEGMENTS - Couinaud



Each segment is supplied by a portal triad / pedicle

- (R) Portal Pedicle
  - ↑ Caudate process
  - Anterior
    - Superior → 8
    - Inferior → 5
  - Posterior
    - Superior → 7
    - Inferior → 6
  - (R) hemi liver

- Caudate lobe ↓
- (L) Portal Pedicle
  - 4a
  - 4b
  - 3
  - 2
  - (L) hemiliver



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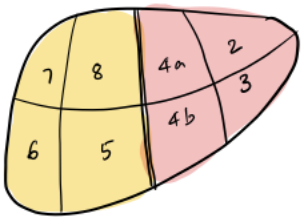
HEPATIC RESECTIONS (Based on Brisbane terminology)

1) SEGMENTECTOMY (Any - I - VIII)

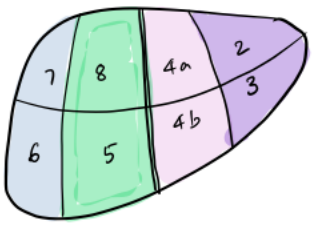
2) HEMIHEPATECTOMIES

- (R) hemihepatectomy 5, 6, 7, 8
- (L) hemihepatectomy 2, 3, 4



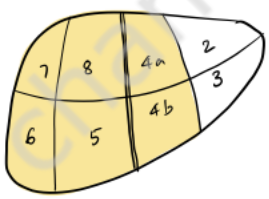
3) SECTORECTOMIES

- (R) Anterior sectorectomy 8, 5
- (R) Posterior sectorectomy 7, 6
- (L) medial sectorectomy 2, 3
- (L) lateral sectorectomy 2, 3

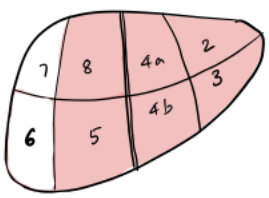


4) TRISECTIONECTOMIES / EXTENDED RESECTIONS

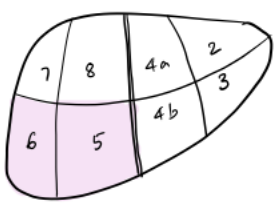
Extended (R) hepatectomy
4, 5, 6, 7, 8



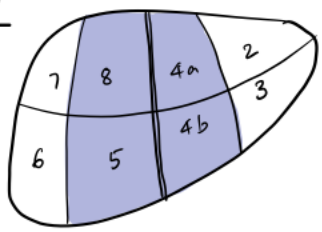
Extended (L) hepatectomy
2, 3, 4, 5, 8



5) BISEGMENTECTOMIES
Any 2 adjacent segments



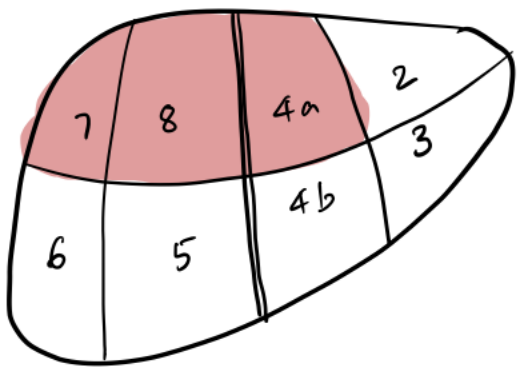
6) CENTRAL HEPATECTOMY
4, 5, 8



- Major resection
  ⇒ ≥ 3 segments
- Minor resection
  ⇒ < 3 segments
- Wedge resection
  → non-anatomic minor resection
- Parenchymal sparing hepatectomy (PSH)
  ↓
  concept of preserving non involved liver tissue

MINIMALLY INVASIVE HEPATECTOMY

FAVORABLE VS UNFAVORABLE LOCATIONS



Posterosuperior - unfavorable

Anterolateral - favorable

SunnyLight Chanuanasri@gmail.com

PORTAL HYPERTENSION

Definition: Portal Hypertension is defined as an elevation in portal venous pressure

Quantitatively, it is defined as ↑ HVPG (Hepatic Venous Portal Gradient)
[Diff between Free HVP & Wedged HVP ≃ Pressure gradient between IVC & portal vein]

Ⓝ Portal Pressure – 6-10 mmHg
Portal Hypertension – Portal pressure ≥ 8 mmHg

Ⓝ HVPG = 1-5 mmHg
Portal HTN HVPG ≥ 5 mmHg → SABISTON
≥ 6 mmHg → SHACKELFORD
Clinically Significant at HVPG ≥ 10 mmHg → Portosystemic collaterals develop
Variceal bleeding at HVPG ≥ 12 mmHg
Clinical decompensation ≥ 16 mmHg
SBP at ≥ 30 mmHg

CAUSES

PORTAL VENOUS PRESSURE = PORTAL FLOW × RESISTANCE TO PORTAL FLOW

- Portal flow → isolated ↑ in portal flow w/out concomitant ↑ in resistance ⇒ Splanchnic AV fistula (VERY RARE)
- Resistance to portal flow → The causes of portal HTN for practical purposes are causes for ↑ portal resistance

↑ PORTAL PRESSURE

PREHEPATIC

1) Portal vein thrombosis (EHPVO)
   - UMBILICAL SEPSIS
   - HYPERCOAGULABLE STATES
   - PANCREATITIS / TUMOR
2) Splenic vein thrombosis
   - Pancreatitis
   - Ca Body / tail of pancreas
3) Extrinsic portal vein compression / invasion
4) AV fistula
   - HEPATIC A – PORTAL VEIN fistula d/t liver biopsy

HEPATIC

- **PRESINUSOIDAL**
  - Schistosomiasis
  - Congenital Hepatic Fibrosis
  - Nodular Regenerative Hyperplasia
  - Idiopathic portal fibrosis
  - GVHD
  - Myeloproliferative disorder
  - Sarcoidosis
- **SINUSOIDAL**
  - Cirrhosis - Viral / Alcoholic
  - PBC, PSC, AIH
  - Metabolic
- **POST SINUSOIDAL**
  - Veno-occlusive disease

POST HEPATIC

1) Budd Chiari Syndrome
2) Veno-occlusive disease
3) IVC web
4) Constrictive Pericarditis
5) Congestive Cardiac Failure

SurgeLight chandanasri@gmail.com

PORTAL VEIN THROMBOSIS

- m/c cause of Non cirrhotic portal hypertension | Extrahepatic portal venous obstruction
- usually occurs due to pro-thrombotic disorders
- clot may involve splenic vein & SMV
- occlusion may be total or partial

↓ Chronicity

Development of collaterals
(Eg: Cavernomatous transformation of portal vein)

Management

- Anticoagulation
  INR - 2-3
- Portal cholangiopathy
  - Biliary stenting
  - TIPSS / surgical shunts
- SURGERY
  - Splenic vein thrombosis → Splenectomy
  - Diffuse thrombosis of portal, mesenteric & splenic veins – Devascularisation procedures

  → In case of variceal bleeding

SurgeLight chandanasri@gmail.com

BUDD CHIARI SYNDROME

Congestive Hepatopathy resulting from obstruction of Hepatic venous outflow

- ABDOMINAL PAIN, ASCITES, HEPATOMEGALY
- OTHER FEATURES OF PORTAL HTN

1° ↓ Endoluminal venous thrombosis

- Hematological disorders
  - Polycythemia vera
  - Paroxysmal nocturnal hemoglobinuria
  - Essential thrombocytopenia
  - Leukemias & Lymphomas
  - Protein C, S, antithrombin III deficiency
  - APLA, Factor V Leiden mutation
- Oral contraceptives
- Pregnancy & post partum
- Connective tissue disorders

2° ↓ External compression / infiltration

- Malignant neoplasms
  - HCC
  - RCC
  - Adrenal malignancy
  - IVC Leiomyosarcoma
- Infections
  - Amebic liver abscess
  - Aspergillosis
  - Gumma (syphilis
- Iatrogenic - IVC filter dysfunction
  - TIPSS displacement

OBJECTIVES OF THERAPY IN BUDD CHIARI SYNDROME

1) Remove the cause of venous thrombosis
2) Relieve the high pressure & congestion within the liver
3) Prevent extension of venous thrombosis
4) Reverse the massive ascites

℞

1) Anticoagulation
2) Thrombolytic therapy ± / ± ant angioplasty
3) TIPSS
4) Surgery
   - Side-to-side porto caval shunt (SSPCS)
   - Mesoatrial shunt
   - Combined SSPCS and Caval atrial shunt
   - Surgical removal of thrombus
   - Liver transplantation

VENO-OCCLUSIVE DISEASE

- involves sinusoids, central & sublobular veins within the liver, rather than hepatic veins
- SUBENDOTHELIAL SCLEROSIS OF SINUSOIDS & VEINS 2° TO ENDOTHELIAL INJURY caused by TOXINS

↓ Diffuse fibrosis
↓ Cirrhosis

Bush teas
Chemotherapy
Bone marrow transplant

℞ - Withdrawal of causative agent
SSPCS, TIPSS

Surgeonlightchandanasri@gmail.com

PORTO-SYSTEMIC COLLATERALS

	PORTAL		SYSTEMIC	RESULT
1	Left gastric (Coronary) vein, short gastric veins	c	Azygous veins	- Esophageal varices
2	Recanalised Umbilical vein (from left portal vein	c	Epigastric veins	- Caput medusae
3	Superior hemorrhoidal veins	c	Middle & Inf Rectal V	- Rectal varices
4	Portal vein branches	c	Inferior phrenic veins	- Bare area
5	Colonic veins (Retroperitoneum)	c	Body wall veins	

WORK-UP

INVESTIGATIONS

ENDOSCOPY

Varices
- size, extent, bleeding, risk

Portal Gastropathy

IMAGING

- Doppler USG - Size & flow through the Portal vein
  Liver morphology
- CT - liver parenchyma
  arterial & venous pattern
- HVPG - to assess severity, prognosis, response
- Angiography - evaluate arterial & venous flow

LIVER FUNCTION

LFT
CBC
- Thrombocytopenia

Hepatitis
Metabolic Panel

↓

Calculate Child - Turcotte - Pugh Score

Bilirubin
Encephalopathy
Ascites
Albumin
Prothrombin time

COMPLICATIONS OF PORTAL HYPERTENSION

1. Variceal Hemorrhage < Gastroesophageal Gastropathy
2. Ascites
3. Splenomegaly
4. Portopulmonary Hypertension
5. Hepatopulmonary syndrome
6. Hepatorenal syndrome
7. Hepatic encephalopathy
8. Portal biliopathy (c ETHPVO)
9. Hepatic hydrothorax
10. Cirrhotic cardiomyopathy

somelight chandanasri@gmail.com

MANAGEMENT OF COMPLICATIONS

★ VARICES

- **ACUTE VARICEAL HEMORRHAGE**

1) Resuscitation
   - Fluids
   - Blood transfusion

2) Pharmacotherapy
   - Antibiotic prophylaxis
   - Somatostatin / Octreotide
   - Vasopressin

3) Endoscopy: Banding > Sclerotherapy

4) Tamponade - Sengstaken Blakemore tube

5) Interventional approach - TIPSS

6) Operative - Esophageal transection
   Portocaval shunt

- **PREVENTION OF RECURRENT VARICEAL HEMORRHAGE**

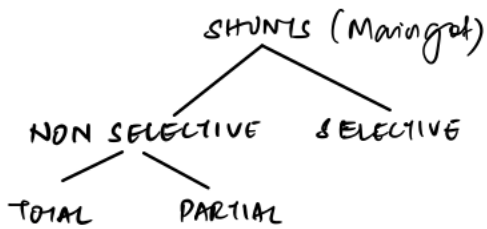
Rebleed risk ~ 40%.

1) Pharmacotherapy
   - β blockers
   - Nitrates - Isosorbide mononitrate

2) Endoscopic therapy - Band ligation > Sclerotherapy

3) TIPSS

4) Surgery -



- Non shunt procedures
  Devascularisation procedures - Sugiura procedure
- Splenopneumopexy
- Liver transplantation

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TIPSS (Transjugular Intrahepatic Portosystemic Shunt)

MELD Score > Child Turcotte Pugh Score in predicting post TIPSS mortality

- An intrahepatic channel is created radiologically between hepatic vein and portal vein in order to reduce portal pressure
  → functionally behaves like a non-selective shunt

- **INDICATIONS**
  - Refractory acute variceal bleeding (gastric / esophageal)
  - Secondary prevention of
    - Portal hypertensive gastropathy
    - Refractory ascites
    - Hepatorenal syndrome
    - Hepatic Hydrothorax
    - Budd chiari Syndrome, Hepatic Veno-occlusive Disease
    - Hepatopulmonary Syndrome
  - → As a bridge to transplantation in advanced liver disease & severe Portal HTN

CONTRAINDICATIONS

RELATIVE
1) HCC (especially if central
2) Obstruction of all hepatic veins
3) Portal venous thrombosis
4) Severe coagulopathy (INR > 5)
5) Thrombocytopenia (< 20,000/μL)
6) RV DD
7) Moderate pulmonary HTN
8) Hepatic encephalopathy

ABSOLUTE
1) Congestive Cardiac Failure
2) Multiple Hepatic Cysts
3) Unrelieved biliary obstruction
4) Uncontrolled infection / Sepsis
5) Severe pulmonary HTN (PAP > 45mmHg)
6) As a means of primary prevention of variceal bleeding
7) Severe tricuspid regurgitation

Procedure:

→ Cross sectional imaging - for hepatic vascular anatomy, portal vein thrombosis

→ Ⓡ IJV > Ⓛ IJV access ⟶ measure Ⓡ atrial, free hepatic & wedge hepatic pressure

→ Cannulation of a suitable branch of Ⓡ hepatic vein → balloon occlusion catheter → wedged hepatic venography ± CO_2 / dilute contrast

↓

Visualisation of portal vein

↓

Needle passes from RHV through liver parenchyma into portal vein under fluoroscopic guidance → aspirate → gush → venography → confirm

↓

Pass guidewire through access needle into portal vein → Splenic/SMV
→ measure the pre-shunt PSPG

↓ track dilatation ± balloon

introduce a PTFE sheathed stent

↓

Measure post TIPSS pressure (should be < 12mmHg) (or fall in HVPG > 20%.)

↓

Can also embolise varices post TIPPS

SurgeLight.chandanasri@gmail.com

COMPLICATIONS FOLLOWING TIPSS

1) TIPSS dysfunction
   due to occlusion - due to pseudointimal hyperplasia
   ↓
   reduced by using PTFE sheathed stent instead of bare metal stent
2) Stent migration into IVC
3) Transcapsular puncture
4) Intraperitoneal bleed
5) Hepatic infarction
6) Hemobilia
7) Fistulae
8) Sepsis
9) Encephalopathy < worsening of pre-existing / new onset } 20-30%.
10) Hemolysis

DIPS - Direct Intrahepatic Portocaval Shunt

- modification of TIPSS

Direct access of PV from IVC via caudate lobe

useful in patients ō challenging anatomy
→ Budd chiari Syndrome
→ Portal vein thrombosis
→ Hepatocellular carcinoma

BRTO- Balloon-occluded Retrograde Transvenous occlusion

Adjunct / Alternative to TIPSS in isolated gastric varices

via transfemoral / transjugular approach, (L) renal vein is approached
↓
Portosystemic gastro-renal anastomosis visualised, embolised / sclerosed

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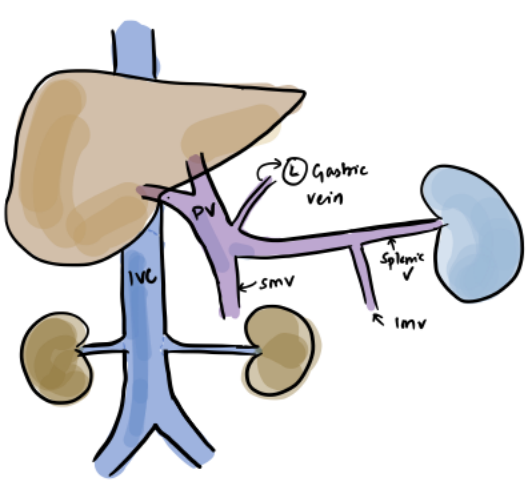
SHUNT SURGERIES

NON SELECTIVE SHUNTS

① END TO SIDE PORTO-CAVAL SHUNT → ECK SHUNT



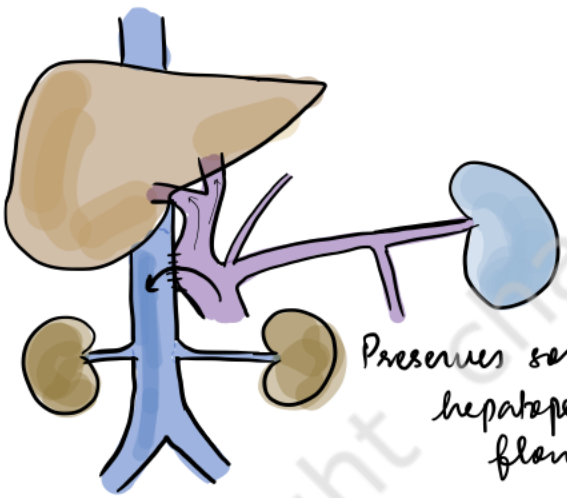
- Completely decompresses the portal system into vena cava

Resolution of varices

Hepatic encephalo-pathy

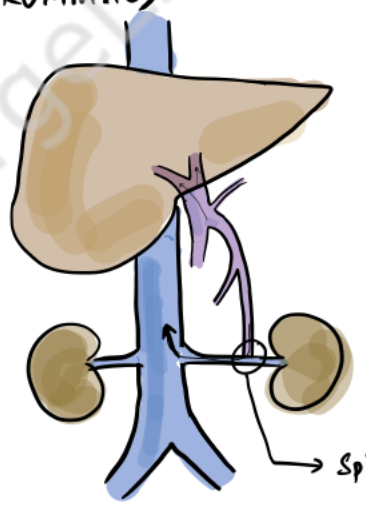
- Completely interrupts portal inflow to liver → early decompensation

② SIDE-TO-SIDE PORTOCAVAL SHUNT



Preserves some degree of hepatopetal portal flow

③ CONVENTIONAL SPLENORENAL SHUNT (PROXIMAL)



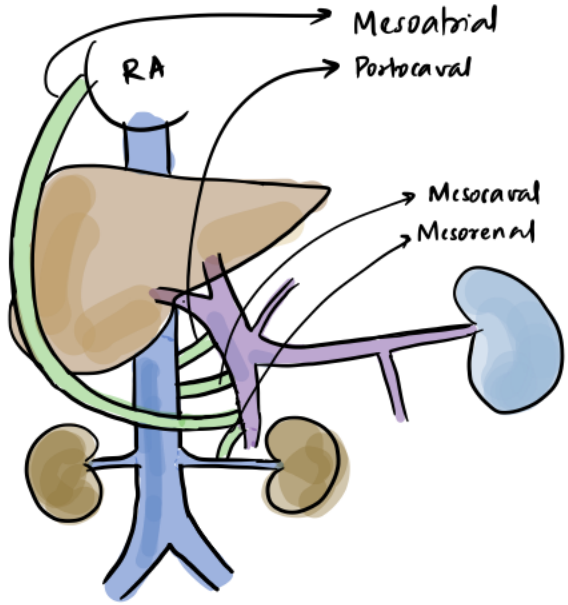
Splenectomy

Might help in mitigating hypersplenism

Splenic vein to renal vein

End to side splenorenal shunt may be done ± splenic preservation

④ INTERPOSITION SHUNTS



The caliber of the interposition grafts decides whether the shunt behaves as a non-selective shunt or a partial shunt

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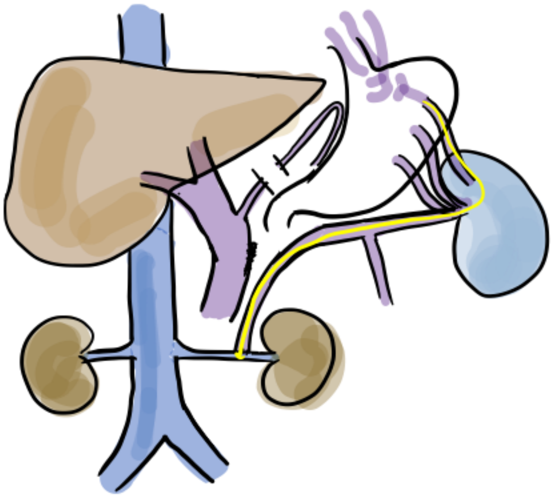
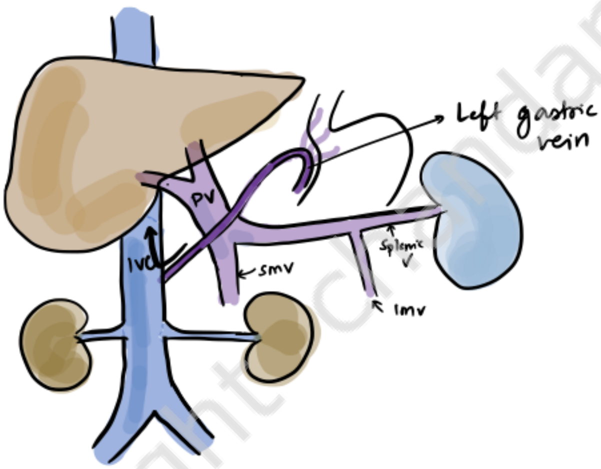
SELECTIVE SHUNTS

1) DISTAL SPLENORENAL SHUNT (Warren shunt)



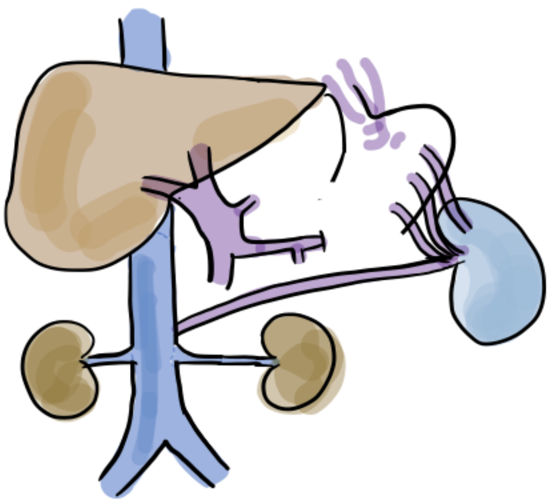
- Selective variceal decompression via short gastric veins
  ↓
  Spleen
  ↓
  Splenic vein
  ↓
  Renal vein
  ↓
  IVC
- Hepatopetal portal flow maintained via SMV to portal vein
- Ligation of (L) Gastric vein, Umbilical vein, gastro-epiploic vein to prevent shunting of hepatopetal flow

2) INOKUCHI (L) GASTRIC VENOUS - CAVAL SHUNT



End to side anastomosis of LGV to IVC c̄/ cont autologous vein graft

3) SPLENOCAVAL SHUNT



End to side anastomosis of Distal splenic vein to IVC c̄/ cont PTFE interposition graft

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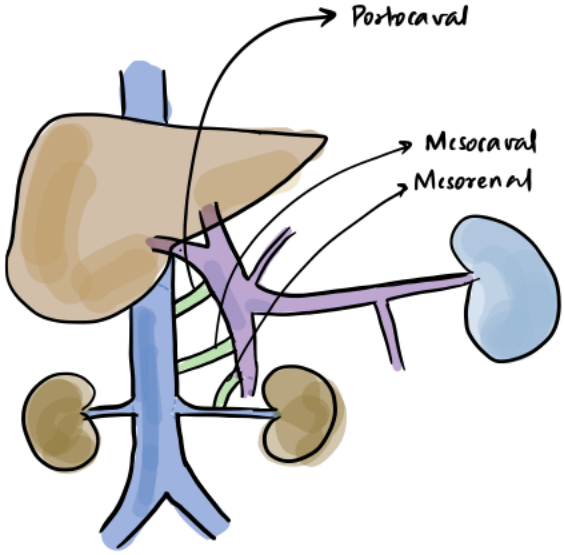
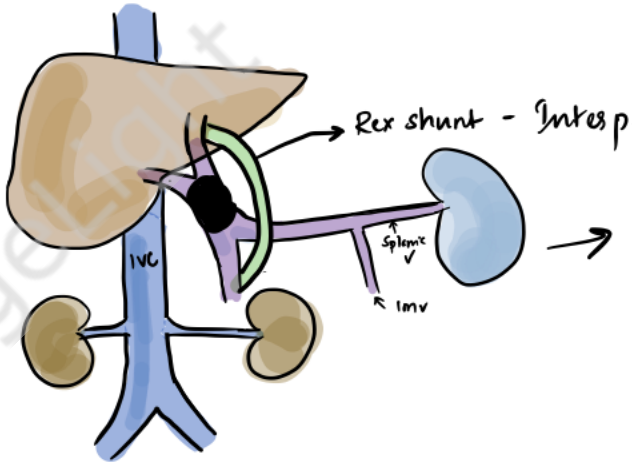
PARTIAL SHUNTS



The caliber of the interposition grafts decides whether the shunt behaves as a non-selective shunt or a partial shunt

PORTOPORTAL SHUNT - Rex Shunt

Done in EHPVD to restore Hepatopetal portal flow



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Liver Trauma

- 2nd m/c abdominal visceral organ injury after spleen

Mechanisms of injury

1) Compression - direct parenchymal damage
2) Shearing forces - tearing of parenchyma
   Vascular disruption, Biliary tree disruption
   Disruption of ligamentous attachments
3) Penetrating trauma - direct laceration of liver parenchyma
   adjacent tissue contusion
   Vascular & biliary tree disruption

AAST Liver injury Grading

I

Hematoma
Subcapsular
<10% SA

Laceration
<1cm depth
Capsular tear

II

Hematoma
10-50%
Subcapsular
<10cm diameter

Laceration
1-3cm depth
Capsular tear
<10cm length

III

Hematoma
>50%
Subcapsular or parenchymal
>10cm diameter or expanding

Laceration
>3cm parenchymal depth

IV

Parenchymal disruption 25-75%
1-3 coinand segments

V

Parenchymal disruption >75%

>3 coinand segments of a single lobe

Vascular -
Juxtahepatic venous injuries - Hepatic veins
- Retrohepatic vena cava

VI

↓
Vascular
↓
Hepatic avulsion

SurgeLight.khandakasri@gmail.com

Investigations

Hb
Group & type

FAST
CT
Stage the trauma

Hemodynamically stable – conserve; close watch for signs of rebleeding / complications
↳ even in higher grade (except major vascular)

Hemodynamic instability ± ongoing bleeding – immediate laparotomy
→ Gr VI – Hepatic autotransplant

Defer immediate repair of extrahepatic biliary tree injuries

BLEEDING HEPATIC TRAUMA

MAJOR BLEEDING

Perihepatic packing
↓
Resuscitate
↙ correct coagulopathy acidosis

Retrohepatic caval injuries not actively bleeding → DO NOT EXPLORE
↓
Pack

Minor bleeding

- Topical hemostatic agents
- Suture hepatorrhaphy
- Electrocautery / Argon beam

BLEEDING CONTROLLED

Damage control surgery
Consider angioembolisation

BLEEDING NOT CONTROLLED

- Pringle Maneuver – hepatoduodenal ligament encircled ± vessel loop / vascular clamp to occlude hepatic blood inflow from hepatic artery & portal vein

(Hepatic venous bleeding will continue)

↓ **BLEEDING NOT CONTROLLED**

↓

SELECTIVE HEPATIC ARTERY LIGATION (SHAL)

↓

Consider vascular isolation ± shunting procedure

↓

SHROCK ATRIOCAVAL SHUNT + PRINGLE MANEUVER

↓

Allows repair of vena caval / hepatic veins

BLEEDING CONTROLLED

- Selective vessel ligation
  Omental pack

LOBECTOMY / RESECTIONS

- Temporary closure
- Second look surgery

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Management of Complications arising during conservative management / Post-op

- **HEMOBILIA** - bleeding from the liver expressed from the biliary tree
  occurs days to weeks after injury
  ↓
  Melena > Risk UGI bleed
  - Jaundice / ↑ Bilirubin

Dx - Angiography ⟶ Rx - Selective angioembolisation
↓ Failure
Operative management

- **BILHEMIA** - abnormal communication between biliary duct & blood vessel
  Bile flows into venous system - profound jaundice

Rx - selective embolisation of vessel
+
decompression of biliary system by ERCP + Stenting

SurgeLight chandanasri@gmail.com

LIVER TUMOURS

Benign Hepatic Neoplasms

Cavernous Hemangioma

- 3-20%
- Vascular malformation or Hamartoma
- Congenital
- F : M - 5:1
- Usually < 5cm
  >10cm – Giant hemangioma

· USG - Hyperechoic
CECT - Rapid peripheral enhancement c̄ centripetal filling
MRI - T_1 - hypointense
T_2 - hyperintense
CT angio - Cotton wool

- Dark purple, soft, compressible lesions, well demarcated, thin capsule ⊕
- Cavernous vascular spaces lined by endothelium separated by connective tissue
- Complications
  - Mass effect
  - Inflammatory Reaction
  - Kasabach Merritt S° - Hemangioma, Consumptive Coagulopathy, Thrombocytopenia

NO RISK OF MALIGNANT TRANSFORMATION

Rx - only if symptomatic
· Surgical enucleation
· Embolisation / Radiation
· Liver transplantation

Focal Nodular Hyperplasia

~1%

Hyperplastic response to a vascular trigger

· F : M - 8:1

usually ≥ 5 cm

· USG - Iso, slightly diff. echogen
CECT - Homogenous enhancement on arterial phase, central supply, sparing of central scar
- Tc99m Sulfur -
- Angiography SPOKE WHEEL

- Lacks a capsule
- central scar ⊕

- Normal hepatocytes in thickened plates, Kupffer cells, fibrous bands

Subtypes: Telangiectatic | Hyperplastic | Adenomatous | c̄ Atypia

Complications - Mass effect, Torsion if pedunculated

NO RISK OF MALIGNANT TRANSFORMATION

Rx - Resection if symptomatic or pedunculated

Hepatocellular Adenoma

< 0.05%

- a/w steroid hormones, NASH, Glycogen storage disorders

· F : M ~ 10:1
Young & child bearing age

MRI - IOC

Well circumscribed mass pseudocapsule
- Areas of lipid rich tissue
- hemorrhage, necrosis, calcifications

4 subtypes
- HNF 1α mutated
- β catenin activated
- Inflammatory
- Non Inflammatory

Complications
- Mass effect
- Bleeding
- Inflammatory Reaction

RISK OF MALIGNANT TRANSFORMATION ⊕
Risk factors: Male, Androgen use, >5cm

Rx - Resection
Glycogen Storage - liver transplant

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MALIGNANCIES OF THE LIVER

Cell of Origin	Tumor
Hepatocyte	Hepatocellular carcinoma Fibrolamellar carcinoma Epithelial variant of Hepatoblastoma
Biliary Epithelial cell	Cholangiocarcinoma Biliary cystadenocarcinoma
Endothelial cells	Angiosarcoma Epithelord hemangioendothelioma

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HEPATOCELLULAR CARCINOMA

75% of primary hepatic neoplasms

Risk factors

1) Hepatitis B
2) Hepatitis C
} → account for 75-80% - Hep B - 50-55% of HCC → Cirrhosis need not precede HCC
Hep C - 25-30% of HCC → Cirrhosis always precedes HCC
3) Alcohol
4) Smoking
5) Aflatoxin exposure - mycotoxin causes p53 mutations
6) Vinyl chloride exposure
7) OCP use
8) Diabetes
9) NASH
10) Hereditary hemochromatosis
11) α_1 antitrypsin deficiency
12) Wilson's disease

Protective factors

1) Statins
2) Vaccination against Hep B

Pathogenesis

Multistep progression

DYSPLASTIC NODULES (Nodular lesions >5mm) → Early HCC → Well diff HCC → Mod diff HCC → Poorly diff HCC

Low Grade → High Grade

→ Increased dependence on hepatic arterial & neoarterial supply

GROSS TYPES (Eggel)

- Nodular type → no extranodal extension / no multinodularity
- Massive type → in livers w/ cirrhosis - ↑ spread
- Diffuse type → multiple small lesions → ↑↑ spread

Fibrolamellar variant

Combined HCC & Cholangio

SurgeEight chandanasri@gmail.com

Paraneoplastic Syndromes a/i HCC

Hypoglycemia
Hypercalcemia
Watery diarrhea
Hypercholesterolemia
Erythrocytosis
Thrombocytopenia
Cutaneous
- Seb keratosis
- Pityriasis rotunda
- Dermatomyositis
- Pemphigus foliaceus
- Porphyria cutanea tarda

Imaging

Hypervascular tumors
↓
Arterial phase enhancement
↓
Washout in portal & venous phase

Avoid FNAC if contemplating liver transplantation
↳ risk of peritoneal seeding

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STAGING

Has to consider liver tumor & underlying liver status

AJCC

T - T_1 < <2cm c/i out vascular invasion
<2cm c/i out vascular invasion
>2cm c/i out vascular invasion

T_2 2-5cm / Solitary or multiple → none greater than 5cm
c vasc. invasion

T_3 >5cm - Solitary or multiple

T_4 - involvement of major branch of portal vein/Hepatic vein
Direct invasion of adjacent organs

N_1 - Nodal mets

M_1 - Distant mets

G - 1 - well diff
2 - Mod diff
3 - Poorly diff
4 - undiff

I - T_1 N_0 M_0

II - T_2 N_0 M_0

III < A - T_3 N_0 M_0
B - T_4 N_0 M_0

IV < A - Any T N_1 M_0
B - Any T, Any N, M_1

OKUDA STAGING SYSTEM

	+ve	-ve
Tumor size	≥ 50% of liver	<50% of liver
Ascites	Clinically ⊕	⊖
Albumin	≤ 3g/dL	>3g/dL
Bilirubin	>3mg/dL	<3mg/dL

Stage < I - No +ve
II - 1/2 criteria +ve
III - 3/4 criteria +ve

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CLIP Scoring System

Ca Liver Italian Program

CTP Score

A	0
B	1
C	2

Tumor morphology

Uninodular, extension ≤ 50%	0
Multinodular, extension ≤ 50%	1
Massive / Extension > 50%	2

AFP

< 400 ng/ml	0
≥ 400 ng/ml	1

Portal Vein thrombosis

+	0
-	1

BCLC - Barcelona Clinic Liver Cancer algorithm

Resection in Child Pugh A

- < 5 cm
- or 3 nodules none > 3 cm
- No Portal hypertension

MILAN Criteria for OLP in Hepatocellular Carcinoma

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HYDATID DISEASE

Zoonotic parasitic disease caused by Echinococcus species - a cestode / tapeworm

Causative organisms
- Echinococcus granulosus - most common
- Echinococcus multilocularis
- Echinococcus oligarthus
- E. vogelii

LIFE CYCLE

2 stages: SEXUAL - Adult tapeworm; ASEXUAL - Metacestode

2 Hosts: Definitive host - Dog; Intermediate host - Sheep, Humans (→ dead end host)

1. Adult tapeworm

Definitive host: DOG

Head / scolex; Proglottids → Strobila or body → attached to the villi of the Dog's ileum

lays eggs → Thousands of ova are deposited in the dog's feces

Parasitic embryo releases an oncosphere containing hooklets

ONCOSPHERE / ovum penetrates mucosa & enters portal circulation

Ingested by Intermediate host

Intermediate host: SHEEP or HUMANS (Accidental Intermediate DEAD END Host)

Duodenum

LIVER (m/c (R) lobe seg 7,8)

IVC → LUNG

Intestinal lymphatics → THORACIC DUCT → IJV → (R) heart → Bypasses liver

No human-to-human transmission

2. METACESTODE

(within 3 weeks of seeding) becomes ~3cm in 3 months

- Pericyst (Adventitial reaction)
- Hydatid fluid
- Endocyst: Laminated membrane; Germinal membrane
- Daughter cyst surrounded by brood capsule (ectogenic vesiculation) → lacks PERICYST (Contains protoscolices) ~4,00,000/μL
- Daughter cyst (Endogenic vesiculation)
- SATELLITE CYSTS

AKA, HYDATID CYST

Ingested by dog, metacestode matures into adult worm in 2 months

surgeLint chandanasn@gmail.com

Clinical Features

- Asymptomatic
- Vague upper abdominal features - epigastric pain, vomiting

- COMPRESSION
  - cyst growth - towards Glisson's capsule
    - → Compensatory hypertrophy of remaining liver tissue → Hepatomegaly (palpable mass)
  - Compression of bile ducts → obstructive jaundice
  - Compression of hepatic venous outflow → Budd Chiari Syndrome
  - Presinusoidal portal vein → Presinusoidal PHTN

- INFECTION -
  d/t bacteremia / cystobiliary communication
  presentation similar to pyogenic liver abscess

- RUPTURE INTO BILIARY TRACT → CYSTOBILIARY COMMUNICATION
  - MAJOR
    - obstructive jaundice
    - cholangitis
  - MINOR
    - asymptomatic
    - often detected post-op d/t bile leak

>5mm communication → may find cyst contents in biliary tree

>10cm cyst → very likely to have intrabiliary rupture

- ENTRAPERITONEAL RUPTURE
  - Spontaneous
  - Traumatic
  - → Release of broad cystic fluid into peritoneal cavity
    - → Acute Abdomen
    - → Dissemination of disease - peritoneal hydatidosis (2° hydatidosis)

- RUPTURE INTO BRONCHIAL TREE
  Upper segments (IV a, VII, VIII) → erode through diaphragm
  ↓
  Biliary-Bronchial fistula

- RUPTURE INTO OTHER CAVITIES / ORGANS

SurgeLight chandanesri@gmail.com

EVALUATION

- LFT - impairment usually indicates cystobiliary communication
  compression d/t large cyst
- Eosinophilia, ↑ IgE } Non specific
- Serological tests - Immunoelectrophoresis, ELISA, Blotting } for epidemiology, follow-up after surgery — Casoni test

RADIOLOGY

1) Ultrasound scan → first line →

A) WHO classification for uniform reporting

CL	CE-1	CE-2	CE-3	CE-4	CE-5
• Cystic lesion • unilocular • No cyst wall	• Cyst wall ⊕ • Hydatid sand ⊕	• Multivesicular • Cyst wall • Rosette-like	• Detached laminated membrane • Water-lily sign • Less round (↓ cyst pressure) • Starting to degenerate • may produce daughter cysts	• Heterogenous • Hypo/hyperechogenic • Degenerative contents • No daughter cysts	• Thick calcified wall • Calcification partial to complete
↓	↓	↓	↓	↓	↓
- usually early stage - not fertile - Ddx necessary	Usually fertile	Usually fertile	TRANSITIONAL	usually no living protoscolices	usually no living protoscolices
↓					
Ddx - simple biliary cyst					
ACTIVE STAGE (GROUP-1)			Gp 2	INACTIVE STAGE	

CE 3a - inactive/active
CE 3b - biologically active

B) GHARBI CLASSIFICATION (old)

- I - Pure-fluid collection
- II - Fluid collection ± split wall
- III - Fluid collection ± septae
- IV - Heterogenous appearance
- V - Reflecting thick walls

2) CT scan - Indications:
1) Subdiaphragmatic location
2) Disseminated disease
3) Extra abdominal location
4) Complicated cysts
5) Presurgical evaluation

3) MRCP - in case of cystobiliary communications

SurgeLight chandan@gmail.com

TREATMENT

GOALS
1) Removal of entire parasite
2) Removal of residual cavity
3) Dx & Rx of biliary fistula

OPTIONS - 1) Benzimidazoles
2) Percutaneous Rx - PAIR, PAIR cath, PEVAC
3) Surgery: Conservative / Radical

- Wait & watch - small & asymptomatic cysts

1) Benzimidazoles

Albendazole
Mebendazole
} Impair glucose uptake by parasite

Albendazole 10mg/kg in divided doses - BD ± meal

Objectives
1) Definitive cure → 3-6m course - 80% success, 25% relapse
2) Reduction in cyst viability
3) Preoperative Rx → start 1 week before procedure
4) Perioperative prophylaxis → Post-op
   - uncomplicated: 3-8 wks
   - Complicated: 3-6 months

Indications
- CE 1, 3a < 5cm
- Inoperable / Unwilling for Sx
- Multiple cysts > 2 organs
- Peritoneal cysts
- Prevent recurrence following Sx/PAIR

Contraindications
1) Pregnancy
2) Uncomplicated CE4/5
3) Bone marrow depression
4) Chronic liver Disease

2) Percutaneous modalities - PAIR, PAIR catheterization, PEVAC

↓USG guidance - needle introduced into cyst (PUNCTURE) - as much fluid as possible aspirated

↓ Injection of protoscolicidal agent into cavity

↓ 15 min

Reaspirate as much as possible

PAIR → Needle withdrawn

PAIR catheterization: Catheter not removed ← allowed to drain ×24h ← Fluoroscopic cystogram ← No Cystobiliary communication ← ABSOLUTE ALCOHOL (95%) × 20 min ← Cystogram -ve ← If +, keep drain until <10ml

Reaspirated → Catheter removed

Follow up USG →
- 3m - heterogenous reflections of cyst content
- 5m - obliteration & pseudotumor appearance
- 9m - Loss of echogenicity & disappearance

- **PEVAC** - Percutaneous evacuation of Cyst Content → PAIR catheterisation → replace catheter ± 14-18 Fr stiff sheath, suction & cutting instrument → irrigation ± scolicidal → reaspirate

Indications for Percutaneous methods - CE 1 / CE 3a > 5cm, Relapse after Sx

Contraindications - CE-5, Biliary fistulae

surgeonlightchandana@gmail.com

SURGICAL TREATMENT < Open / Laparoscopic

Indications

1) Removal of large CE2 - CE3b cysts : multiple daughter cysts
2) Single liver cysts situated superficially which may rupture spontaneously or ; PAIR
3) Infected cyst
4) Cystobiliary communication

CIs : Uncomplicated CE4/5
Very small cysts

Pre-op evaluation

CMP, ECG, PT/INR
liver imaging ó triple phase CT
MR cholangio for central cysts close to hilum

SCOLICIDES 70 - 95% ethanol
15 - 20% Hypertonic saline → contact time ≃ 15min
0.5% Cetrimide

SURGERIES

CONSERVATIVE

- Cystectomy / Closed Cystectomy / unroofing

1) Puncture - aspiration
2) Injection - if no contraindication
3) Hydatidectomy - Removing content, (Laminated & germinal layers)
4) Unroofing
5) Oversewing of cut edges
6) Irrigation of cavity ó scolicides if no cystobiliary communication

To rule out cystobiliary communication,
gentle pressure on gall bladder
Intra-op cholangiogram
Transcystic duct rule - methylene blue
obvious biliary orifices should be sutured

Cavity < leave drain / plug ó omentum

POST-OP COMPLICATIONS

1) Biliary fistula
2) Biliary stricture
3) Recurrence

RADICAL

- Radical Cystectomy / Capsulectomy / Total Pericystectomy / Cystopericystectomy
- A plane is created outside the pericyst layer without entering the cyst
- Parasite & adventitial layer are excised en-bloc
- CUSA - Cavitron Ultrasonic Aspirator - may be used to isolate vessels & biliary radicles

Contraindications :
- Cyst impinging on major hepatic veins / IVC / liver hilum

- **Liver resections**
  for Echinococcus multilocularis
  - pedunculated, peripherally placed cysts
  - when remaining liver parenchyma is atrophic
  - when large bile leak cannot be safely managed ó Roux-loop

RARELY INDICATED

Surgeonsignandanasri@gmail.com

HYDATID DISEASE OF THE LUNG

Lung - 2nd m/c organ affected
Ⓡ lung - lower lobes most commonly involved
Mode of spread

- Duodenum ① → Portal circulation → Liver → IVC → Ⓡ heart
  ② → Intestinal lymphatics → Thoracic duct → IJV → SVC → Ⓡ heart
- Direct inhalation of eggs ③ → Lungs

Lung cysts grow faster than liver cysts; Peripherally located cyst - pericyst may be absent
usually single / unilateral; can also be B/L & multiple

Clinical Features

- Asymptomatic → incidentally detected
- Bronchial compression → cough, wheeze
- Intrapulmonary rupture → Fever, chills, Urticaria, Anaphylaxis, Hemoptysis
  Intrabronchial rupture - salty fluid in mouth

Imaging

CxR - Rounded / oval lesions
Crescent / Meniscus sign - Erosion of bronchioles - results in air entering between pericyst & laminated membrane - radiolucent crescent
or
Cumbo / Onion peel sign
↓
Sign of impending rupture
Camellotte / Water-lily sign - when cyst ruptures, endocyst floats in residual cyst fluid

Rx - Surgery - Cystotomy
Capitonnage - surgical closure of cyst cavity
Pericystectomy
Segmentectomy
Pneumonectomy

- Pharmacotherapy in small asymptomatic cysts
- Percutaneous aspiration is generally avoided

BRAIN HYDATID : <2%

- Single → Mode of infestation
- Multiple → Cyst rupture in Ⓛ heart → multiple protoscolices released

Ⓡ heart → Lung → Ⓛ heart → Aorta → Carotid → MCA (m/c)

- very thin pericyst in brain

- Clinical presentation: ICSOL

Rx - Benzimidazoles
En bloc surgical excision

SurgeLight chandan@gmail.com

MIRIZZI SYNDROME

Complicated gallstone disease resulting from the compression of the CHD by a stone **chronically impacted in the neck** of the gall bladder

PATHOGENESIS

Impacted stone

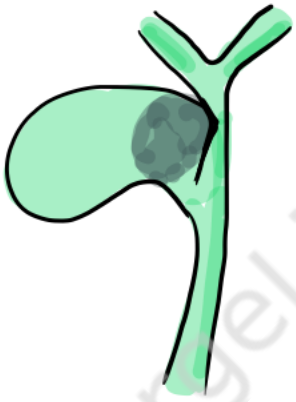
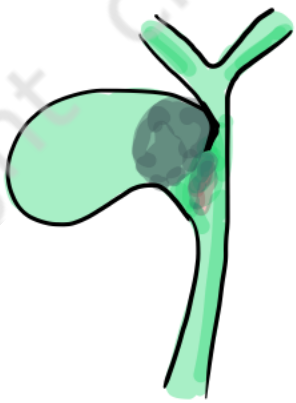
- → chronic Pericholecystic inflammation → Stricture
- → External compression of common hepatic duct

Chronic Pericholecystic inflammation → Extensive fibrosis in CALOT's triangle

Stricture → BILIARY OBSTRUCTION

External compression of common hepatic duct → Pressure necrosis of the intervening bile duct wall → Cholecystocholedochal fistula

TYPES OF MIRIZZI SYNDROME

MCSHERRY TYPE I - No fistula	MCSHERRY TYPE-II - WITH FISTULA		
CSENDES - TYPE-I	CSENDES - II	CSENDES III	CSENDES IV
			
External compression of CHD	< 1/3rd destruction of common wall	1/3rd – 2/3rd destruction of common wall	> 2/3rd – Complete destruction of common wall

SurgeLight chandanasri@gmail.com

PRESENTATION

- Repeated attacks of cholecystitis / Cholangitis
- Obstructive jaundice
- Asymptomatic

EVALUATION

- CBC, LFT
- USG Abd → initial screening
  - stone size, number, location
  - Duct status - dilatation, stones
  - IHBRD
  - Inflammation

R/O
- Ca GB
- Extrahepatic biliary Ca

- ERCP - as a part of evaluation of Obstructive jaundice to s/o & treat CBD stones
  helps demonstrate compression & fistula
- MRCP - accurate delineation of fistula / biliary anatomy
- IOC

MANAGEMENT - SURGERY

Extensive calot's adhesions, fibrosis ⊕

Open GB at Hartmann pouch

↓

Empty GB contents

↓

Identify cystic duct opening (from inside)

↓

Secure cystic duct

Avoid ↑ dissection → subtotal cholecystectomy - safe

Small fistula - partial cholecystectomy - use GB stump to repair duct (Choledochoplasty)

More extensive fistula - Bilioenteric anastomosis - Roux Y HJ

SurgeLight.chandan@gmail.com

CHOLEDOCHAL CYSTS

Congenital lesions of the biliary system

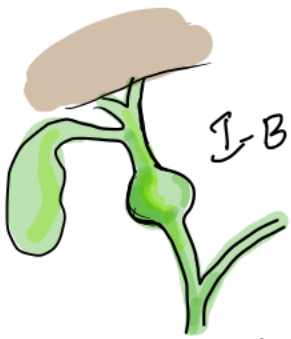
m/c age - <10y

PAIN, MASS, JAUNDICE

CLASSIFICATION - ALONSO-LEJ — TODANI MODIFICATION

TYPE-I-INVOLVEMENT OF EXTRAHEPATIC BILIARY TREE

Cystic dilatation of Extrahepatic Biliary duct

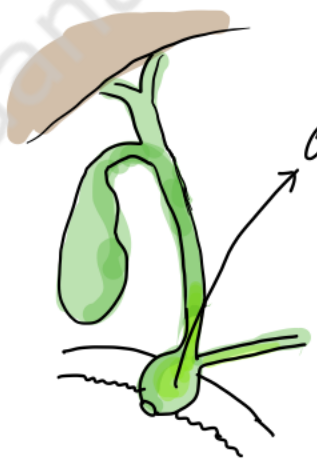


Focal segmental dilatation of extrahepatic biliary duct

Fusiform dilatation of entire extrahepatic biliary duct

TYPE-II

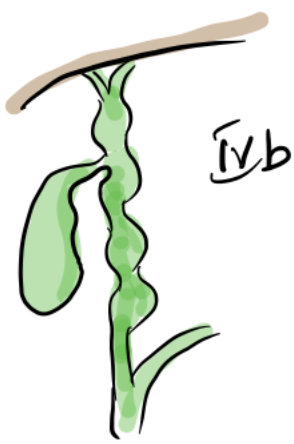
Simple diverticulum of extrahepatic biliary duct



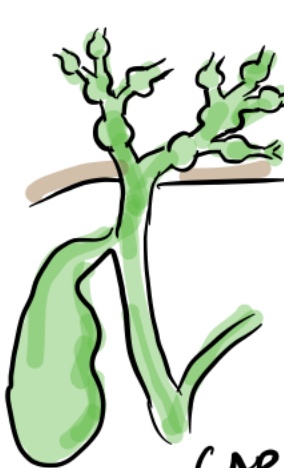
Type III

Cyst / Choledochocele of intramural portion of distal CBD

Intra + Extrahepatic duct Dilatation



Multiple extrahepatic dilatation



Multiple Intrahepatic dilatations

CAROLI'S DISEASE

Sunlight chandanasi@gmail.com

Etiology

Not very clear

→ APBDJ → long common channel / inability of Sphincter of Oddi to regulate flow

↓

mixing of pancreatic juice & bile

↓

Activation of pancreatic enzymes

↓

damage to biliary epithelium

↓

Wall weakening

↓

Dilatation

APBDJ - only found in 50-80% CCs

→ Embryological - purely congenital - d/t overproduction of biliary epithelium

→ Issues i remodelling of ductal plate

Associated anomalies - Colonic / duodenal atresia, imperforate anus, AVM

COMPLICATIONS

① Malignancy - CCs are premalignant

Risk - 10-15%

68% - Type I

Site: Extrahepatic bile duct > GB > IHBD > Liver & Pancreas

② Cholangitis

③ Pancreatitis

④ Secondary biliary cirrhosis

⑤ Spontaneous rupture

⑥ Cystolithiasis

SurgeLight chandanasri@gmail.com

Inv

- USG — Transabdominal
  - EUS - esp for choledochocele
- CT
- ERCP / MRCP / PTC

Management

Complete Excision + Biliary-enteric anastomosis

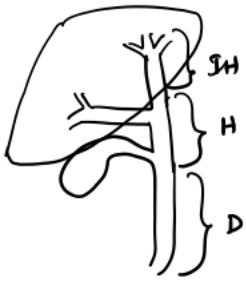
Hepaticojejunostomy > Hepaticoduodenostomy

Caroli's - Liver transplant

SurgeLight chandanasri@gmail.com

CHOLANGIOCARCINOMA

- Primary malignancy of cholangiocytes - epithelial lining of biliary tree



TYPES

- INTRAHEPATIC (10%)
- EXTRAHEPATIC (90%)
  - PROXIMAL / HILAR / KLATSKIN (60%)
  - DISTAL (30%) → Managed as periamp ca

PATHOLOGICAL TYPES:
1) Exophytic → Intrahepatic m/c
2) Infiltrative / Sclerosing / Periductal → m/c → Hilar
3) Nodular
4) Polypoid → Distal CC

INTRAHEPATIC CC - 2nd m/c 1° Liver malignancy after HCC

Predisposing factors:
1) Hepatolithiasis - 10% develop CC
   70% of Intrahepatic CC have hepatolithiasis
2) Caroli's disease
3) Viral hepatitis - HBV, HCV

Presentation → Generally detected incidentally
- Non specific features - mass PA
- hilar involvement - obstructive jaundice

Evaluation - Exophytic masses - large when detected
CT - hypovascular lesion ± central necrosis
Diff from - HCC (αFP ⊘, Ca 19-9 ↑)
Liver mets (No other 1°)

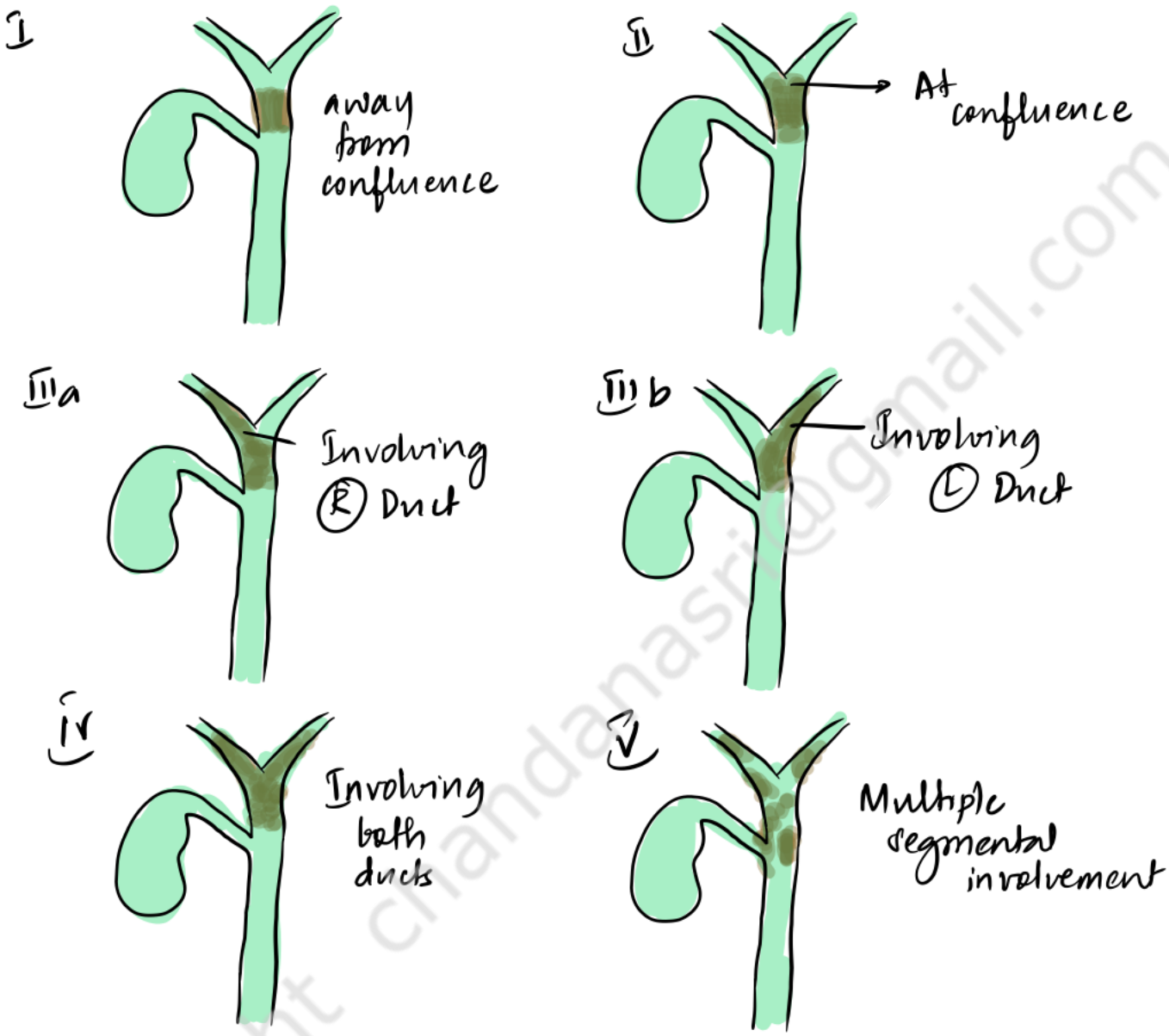
Management
If curative resection is feasible - good survival

Unresectability Criteria:
1) Multiple liver mets (Small satellite nodules OK - But poor px)
2) Distant mets
3) Vascular involvement - main portal vein
4) Metastasis to celiac / RP nodes

SurgeEight chandanrasr@gmail.com

PROXIMAL / HILAR CHOLANGIOCARCINOMA - KLATSKIN TUMOR

BISMUTH-CORLETTE CLASSIFICATION



RISK FACTORS FOR DEVELOPMENT OF HILAR CCs

1) Primary Sclerosing Cholangitis - ~40% CC rate
   CC in the setting of PSC - usually unresectable - usually multifocal
   ~30-50y age
2) Caroli's Disease - risk of 15-20%
3) Bile duct adenomas / Papillomatosis
4) Thorotrast
5) PARASITES - Opisthorchis viverrini

Surgical light chandanasri@gmail.com

PRESENTATION

OBSTRUCTIVE JAUNDICE - 90-98 %.

Cholangitis

EVALUATION

LFT - Obstructive jaundice

CA 19-9 - progressive ↑

USG - Obstruction - invasion / encasement of portal vein

MRCP - Biliary anatomy - atrophy-hypertrophy complex

CT / FDG-PET - for distant mets

ERCP - ? Brush cytology

Rx - Resectable - En-bloc hepatectomy + biliary enteric anastomosis

Pre-op biliary drainage - if FLR < 30 %
↳ PTBD if portal vein embolization is planned

? Liver transplant

NO PROVEN ROLE OF CHEMO / RT in Cholangiocarcinoma !

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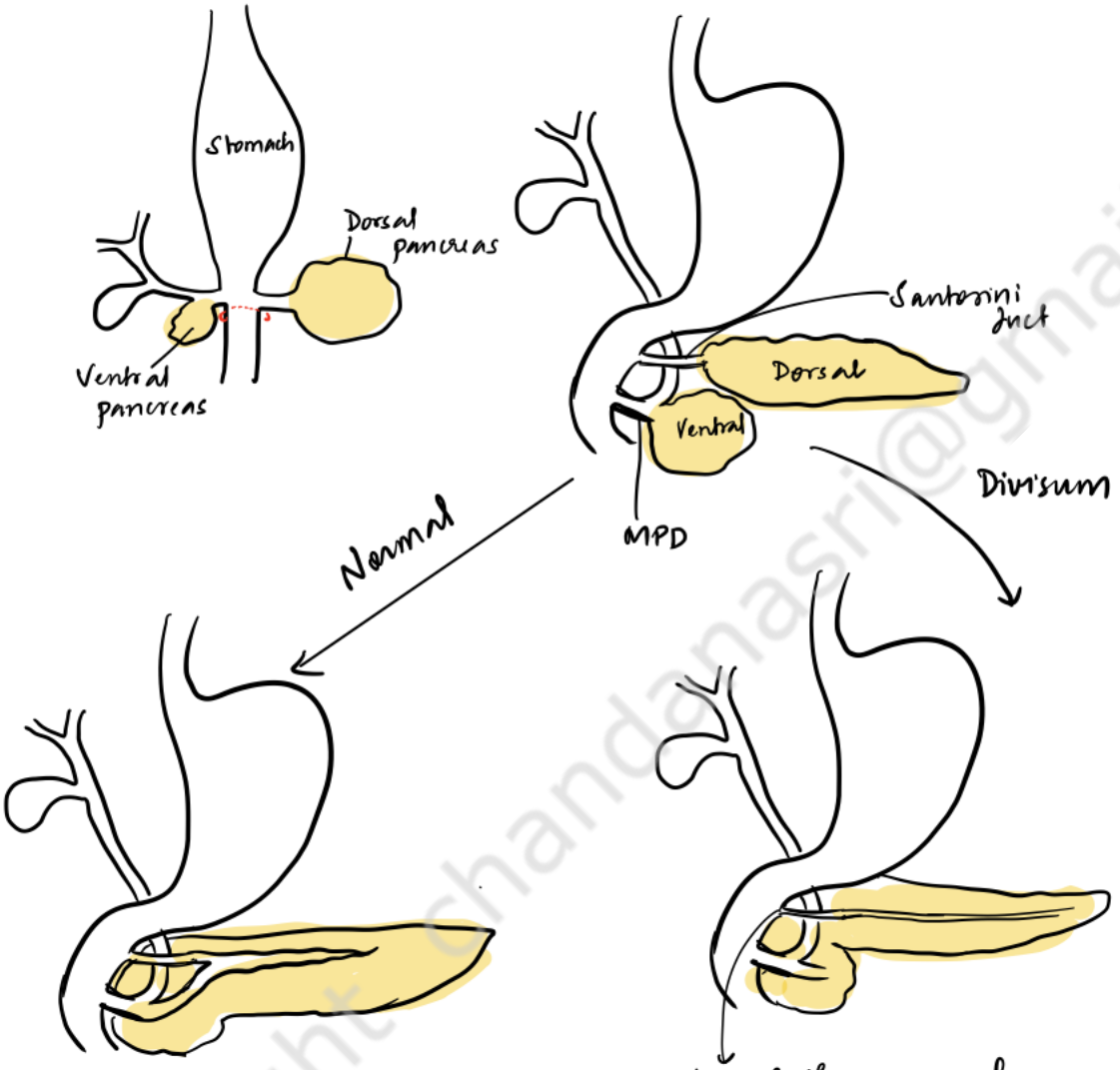
PANCREAS DIVISUM

Congenital anomaly - absence of communication between the MPD (Duct of Wirsung) & Duct of Santorini

EMBRYOLOGY



Majority of the parenchyma is drained into the MINOR AMPULLA via diminutive accessory duct of Santorini

↓

Ductal Hypertension

↓

Pancreatitis

Rx - Ductal Decompression → Lesser papillotomy / Ductal drainage

SurgeLight - chandanasri@gmail.com

ACUTE PANCREATITIS

- inflammatory disorder of the pancreas characterised by edema and necrosis of the pancreatic parenchyma
- presents with abdominal pain (Persistent severe epigastric pain radiating to back) ≥2/3 = AP
  ≥3x ↑ in S. Amylase / Lipase
  characteristic features on imaging (CECT/MRI/USG)

ETIOLOGY

- ALCOHOL
- BILIARY TRACT DISEASE
- Hyperlipidemia
- Hypercalcemia
- Trauma
  - External
  - Surgical
  - ERCP
- Ischemia - Hypoperfusion
  - Atheroembolic
  - Vasculitis
- Pancreatic ductal obstruction
  - Neoplasms
  - Pancreas divisum
  - Ampullary & duodenal lesions
- Infections
- Venom
- Drugs
- Idiopathic

Most cases - self limiting

10-20% - progress

M > F

Alcohol & drug-induced pancreatitis
3rd - 4th decade

Gall stone pancreatitis - older pts

BIMODAL MORTALITY

- Early → within 2 weeks → MODS
- Late → >2 weeks → Septic complications

TYPES

- ACUTE EDEMATOUS INTERSTITIAL PANCREATITIS
- ACUTE NECROTISING PANCREATITIS

BASED ON SEVERITY: MILD
MODERATE
SEVERE

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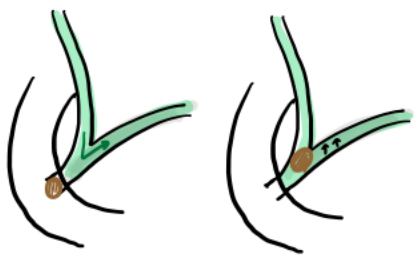
PATHOGENESIS

GALLSTONES

- Common channel hypothesis
  - stone obstructing ampulla forms a common channel for bile & pancreatic enzymes
  - biliary reflux into PD
- Gallstone obstructing PD
  - Ductal hypertension
- transient incompetence of SOD 2/t passage of gall stone → biliary reflux into PD



ERCP

esp if contrast is injected repeatedly & ↑ pressure

(PREVENTED BY RECTAL NSAIDS)

ALCOHOL

Acinar cells metabolise alcohol

↓

- ↑ Secretory activity
- ↑ activation of pancreatic stellate cells

-Proinflammatory pathways (NF-KB)

↓

↑ TNF-α

IL-1

↑ expression & activity of caspases

Tumors

Dyslipidemia

Direct injury due to triglyceride metabolites

DRUGS

HYPERCALCEMIA

Ca^{2+} activates pancreatic zymogens

Hereditary

Trypsin gene & binding protein mutations

PRSS 1

& PINK 1

Colocalisation of ZYMOGEN GRANULES + LYSOSOMES in acinar cells

↓ ← cathepsin B

ACINAR CELL AUTODIGESTION

↓ IL-1, TNF α

Inflammatory cascade

↓

SIRS, MODS

Spotlight chandanasri@gmail.com

COMPLICATIONS OF ACUTE PANCREATITIS

LOCAL COMPLICATIONS

1) Peripancreatic fluid collections
   - Pancreatic Pseudocyst
   - Pancreatic ascites
   - Pancreaticopleural fistula

2) Pancreatic Phlegmon
   - Bowel obstruction, Obstructive jaundice

3) Pancreatic Necrosis
   - severe inflammation
   - ↓
   - failure of pancreatic perfusion
   - ↓
   - NECROSIS

4) INFECTED NECROSIS / ABSCESS
   - Bloodborne / Bacterial translocation from intestine / direct contamination by interventional procedures

5) VASCULAR COMPLICATIONS
   1. - Pseudoaneurysm
      - Pancreatic elastase → vessel wall damage
      - m/c - SPLENIC A
      - SMA, cystic A, GDA
      - Spontaneous rupture
      - ↓
      - HEMORRHAGIC PANCREATITIS
      - (Cullen's sign, Grey Turner's sign, Fox sign)
      - Hemoperitoneum, shock
   2. Vascular thrombosis
      - SPLENIC VEIN - m/c
      - can involve SMV, IMV, Portal V
      - → Splenectomy is Rx

6) PANCREATIC FISTULAS
   - Pancreaticoenteric
   - colic
   - cutaneous

SYSTEMIC COMPLICATIONS

A. Pulmonary
   - Pneumonia, atelectasis
   - ARDS
   - Pleural effusion

B. Cardiovascular
   - Hypotension (2/t 3rd spacing)
   - Pericardial effusion
   - Non specific ST-T changes

C. Hematologic - Hemoconcentration
   - DIC

D. GI hemorrhage - Stress ulcer
   - Varices

E. Renal - AKI
   - Renal artery / vein thrombosis

D. Metabolic
   - Hyperglycemia
   - Hypocalcemia
   - Hypertriglyceridemia
   - Encephalopathy
   - Sudden blindness (Purtscher's retinopathy)

E. CNS
   - Psychosis

H. Fat necrosis
   - Intra-abdominal saponification
   - Subcutaneous tissue necrosis

Sumedh chandanasri@gmail.com

PRESENTATION

PREDICTION OF SEVERITY

① RANSON'S CRITERIA

NON-GALL STONE PANCREATITIS		GALL STONE PANCREATITIS	
AT ADMISSION	During 48h	AT ADMISSION	During 48h
Age > 55y	· Hematocrit fall by >10	Age > 70y	· Hematocrit fall >10 points
TLC > 16,000/μL	· BUN ↑ by >5mg/dL	TLC >18,000/μL	· BUN ↑ by >2mg/dL
RBS > 200mg/dL	· Serum Ca <8mg/dL · Arterial PO_2 <60mmHg	RBS >220mg/dL	· S. Calcium <8mg/dl
LDH > 350 IU/L	· Base deficit >4mEq/L	LDH > 400mg/dL	· Base deficit >5mEq/L
AST > 250 IU/L	· Fluid sequestration >6L	AST >250 IU/dL	· Fluid sequestration >4L

< 3 points → Mild

> 6 points → Severe → 50% mortality

② GLASGOW IMRIE SCALE

③ APACHE-II

④ BISAP - Bedside Index for Severity of Acute Pancreatitis

⑤ Revised ATLANTA classification
- Based on local & systemic complications organ failure

⑥ CT severity index

Points for
- Pancreatic inflammation
- Pancreatic necrosis %

total 10 max

0-3 – mild
4-6 – mod
7-10 – severe

⑦ SOFA score

SurgeLight - chandanasri@gmail.com

MANAGEMENT

Algorithm for the evaluation and management of acute pancreatitis

1. Diagnosis
   - History of abdominal pain consistent with acute pancreatitis
   - >3x elevation of pancreatic enzymes
   - CT scan if required to confirm diagnosis
2. Initial assessment/management (first 4 hrs)
   - Analgesia → use morphine cautiously → SOD spasm
   - Fluid resuscitation → target U/OP
   - Predict severity of pancreatitis
     - Ranson's criteria
     - HAPS score
   - Assess systemic response
     - SIRS score
     - SOFA (organ failure)
3. Reassessment/management (4 to 6 hrs)
   - Assess response to fluid resuscitation
     - mean arterial pressure
     - heart rate
     - urine output
     - hematocrit
   - Determine etiology
     - Ultrasound for gallstones/sludge
     - History of alcohol consumption
     - Laboratory evaluation of other causes
   - MRCP and/or Urgent ERCP if concomitant cholangitis is present
     - not for cholestasis or predicted severe disease per se
   - Transfer to ICU or specialist center as needed
     - Deterioration or failure to respond to initial management
     - Intensive support for persistent organ failure
   - Commence enteral nutrition
     - Once normovolemia restored (usually after 6 hours)
     - Commence via NG tube if no gastric stasis
   - No prophylactic antibiotics or probiotics — given for proven infection
4. Conservative management and monitoring (at least daily)
   - Clinical evaluation
     - Assess cardiovascular, respiratory, and renal function
     - Detect peritonitis and abdominal compartment syndrome
   - Daily C-reactive protein
   - Classify severity (mild, moderate, severe, critical)
   - Detect intolerance of NG EN
     - Advance tube for NJ feeding if needed
     - Consider supplemental parenteral nutrition by day 4
5. Indications for "pancreatic protocol CT scan" (rarely in first week)
   - For significant clinical deterioration and elevated CRP
   - For suspicion of local pancreatic complications
   - For suspected bowel ischemia
   - For acute bleeding (CTa) (if stable enough and consider embolization)
   - For abdominal compartment syndrome
6. Invasive intervention
   - For deteriorating patient with suspected infected local complication
   - "Step up approach" with initial drain guided by current CT scan (percutaneous or endoscopic drainage)
   - Delay for 3 to 4 weeks with intensive care support, if possible
   - If failure to respond or secondary deterioration, repeat CT scan, and select appropriate minimally invasive technique based on available expertise and equipment
     - Video-assisted retroperitoneal debridement or percutaneous nephroscopic debridement
     - Endoscopic transluminal debridement
     - Ongoing large bore drainage and irrigation
7. Indication for laparotomy
   - Failed "step-up approach" for further debridement/drainage
   - Acute abdomen (perforation or ischemia)
   - Severe abdominal compartment syndrome (rarely)

CHOLECYSTECTOMY – TIMING

Early vs Delayed (≥6 weeks)

Early → ↓ rate of recurrent GS Pancreatitis related complications → Lap chole can be performed just before discharge in MILD biliary pancreatitis

Delayed → Preferred in severe necrotizing pancreatitis

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STEP UP APPROACH FOR PANCREATIC NECROSIS

Sterile necrosis : no role of intervention in acute setting → WON → symptomatic → Intervention after acute illness resolves

NECROTISING PANCREATITIS

1-2 weeks

- No improvement
- Worsening

↓

? Infected necrosis

↓

Pancreatic Protocol CECT

- Acute necrotic collections
- Walled off necrosis
- Gas bubbles within collections
  ⇒ Gas forming organisms or fistulation into digestive tract

↓ Equivocal

FNAC of collection → microscopy, culture

IV antibiotics

Carbapenems > Fluoroquinolones

↓

Invasive intervention delayed

↓ Failure to improve

Minimally invasive approaches

1) Lap : Video-assisted Retroperitoneoscopic debridement
2) Endoscopic : Transgastric necrosectomy
3) Percutaneous catheter drainage

↓ Failure

SURGICAL INTERVENTION

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SURGERY FOR ACUTE PANCREATIC NECROSIS (Pancreatic Necrosectomy)

Midline laparotomy

↓

Division of Gastrocolic ligament

↓

Enter lesser sac

↓

Thorough debridement of necrotic tissue & drainage of collections

Precautions: Friable tissue → Vascular & Bowel injury
Blunt dissection > Sharp dissection

↓ options after necrosectomy

1) Closed Continuous Lavage (Beger): Tube drains left in - continuous lavage

2) Closed drainage - cavity packed with gauze-filled penrose drains and closed suction drains

3) Open packing until clean granulating cavity

4) Closure & relaparotomy q2-3d until raw area granulates

ADD FJ for feeding

SurgeEight.chandanasri@gmail.com

CHRONIC PANCREATITIS

- progressive inflammatory process characterised by irreversible destruction of pancreatic tissue

- pain
  exocrine & endocrine pancreatic insufficiency

ETIOLOGICAL CLASSIFICATION

TIGAR-O

Toxic-metabolic - Alcohol, tobacco, hypercalcemia, hyperlipidemia, CKD, drugs, toxins
Idiopathic - tropical
Genetic - PRSS-1, SPINK1, CFTR
Autoimmune - AIP
Recurrent acute severe pancreatitis
Obstructive - divisum, SOD dysfunction, trauma

Histopathology + Etiology - Singer & Chiari

1) Chronic Calcific / Lithogenic pancreatitis - m/c due to alcohol abuse
2) Chronic Obstructive pancreatitis - due to compression/occlusion of proximal ductal system (Pancreas divisum, GS)
3) Chronic Inflammatory pancreatitis - mononuclear infiltration + diffuse fibrosis (autoimmune)
4) Tropical / Nutritional Pancreatitis — Tapioca
5) Asymptomatic Pancreatic fibrosis - Diabetic Exocrine pancreatopathy
6) Idiopathic pancreatitis

PATHOPHYSIOLOGY

Fibrosis - due to activation of pancreatic stellate cells

Stones: Calcium Carbonate crystalls trapped in a matrix of fibrillar material
PSP- Pancreatic stone protein

Duct Disruption

surgeeight.chandanasri@gmail.com

PRESENTATION

1) Pain - midepigastric / LUQ / RUQ radiating to back
steady 'boring' not colicky
persists for hours to days
exacerbated by alcohol, meals

Causes: Ductal hypertension
Retroperitoneal inflammation with persistent neural involvement

2) Pancreatic insufficiency (Burned out pancreatitis)

Exocrine
Diarrhea
Steatorrhea
Malabsorption
Weight loss
↓
Rx - Pancreatic enzyme supplements
Lipase, trypsin

Endocrine
↓
Pancreatogenic Diabetes
(Type 3c Diabetes)
Global loss of insulin, glucagon & Pancreatic polypeptide
Paradoxical combination of
- enhanced peripheral sensitivity to insulin
- ↓ hepatic sensitivity
↓
'BRITTLE DIABETES'

COMPLICATIONS OF CHRONIC PANCREATITIS

1) Pseudocysts
Duodenal / Gastric Obstruction
Infection
Perforation
Erosion into visceral artery
Splenic vein thrombosis

2) Inflammatory mass in head of pancreas
Bile duct stenosis
Portal vein thrombosis
Duodenal obstruction

3) Duct strictures & stones
Ductal HTN & Dilatation

4) Pancreatic Carcinoma

5) Extrapancreatic
↓
- Pancreatic duct leak
ascites fistulae

SurgeRight chandanasri@gmail.com

EVALUATION

A. Imaging — X Ray - calcifications

- Initial - USG

EUS - Duct:
- Ductal dilatation (Diameter >3mm)
- Tortuous pancreatic duct
- Intraductal echogenic foci / stones
- Echogenic duct wall - ductal fibrosis
- Side branch ectasia

Parenchyma of:
- Edema
- Calcifications
- fibrosis

Pseudocysts

- CECT Abdomen - MPD, SBD
  - Cysts
  - Focal pancreatitis
  - Parenchymal status
  - vascular anatomy
- MRCP - Ductal delineation - chain of lakes
- ERCP - invasive - diagnostic + therapeutic
  - Sphincteroplasty
  - Stenting for Ductal HTN

B. Laboratory studies - Tests of pancreatic function

Direct

Enzymes - Amylase, Lipase

↓

aspiration of pancreatic juice from duodenum after
- Nutrient - Lundh test meal
- CCK / secretin stimulation

Indirect - metabolites of compounds digested by pancreatic enzymes
- BENTIROMIDE
- SCHILLING
- FECAL FAT
- ^{14}C-OLEIN

SurgeLight chandanasri@gmail.com

TREATMENT

MEDICAL – Alcohol cessation

- Analgesics – NSAIDs
  ↓
  Gabapentin, SNRI, SSRI, TCA
  ↓
  Opioids
  – Longer acting
  transdermal patch
- Enzymes
  - Vit D, Calcium
- Antisecretory therapy
  – Somatostatin – in chronic abdominal pain d/t Obstructive pancreatopathy
- Neurolytic therapy

  Celiax plexus neurolysis

ENDOSCOPIC THERAPY

Pancreatic / biliary sphincterotomy
Stricture dilatation / stenting
Lithotripsy / stone extraction

- SURGERY (refer notes)

Surgeeight.chandanasri@gmail.com

SURGICAL MANAGEMENT OF CHRONIC PANCREATITIS

Indications

1) Intractable pain ↳ d/t
   - Ductal Hypertension **
   - Pancreatic Neuropathy
     - Infiltration by infl. cells
     - Neural edema
     - ↑ Nerve density
   - Upregulation of Nociceptive markers NGF, BNP, Cytokines
   - Central Neuroplasticity

   & Failed Med Rx
2) CBD - obstruction → CJ / HJ } Whipple's
3) Duodenal obstruction → GJ }
4) Pseudocyst
5) Pancreatic ascites
6) Vascular complications - pseudoaneurysm / thrombosis
7) Suspected malignancy

PRINCIPLES

1) Ductal Decompression
2) Resection of Diseased tissue
   - Inflammatory mass
   - Focal fibrosis
   - Regions suspicious of malignancy
3) Combination of 1) & 2)

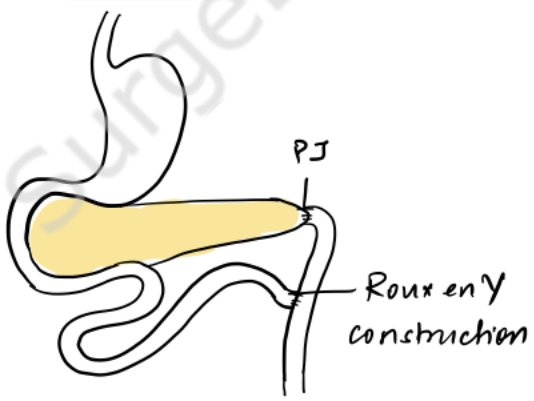
PROCEDURES

1) SPHINCTEROPLASTY: aims to treat OBSTRUCTION (focal) at SPHINCTER OF ODDI
   sclerosing papillitis (d/t pancreatitis / Gallstone)

   - ENDOSCOPIC: dilatation, stenting, lithotripsy, stone extraction
   - SURGICAL → Transduodenal sphincteroplasty
     ↓ incision of septum b/w PD & CBD

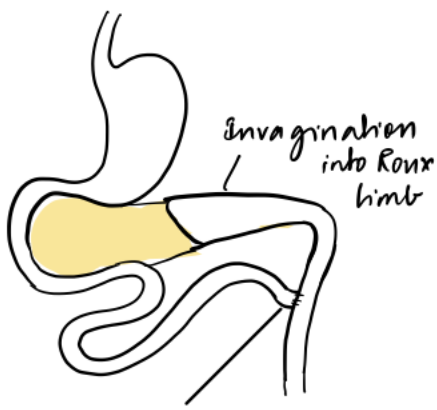
2) DRAINAGE PROCEDURES: Ductal decompression

CATTELL & DUVAL's CAUDAL PJ

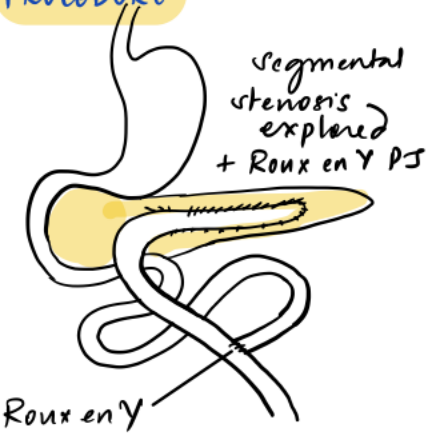


Failure d/t
- Restenosis
- segmental obstruction

PUESTOW & GILLESBY's LONGITUDINAL PJ



PARTINGTON-ROCHELLE MODIFICATION OF PUESTOW PROCEDURE



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3) Resection Procedures

A. Distal Pancreatectomy

– for patients ē focal inflammatory changes localised to body / tail of pancreas ē no significant ductal dilatation

- **Partial Distal Pancreatectomy** → 40–80% Pancreatic Parenchyma resected
- **Frey & Child's '95%' Distal Pancreatectomy** → Rim of pancreas preserved in pancreatico-duodenal groove – preserves CBD & common blood supply
  - done for extensive sclerotic small duct disease

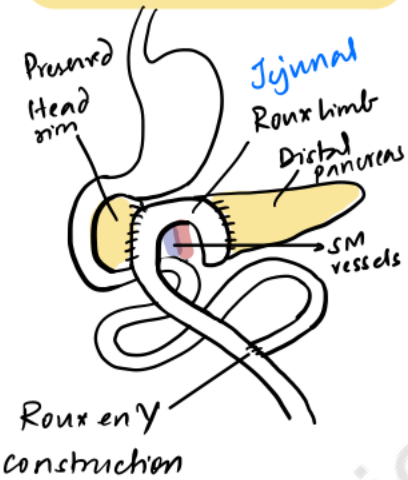
± Splenectomy

B. Proximal Pancreatectomy – Resection + Drainage

- **DPPHR** – Duodenum Preserving Pancreatic Head Resection
- **Whipple's Procedure**
  - Classical + HJ + PJ + GJ
  - Pylorus Preserving (Traverso Longmire PPPD)

DPPHR

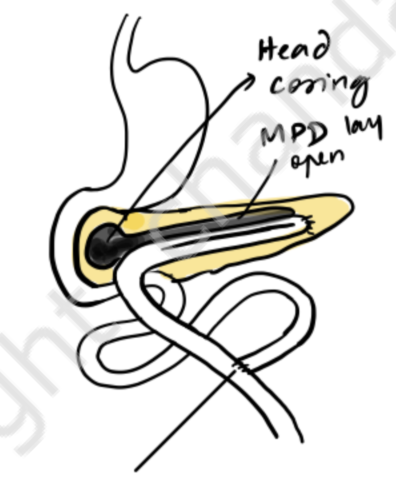
Beger Operation



- Transection of pancreatic neck
- Subtotal resection of pancreatic head
- Preservation of CBD & Duodenum

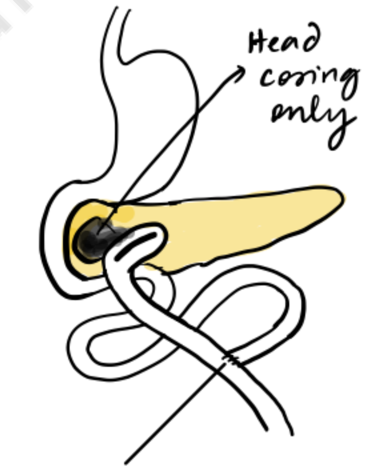
Reconstruction by anastomosis of Head rim & distal pancreas to same Roux limb

Frey Procedure – LR-LPJ (local resection)



- Head coring
- Longitudinal dochotomy of MPD
- Lateral side to side Roux en-Y PJ

Berne Modification of DPPHR



- Just head coring
- No dochotomy

Roux limb of jejunum sewed to residual pancreatic head rim

Hamburg Modification of Frey's / Izbicki Procedure

Done for small duct disease (Duct < 7mm) ē inflammatory head mass

Extensive head coring ē LPJ

V- Plasty

↓ for small duct disease

- V-shaped excision + Roux en Y PJ

C. Total Pancreatectomy ē Islet Cell transplantation

SurgeLight chandanasric@gmail.com

PANCREATIC NEUROENDOCRINE TUMORS

NON FUNCTIONING TUMORS (70-90%)
↓
2/3RDS - MALIGNANT

FUNCTIONING TUMORS - (10-30%)

CELL TYPE	LOCATION		HORMONE	MALIGNANT %	TUMOR SYNDROME	HORMONE LEVELS
	WITHIN ISLET	IN PANCREAS				
α	Peripheral	Evenly distributed	GLUCAGON	>70%	Glucagonoma: Diabetes Necrolytic erythema migrans Hypoaminoacidemia	(N) - <150 pg/mL Tumor FASTING >1000 pg/mL
β	Central	Body, tail	INSULIN (TRH, amylin, CGRP, PRL, Pancreastatin)	10%	INSULINOMA - Whipple's triad	>5 μIU/mL despite hypoglycemia
D	Evenly distributed	Evenly distributed	SOMATOSTATIN	~70%	Somatostatinoma - Diabetes, Gallstones, steatorrhea	(N) 10-25 pg/mL Tumor - >160 pg/mL
D_2	Evenly distributed	Evenly distributed	VIP	50%	VIPoma - Watery Diarrhea, Hypokalemia, Acidosis, Achlorhydria (Verner Morrison S°)	(N) <200 pg/mL Tumor - 225-2000 pg/mL
G	—	PASSARO Δ	GASTRIN		Zollinger Ellison S° Acid Hypersecretion Diarrhea Peptic Ulcer Disease	(N) - <100 pg/mL ↑ >1000 pg/mL ↑ of >200 pg/mL i secretin

WHO GRADING SYSTEM FOR PNETs

WELL DIFFERENTIATED
Homogenous small round cells ∴ abundant expression of neuro-endocrine markers

- LOW GRADE G_1
  - <2 mitoses/10 hpf
  - Ki67 - <3%
  - INDOLENT
- INTERMEDIATE GRADE G_2
  - 2-20 mitoses/10 hpf
  - Ki67 - 3-20%

POORLY DIFFERENTIATED
Pleomorphic cells ∴ nuclear irregularity necrosis

- HIGH GRADE G_3
  - >20 mitoses/10 hpf
  - Ki67 >20%
  - AGGRESSIVE
  - Also called Neuroendocrine carcinoma

Malignancy in PNETs can be definitely determined only in the presence of metastasis

Predictors of aggressive growth - >2cm size, Non functional tumor, liver/lymphnode mets

surgeonmitchandanasri@gmail.com

MOLECULAR GENETICS

1) Most PNETs are sporadic
2) Genetic Syndromes
   - MEN-1 – multiple, a/i other tumors
   - VHL – usually indolent PNETs
   - NF-1

CLINICAL PRESENTATION

- DUE TO SECRETION OF ACTIVE GASTROINTESTINAL HORMONES → Functional Syndromes
- DUE TO PHYSICAL EFFECT OF TUMOR
  - Pain
  - Biliary obstruction / Jaundice
  - Anorexia, wt loss
  - Metastasis

EVALUATION

- FUNCTIONAL SYNDROME → ELEVATED SERUM LEVELS OF SUSPECTED PEPTIDE → LOCALISATION & STAGING
- MASS EFFECT → Imaging features suggestive of PNET → R/o Functional Syndromes → Confirm PNET
  - ↑ Chromogranin A
  - Neurotensin
  - Pancreatic Polypeptide

IMAGING

1) USG → Sonolucent w.r.t. surrounding pancreas
2) CECT → Pancreatic Protocol
   PNETs are hypervascular tumors → Hyperenhancement in arterial phase (Primary, Liver metastasis)
3) MRI → BRIGHT on T_2 weighted imaging, ↓ signal intensity on T_1
   → Sensitivity ∝ size of the tumor
4) EUS – also allows guided FNAC; not very good for small duodenal tumors
5) SOMATOSTATIN RECEPTOR SCINTIGRAPHY (Principle: abundance of somatostatin receptors on most PNETs except INSULINOMA) – for non-insulinoma PNETs – 1° & 2°
6) SPECT/CT
7) Angiography – characteristic vascular blush

OTHER LOCALISING MODALITIES – Portal Venous Sampling:
- Ca^{2+} stimulation (INSULIN)
- Secretin stimulation (GASTRIN)

surgeonlight.chandanasri@gmail.com

MANAGEMENT

① Non-metastatic Disease Localised pre-operatively

↓

Surgical resection – IO + Regional LNs

② Non-metastatic Disease NOT LOCALISED preoperatively

Intra-op US (7.5-10 MHz – Pancreas
5 MHz – liver)

↓

- Islet tumors appear sonolucent
- Identification of relation to ductal system
- Doppler – vascular anatomy

Intra-op localisation is better i mobilisation of pancreas

③ Metastatic Disease

- Resection in select cases → Total resection + liver transplant for tumors i favorable characters
- Liver directed therapies – RFA
  HAI
  TACE – Dmg eluting beads
  Radioembolisation – Yttrium 90 microspheres

SYSTEMIC THERAPY

Well Diff → Respond poorly to cytotoxic Rx
SOMATOSTATIN (after cholecystectomy)
Everolimus
Sunitinib

Poorly Diff → Respond well to cytotoxin chemotherapy (Platins + Etoposide)

NON-INSULINOMA PANCREATOGENOUS HYPOGLYCEMIA / NESIDIOBLASTOSIS

Excessive pancreatic β cell function
a/i pancreatic islet hyperplasia & Dysplasia
-disease of infancy; rarely seen in adults

HALLMARK – POST PRANDIAL HYPOGLYCEMIA (WITHIN 4hr of meal)

Rx – Distal pancreatectomy

Surgeonlight chandanasri@gmail.com

INSULINOMA

- m/c functioning PNET
- 1-2/million

Whipple's Triad

1) Neuroglycopenic symptoms consistent ↓ Hypoglycemia
2) Low Blood Glucose at time of symptoms
3) Relief of symptoms ↑ Glucose administration

Generally small, benign & solitary (85-95%)
5% MEN-1 - Multiple Malignant

DIAGNOSIS

1) GOLD-STANDARD:
72 hr monitored fast
6 hrly measurements of Insulin, C-peptide, proinsulin, β-hydroxybutyrate until Blood glucose drops to 60mg/dL
→ Thereafter 1-2 hrly measure
↓
DEMONSTRATION OF INAPPROPRIATELY ↑ INSULIN LEVELS IN HYPOGLYCEMIA → DIAGNOSTIC

2) INSULIN GLUCOSE RATIO > 0.3 → Insulinoma

3) C peptide >1.2μg/mL (when glucose is <40mg/dl)

4) βHBA<2.7mmol/L (insulin is antiketotic)

Imaging

SRS is useless
CT/MRI → EUS → Angiogram ± Calcium → intra op US

Rx

- Peri-op glucose
- DIAZOXIDE (inhibits insulin release)
- Resection

METS
- Somatostatin
- Hepatic artery Embolisation
- DIAZOXIDE
- STREPTOZOCIN + 5FU

GASTRINOMA

- 2nd m/c functioning PNET

Zollinger Ellison S°

1) Acid Hypersecretion
2) Non β islet cell tumor
3) Peptic Ulcer disease

Hypergastrinemia

Duodenal > Gastric Ulcer
JEJUNAL ULCERS **

Acid induced DIARRHEA (↓ ± RTA)

90% in PASSARO △
>60% in duodenum

Gastrin levels

>1000pg/mL + pH < 2

(↑ pH ± 100-1000pg/mL gastrin → PPI/H_2B, Atrophic gastritis)

Secretin stimulation

Gastrin >200pg/mL after secretin

Ddx Hypergastrinemia

- G cell hyperplasia
- GOO
- Retained antrum
- H_2B/PPI
- Vagotomy
- Atrophic gastritis

IMAGING

CT/MRI (water as contrast for duodenal lesions) → EUS
↓
SRS (almost all gastrinomas express Somatostatin receptors)

Rx

PPI therapy
Resection
Gastrectomy

GLUCAGONOMA

5-17% MEN1

- RARE
- ~1/20 million
- F>M
- α cells

4D's

- Diabetes
- Dermatitis (NEM)
- DVT
- Depression

NEM - ~2/3rd cases, precedes other findings -d/t amino acid deficiency

50-80% Malignant
↓
80% have liver mets at diagnosis

Imaging

LARGE - 5-10cm
Body & tail of pancreas
Easy to localise

Glucagon levels

Fasting level - >1000pg/mL (N <100pg/mL)

Management

- Correction of amino-acid & vitamin deficiencies
- Octreotide to reverse the catabolic state + NUTRITION
- DVT prophylaxis

Rx - Resection

VIPOMA

5% MEN-1

- RARE
- ~1/million
- D_2 cells

VIP is secreted by Brain, antral G cells, adrenal medulla, gut mucosa & pancreatic neurons

Watery Diarrhea
Hypokalemia
Achlorhydria
Hypercalcemia
PANCREATIC CHOLERA
'Verner Morrison S°'

Diarrhea

(Explosive, >5L/d
↓
secretory diarrhea, persists ± fasting despite RTA (c/f 2ES)

Hypokalemia - d/t diarrheal K+ loss

Achlorhydria - VIP inhibits acid secretion

Hyperglycemia (25-50%) d/t Glycogenolysis (VIP action)

>2/3rds - MALIGNANT
70% - metastatic at Dx

Imaging

- Solitary
- Large (>3cm)
- Body & tail

VIP - 225-2000pg/mL (N <200pg/mL)

Rx - Somatostatin
Stabilize
Resection

SOMATOSTATINOMA

- RARE
- < 100 cases reported

'D' cells

Unregulated secretion of Somatostatin causes inhibition of secretion of several enzymes and hormones

DIABETES
STEATORRHEA
MALABSORPTION
} Pancreatic hormone & enzyme inhibition

CHOLELITHIASIS → d/t ↓ GB emptying (Because of ⊖ CCK secretion)

Fasting Plasma Somatostatin >160pg/mL

Larger tumors
85% >2cm
- 60% found in head of pancreas
- 90% are malignant
↓
liver and lymphnode metastasis at diagnosis

Associated ± NF-1
Pheochromocytomas
(Rarely ± MEN-1)

Imaging

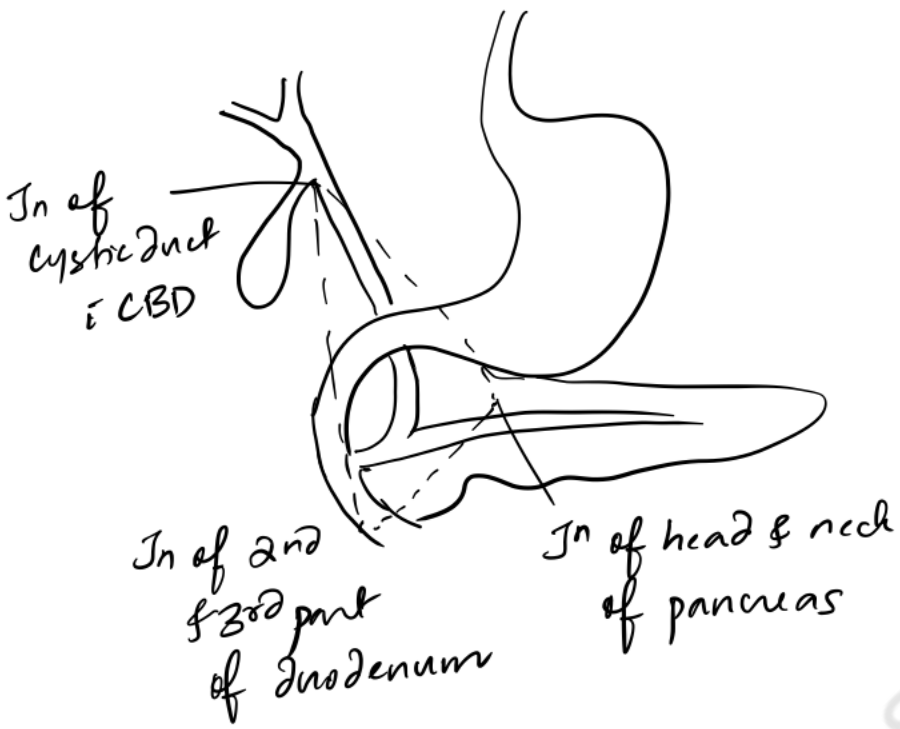
Easy ± cross sectional imaging (due to large size)

Management

- If non-metastatic → SURGICAL RESECTION
- Metastatic - Surgical debulking helps in relief of symptoms

surgeonchandrasagar@gmail.com

PAZZARO △



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CYSTIC NEOPLASMS OF PANCREAS

SEROUS CYSTIC NEOPLASMS

- Serous cystadenoma
  - Microcystic (Polycystic)
  - Oligocystic
- Serous cystadenocarcinoma

MUCINOUS CYSTIC NEOPLASMS

Mucinous cystic neoplasm :
- low/intermediate grade dysplasia
- high grade dysplasia
- associated invasive carcinoma

INTRADUCTAL PAPILLARY MUCINOUS NEOPLASM

IPMN : ↓ or int dysplasia

IPMN : ↑ gr. dysplasia (non-invasive)

IPMN : invasive Ca

→ cause cyst-like dilatations of MPD/SBD

SEROUS CYSTIC NEOPLASM

F > M - 7:3

60-80y

- Polycystic (microcystic) : central stellate scar
- May be oligocystic

Single layer cuboidal cells rich in GLYCOGEN

Imaging - HONEYCOMB PATTERN around central stellate scar

Fluid analysis

↓ Viscosity
↓ CEA
↓ Amylase

Cells : Glycogen rich cytoplasm

Almost always benign

Rx
- Stable → Surveillance
- Symptomatic/Enlarging → Resection

MUCINOUS CYSTIC NEOPLASM

F > M → 10:1

Perimenopausal - 40-50y

Large, macrocystic thick-walled

Subendothelial ovary like stroma - tall columnar mucinous epithelium

Imaging - Large cyst near body/tail
- no duct communication
- Calcifications

Fluid - ↑ Viscosity
↑ CEA
↓ Amylase

Potentially malignant

<3cm - Surveillance

>3cm - Resect

IPMN

F = M 1:1

60-70y

Multilocular - involve MPD/SBD

Tall columnar mucinous epithelium : varying degrees of dysplasia

Imaging
- MD-IPMN
- SD-IPMN
- Mixed duct IPMN (Ductal communication)

↑ Viscosity
↑ CEA
↑ Amylase

Potentially malignant

Rx - Resect

SurgeEight chandanasri@gmail.com

PANCREATIC PSEUDOCYST

Definition: A pseudocyst is a well circumscribed fluid collection (with no associated tissue necrosis, WON) that is present for 4/more weeks after disease onset

Location: m/c – peripancreatic – LESSER SAC
- can also be found in pelvis/thorax/mediastinum

PATHOGENESIS

PANCREATIC DUCTAL DISRUPTION

d/t
- inflammation in acute pancreatitis (10-15%)
- trauma → pseudocysts develop ant. to neck & body
- duct obstruction in chronic pancreatitis (20-40%)

↓

Leakage of enzyme rich secretion

↓

Marked inflammatory reaction in the peritoneum retroperitoneal tissue

↓

Fluid is contained by a developing layer of granulation tissue and fibrosis → Prevents fluid resorption

↓

Matures → PSEUDOCYST
- communication of the duct may/may not persist
- Persistent communication → ↑↑ enlargement

D'EGIDO CLASSIFICATION OF PSEUDOCYSTS

	Context	Pancreatic Duct	Ductal Communication	
Type-1	Acute Pancreatitis	(N)	No	Rx: Drainage
Type-2	Acute on chronic Pancreatitis	Diseased but no stricture	± 50:50	Rx: Drainage + Resection
Type-3	Chronic Pancreatitis	Duct stricture ↓ obstruction	(+)	Rx: Drainage + Decompression of duct

SurgeLight chandanasri@gmail.com

CLINICAL FEATURES

- Abdominal pain
- Fullness - Mass PA - "Baid test" - RT felt PA ∵ cyst pushes stomach anteriorly
- Early satiety / Anorexia

COMPLICATIONS

- Infection
- Biliary obstruction → Jaundice
- GI obstruction
- Intraysic hemorrhage
- Peritonitis d/t rupture

EVALUATION

1) S. Amylase △↑ / N
2) Imaging - USG
   CECT — Cyst size
   location
   relations
   wall thickness
   Pancreatic duct - size / strictures / calcifications
   stones
   Pancreatic parenchyma - calcifications / atrophy
   Vascular anatomy → pseudoaneurysm
   MRCP - Ductal communication
   Any solid components — WON → necrosectomy needed
   ERCP - not routinely required
   symptomatic, intervention being planned
3) UGI scopy - to plan management
4) Fluid aspiration & analysis
   If infection suspected
   Gram stain
   C&S
   If cystic neoplasm suspected
   Enzymes
   CEA, CA-19-9
   Mucin

SurgeLight chandanasri@gmail.com

MANAGEMENT

- Asymptomatic ± Not enlarging → Observe
- Infected → Percutaneous drainage
- SYMPTOMATIC ± Enlarging
  - ↳ If pseudocyst is >6 cm & has been + for >6 weeks & wall thickness >6(4) mm → unlikely to resolve spontaneously
  - Persistence - more likely i/c/o distal stricture of MPD proximal communication b/w PD

∴ Symptoms ± Complications + Non resolution → INTERVENE

2 important precautions before intervening in pseudocyst

1) Always r/o cystic neoplasm
2) Do not electively (for non-infected) drain a pseudocyst EXTERNALLY if there is r/o ductal communication b/w distal obstruction

→ will lead to PANCREATICOCUTANEOUS FISTULA!

MINIMALLY INVASIVE PROCEDURES

RADIOGRAPHIC

Percutaneous drainage
- D'Egidio (I) → External drainage
- (N) duct

Percutaneous transgastric drainage
- ↳ Percutaneous cystogastrostomy

ENDOSCOPIC

Transpapillary PD stent → when ductal stricture (+)

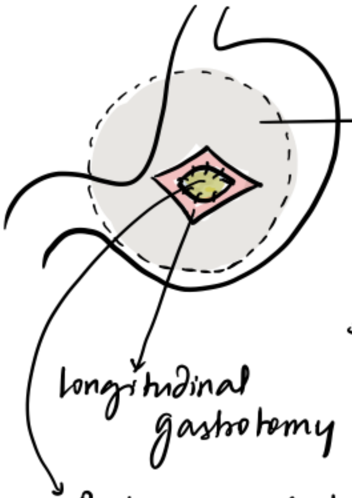
Transgastric stent
Transduodenal stent
> Endoscopic transmural internal drainage } when ductal communication (+)

Surgical Light dr.adarsh@gmail.com

SURGERY < Open / Lap } Preferred when there is
- ductal stricture
- ductal communication
- Disease that has to be resected

Procedures: CG / CD / CJ } ± LPJ / DP ± Splenectomy

CYSTOGASTROSTOMY



Posterior gastrotomy
↓
2cm disc of posterior gastric wall & subjacent pseudocyst wall excised → sent for HPE
↓
r/o cystic neoplasm

Pseudocyst wall sewn to posterior gastric defect to form an internal stoma → debride cyst wall & aspirate

Disadvantage:

CG is NOT a dependent stoma
- may act as sump
- Gastric debris may collect

CYSTODUODENOSTOMY

↓

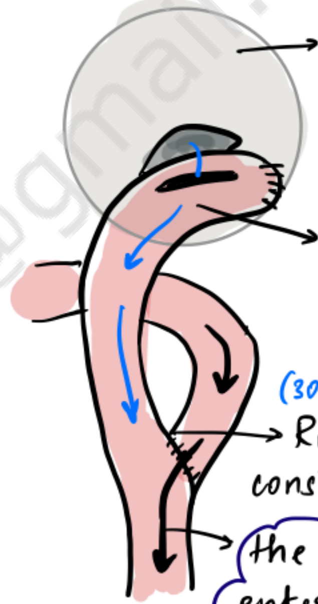
Rarely necessary except when
- pseudocyst is located at pancreatic head
- directly abuts duodenal wall

Longitudinal duodenotomy to expose medial wall of duodenum
↓
Medial duodenal wall + 2cm of cyst wall excised (take care not to injure Gastroduodenal artery or MPD)
↓
Disadvantage

↑ Morbidity & Mortality a/e
- Anastomotic dehiscence
- Abscess formation

↓
∴ AVOID CD

CYSTOJEJUNOSTOMY (ROUX EN-Y)



↓
Roux limb is versatile and can be brought to any pseudocyst location within abdomen

Anastomosis is usually fashioned through a window in the transverse mesocolon

Surgeright chandanasri@gmail.com

Pancreatic Fluid Collections

Definitions of pancreatic fluid collections

TERM	DEFINITION
Peripancreatic fluid collection ↓ No wall	A collection of enzyme-rich pancreatic juice that occurs early in the course of acute pancreatitis, or that forms after a pancreatic duct leak; located in or near the pancreas; it lacks a well-organized wall of granulation or fibrous tissue
Early pancreatic (sterile) necrosis	A focal or diffuse area of nonviable pancreatic parenchyma, typically occupying >30% of the gland and containing liquefied debris and fluid
Late pancreatic (sterile) necrosis (Wall ✓)	An organized collection of sterile necrotic debris and fluid with a well-defined margin or wall within the normal domain of the pancreas
Acute pseudocyst	A collection of pancreatic juice enclosed within a perimeter of early granulation tissue, usually as a consequence of acute pancreatitis that has occurred within the preceding 3–4 wk
Chronic pseudocyst	A collection of pancreatic fluid surrounded by a wall of normal granulation and fibrous tissue, usually persisting for >6 wk
Pancreatic abscess	Any of the above in which gross purulence (pus) is present, with bacterial or fungal organisms documented to be present

Surgelight channel (@gmail.com

Local complications of acute pancreatitis

	ACUTE (<4 WEEKS, NO DEFINED WALL)		CHRONIC (>4 WEEKS, DEFINED WALL)	
CONTENT	NO INFECTION	INFECTION	NO INFECTION	INFECTION
Fluid	Acute pancreatic fluid collection (APFC)	Infected APFC	Pseudocyst	Infected pseudocyst
Solid ± fluid	Acute necrotic collection (ANC)	Infected ANC	Walled off necrosis (WON)	Infected WON

Reproduced with permission from Windsor JA, Petrov MS: Acute pancreatitis reclassified, *Gut*. 2013 Jan;62(1):4-5.

D'EGIDIO CLASSIFICATION OF PSEUDOCYST

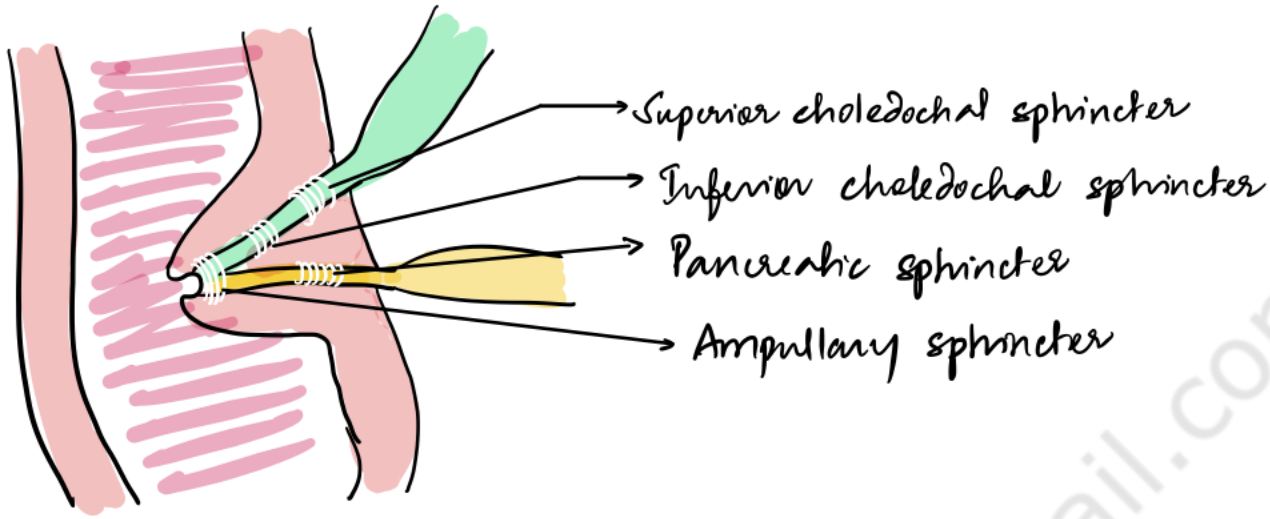
I : Post acute Pancreatitis
(N) Duct anatomy
No fistula / communication

II : Post acute on chronic Pancreatitis
Abnormal anatomy ó ant stricture
Communication (±) 50 % chance

III : Post chronic pancreatitis
Abnormal anatomy ó stricture
Communication (+)

SurgeLight chandanasri@gmail.com

SPHINCTER OF ODDI



SOD - 6-10 mm long
- lies within duodenal wall

① ABNORMAL PANCREATICOBILIARY DUCT JUNCTION
- Long common channel → predisposes to reflux of pancreatic juice into the biliary tree

Predisposes to
- Choledochal cysts
- Cholangiocarcinoma

② SOD dysfunction
- Biliary type
- Pancreatic type

BILIARY

Typical biliary type pain
+
1) AST/ALT/ALP > 2x(UNL) on ≥2 episodes of pain
2) CBD ≥12mm
3) Prolonged biliary drainage time

Type ① - all of the above
Type ② - Pain + ≥1 criteria
Type-3 pain only

PANCREATIC

Typical pancreatic pain
+
1) Amylase/lipase > 2x(UNL) on ≥2 episodes of pain
2) MPD < Head → 6mm, Body → 5mm
3) Prolonged biliary drainage time

Type ① - all of the above
Type ② - Pain + ≥1 criteria
Type-3 pain only

surgeonlight chandanasri@gmail.com

Access Loops In Biliary-Enteric Anastomoses

Roux-en-Y hepaticojejunostomy is the standard procedure used by most hepatobiliary surgeons for biliary reconstruction following iatrogenic bile duct injury, benign and malignant CBD strictures, choledochal cysts and biliary tract tumors management.

The incidence of anastomotic stricture following hepaticojejunostomy in experienced centers ranges between 5%-22%.

Hepaticojejunostomy stricture is a serious complication of biliary surgery, if untreated, can lead to repeated cholangitis, intrahepatic stones formation, biliary cirrhosis, hepatic failure and eventually death.

Revision of hepaticojejunostomy is a complex procedure, the surgical procedure being made difficult by the sequelae of long-standing unrelieved biliary obstruction like portal hypertension due to secondary biliary cirrhosis, atrophy of liver lobes and presence of cholangiolytic liver abscess.

Endoscopic management is not only the least invasive but also very effective via either balloon dilatation or stenting of the stricture.

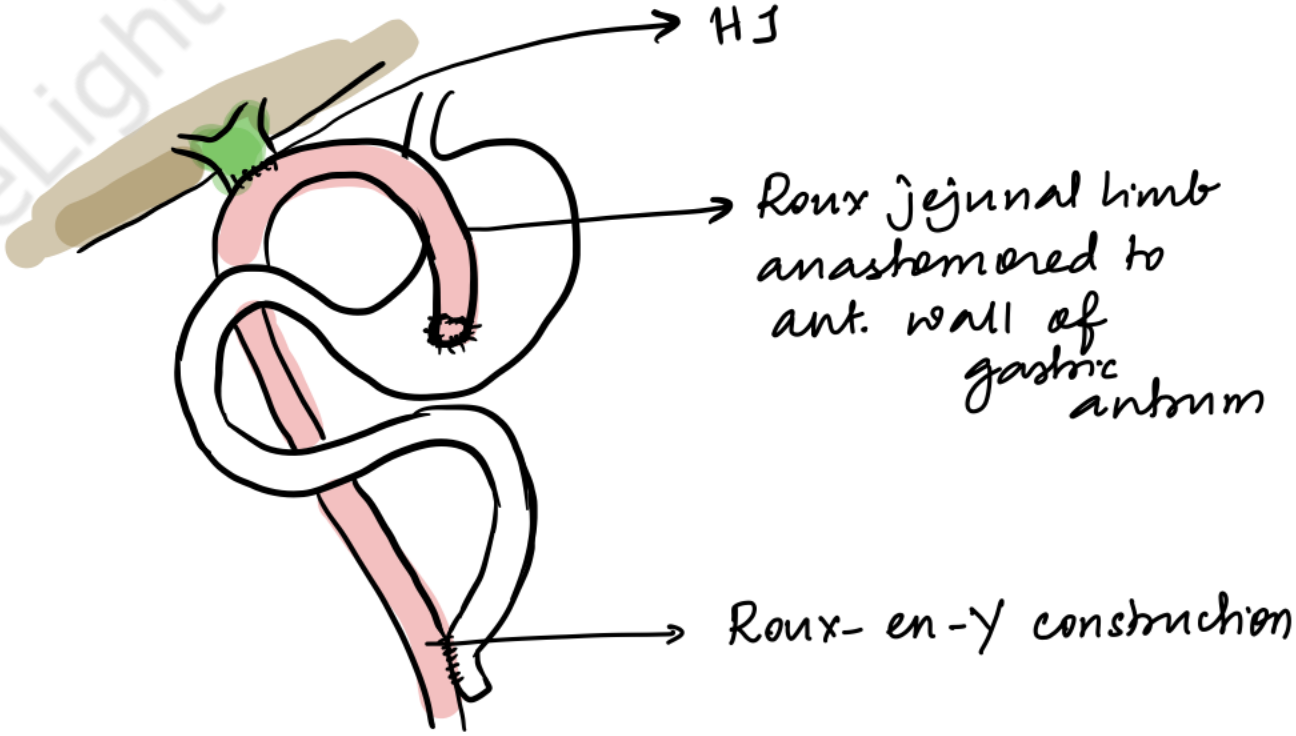
surgeonlight.chandanasri@gmail.com

In patients with "Roux-en-Y" hepaticojejunostomy, the endoscopic access to the anastomosis is hampered by the distance traveled by the jejunal loop until reaching the angle of the enteral anastomosis.
Many modifications of hepaticojejunostomy to provide permanent endoscopic access have been described in the literature including duodenal, gastric and subcutaneous access

Gastric Access Loop

In the gastric access loop, the same steps are done for performing roux-en-Y hepaticojejunostomy.

The end of the Roux jejunal loop taken up for hepaticojejunostomy is not closed but is anastomosed to the anterior wall of the gastric antrum near the pyloric orifice.



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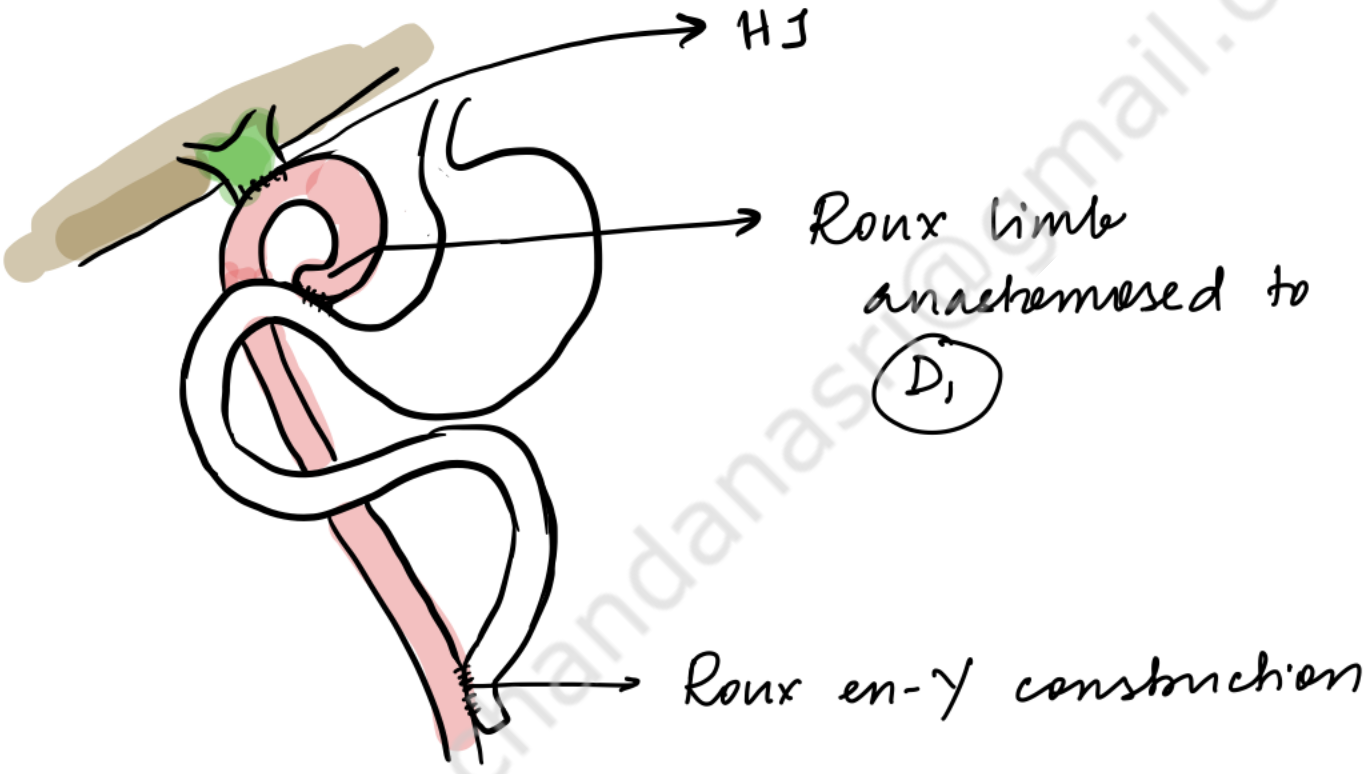
DUODENAL ACCESS LOOP

After completion of hepaticojejunostomy (HJ), the free end of the Roux loop is anastomosed to the first part of duodenum in a side to side fashion.



- Makes the anastomosis endoscopically accessible

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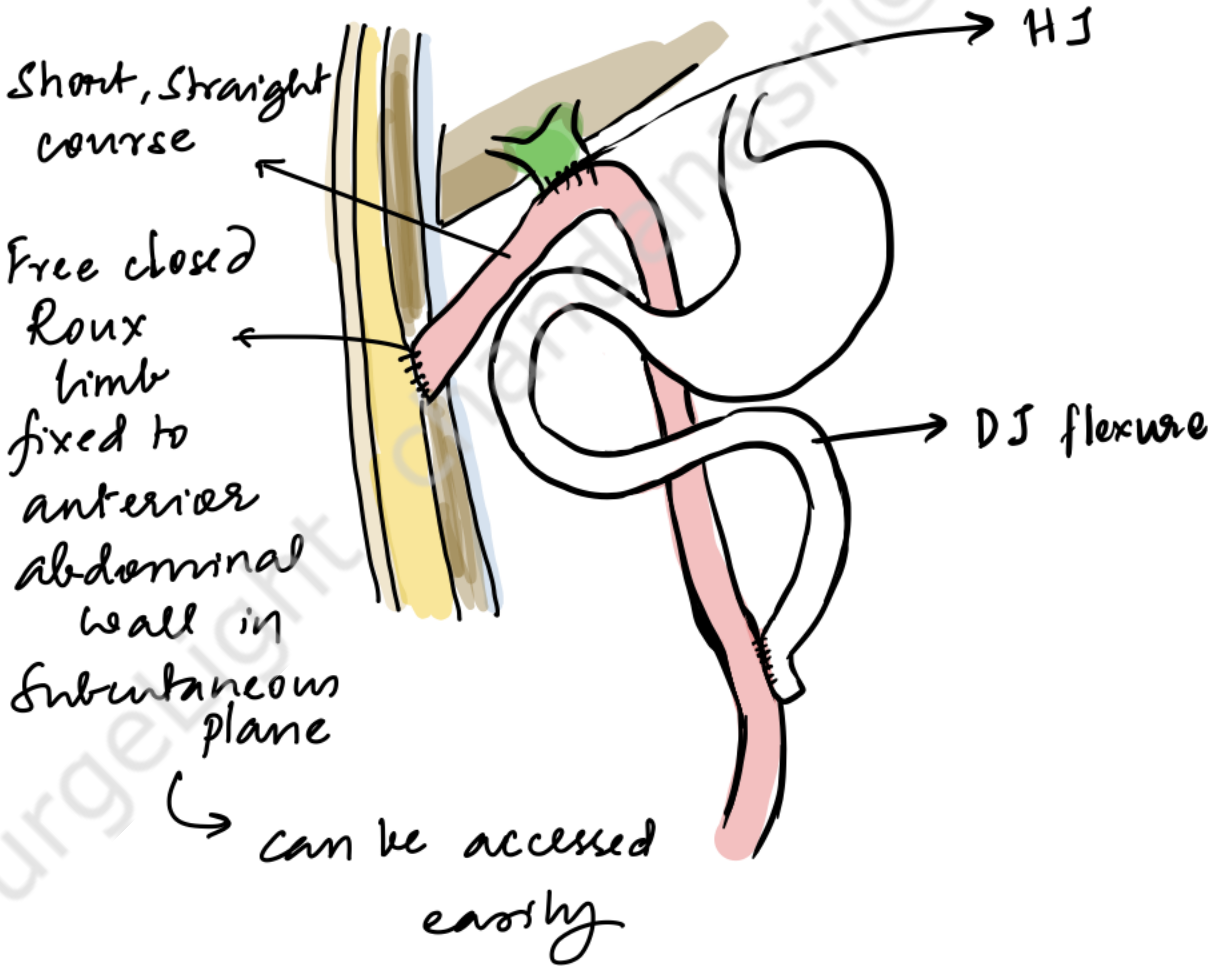
Subcutaneous Access Loop

The closed free end of roux loop is passed through the anterior abdominal wall in the right subcostal area and then fixed to the wall in a subcutaneous position using 3/0 polyglactin sutures. The limb between the hepaticojejunal anastomosis and the subcutaneous fixation should be short and straight.



Surgeonlight.chandrasri@gmail.com

ADHESIONS

m/c/c of intestinal obstruction

Causes
- Iatrogenic – m/c — Post-surgical
- Inflammatory conditions
  - Abdominal TB
  - PID, Asherman so
  - Fitz Hugh Curtis Syndrome
  - Appendicitis
  - Cholangitis
  - Cholecystitis
  - Enteritis
  - IBD
  - Radiation
- Congenital
  - Eg: LADD'S BAND
    MECKEL'S DIVERTICULUM
    PERSISTENT URACHUS

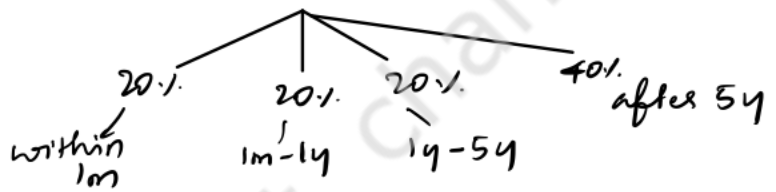
TYPES

FIBRINOUS
- 5-7d post op
- thin/slimey fibrin exudate between surfaces

FIBROUS
late
- band/bridge between adjacent surfaces
  ↓
  mesothelization
  ↓
  fibrin matrix replaced by granulation tissue covered by mesothelium

Presentation

- CAN CAUSE INTESTINAL OBSTRUCTION



- Chronic Pelvic Pain
- Infertility

RISK FACTORS
- Inframesocolic surgeries
- Pelvic surgeries
- Open procedures & tissue handling
- Use of gloves & starch granules
- gallstone spillage
- excessive peritoneal sanguinous fluid during closure

Surgical Light Chandanasri@gmail.com

STEPS TO DECREASE ADHESIONS

- Use lap / minimally invasive approaches when possible
- Minimal, gentle, meticulous tissue handling
- Strict hemostasis
- Sharp dissection
- thorough irrigation
- PRODUCTS
  - Barrier products:
    Polymers - Hyaluronic acid
    Gelatin
    collagen
    cellulose, dextran
    Carboxymethyl cellulose
    PEG
  - Antiadhesive agents - Anti fibrin action
    streptokinase, TPA, PAI-1
  - to limit cell proliferation - 5FU, mitomycin C
  - Antiinflammatory - Vit C

MANAGEMENT

AIO - Laparotomy
Careful band release
Adhesiolysis
Resection of unhealthy segments

Recurrent episodes

- Noble's plication
- Charles Phillip Transmesentric plication
- Intestinal intubation

Surgeonlight chandanashri@gmail.com

ABDOMINAL TUBERCULOSIS

Abdominal tuberculosis : 5% of all cases of TB
11% of all cases of Extrapulmonary TB
↳ 6th m/c form of EPTB

FORMS OF ABDOMINAL TB

PERITONEAL

ACUTE
↓
TB Peritonitis
↓
Rare

CHRONIC
- WET TYPE - ASCITES
- FIXED FIBROTIC omentum, mesentery, bowel matted ENCYSTED ASCITES
- DRY/ PLASTIC - Fibrous peritoneal reaction, nodules, adhesions
- PURULENT - TB SALPINGITIS

GI TRACT
Esophagus
Stomach
Intestine
Anorectum

SOLID VISCERA
Liver
Pancreas
Spleen

LYMPHNODE
Mesenteric
Peri-portal
Para-aortic

- Pseudomesenteric cyst / cold abscess
- Tabes mesenterica - large LN mass

Modes of infection

1) Ingestion ⟶ Swallowed infected sputum
undercooked meat / unpasteurised milk
2) Hematogenous spread
3) Contiguous spread (from genitourinary TB)
4) Spread via Lymphatic channels
5) Reactivation of latent focus

RISK FACTORS FOR TB

- Poor immunological status
  - HIV
  - Cirrhosis
  - T_2 DM
  - Malignancy
- Treatment with anti TNF agents - Infliximab
- Use of peritoneal dialysis

SurgeLynchanasri@gmail.com

PERITONEAL TUBERCULOSIS

- <1% of all cases of TB
- 4% of EPTB
- 25-60% of Abdominal TB

Modes of infection

1) Reactivation of latent foci in peritoneum previously acquired by hematogenous spread from lung primary → m/c
2) Hematogenous spread from active TB
3) Contiguous spread — Rupture of TB abd. node
   — Fallopian tube
4) Via lymph channels from abd. TB nodes

Forms of Peritoneal TB

Acute
TB peritonitis
presents : pain, tenderness, vomiting, distension
Lap → Ascites, tubercles

Chronic
- Ascitic → a/c hydrocele
- Encysted or loculated
- Plastic → adhesions
- Purulent → can lead to parietal abscess, Umbilical fistula

CLINICAL FEATURES

- Insidious onset
- Constitutional symptoms
- Abd mass :- doughy abdomen, rolled up omentum
  tenderness
- Ascites
- Hepatomegaly

Intra-op: multiple, yellow-white tubercules <5mm studding visceral and parietal peritoneum, serosal surface, mesentery
Ascites
Mesenteric nodes
Adhesions - Matted bowel loops
Omental thickening

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INTESTINAL TUBERCULOSIS

ULCERATIVE — M/C – 60%.

- usually 2° to pulmonary TB
- more virulent organism +/or poor host defence

Swallowing infected sputum (PT ± pulmonary TB)
↓
TB bacilli colonise the lymphatics in terminal ileum
↓
Granulomas → caseation → necrosis
↓
ULCERATION (c.f. typhoid - longitudinal) + endarteritis + edema
↓
TRANSVERSE ULCERS & TYPICAL UNDERMINED EDGES

often circumferential (along lymphatics) (GIRDLE ulcers)
multiple - skip lesions

- Generally presents ± anemia, weight loss, debility

Mixed — ~30%.

Ulceroconstrictive
Ulcerohypertrophic

HYPERPLASTIC — ~10%.

- generally 2/t 1° intestinal TB - *M. bovis*
- less virulent organism & good host defence

Organism causes a pronounced HOST RESPONSE in terminal ileum 2/t
abundance of lymphoid follicles
↓
Hyperplasia
↓
Luminal narrowing
Bowel wall thickening
adhesions
↓
fibrosis → bowel shortening
↓
Pulled up caecum (subhepatic position)

- Generally presents ± SAIO Mass in RIF

REASONS FOR PREDILECTION FOR ILEUM IN INTESTINAL TB

- ↑ Physiological stasis → greater contact time
- Abundance of lymphoid follicles
- ↓ digestive activity
- ↑ Liquid content

LIVER INVOLVEMENT

Intestinal → Portal → local Granuloma
Pulmonary / EPTB —hematogenous→ Hepatic A
Miliary TB

Sunlight chandanasri@gmail.com

EVALUATION OF ABDOMINAL TUBERCULOSIS

EVIDENCE OF TUBERCULOSIS

- Pulmonary tuberculosis
  - local / constitutional symptoms
  - Suggestive CXR findings
  - Sputum AFB
    - Culture - BACTEC (liq)
    - CBNAAT
- Abdominal TB
  - ASCITIC TAP
    - Exudative (Protein > 3g/dL, SAAG < 1.1 g/dL)
    - lymphocytosis
    - AFB ± / Culture ±
    - CBNAAT
    - ADA > 30 U/L (Serum - 40 U/L)
    - ↑ Specific gravity
    - Glucose < 30 mg/dL
    - ↓ pH
    - LDH > 90 U/L
  - FNA / Biopsy - Granuloma, Caseation
- Mantoux test
- ESR - Contributory if ↑

EXTENT / PATTERN OF DISEASE

- Plain X Ray - abd - evaluating SAIO
  - Calcified LN / granuloma
- BARIUM STUDIES
  - Barium meal follow through
  - Enteroclysis
  - Barium enema
- USG - Ascites, loculated / encysted fluid
  - Club sandwich / sliced bread sign - fluid between matted loops
  - Peritoneal thickening
  - Pseudokidney sign
  - LNs
  - Mesentery thickening
- CT - LNs, Bowel thickening
- Colonoscopy
- Laparoscopy

BARIUM STUDIES IN TUBERCULOSIS

- Pulled up caecum
- Contracted / Conical caecum
- Obtuse IC angle
- CHICKEN INTESTINE - Hypersegmentation, Flocculation of Barium
- STERLEIN SIGN - rapid transit of barium in inflamed segment
- FLEISCHNER SIGN - Narrow ileum & thickened ICT
  - "Inverted umbrella sign"
- STRING SIGN

Rx - ATT

Surgery indications: Perforation, Massive bleeding, Complete obstruction, Abscess / fistula

Stricturoplasty vs RCA

→ RHC → ↑ output, failed Rx

GASTROINTESTINAL FISTULAS

Fistula - Abnormal communication b/w 2 epithelial surfaces

TYPES

BASED ON ETIOLOGY

NON-OPERATIVE (25%)

- Malignancy
- Inflammation
  - IBD
  - Diverticulitis
  - Perforated bowel / ulcer
  - Ischemic bowel
  - TB
  - Infections
- Radiation
- Trauma

POST-OPERATIVE (75%)

EARLY

- Breakdown of GI anastomosis / enterotomy closure
- Missed enterotomy

INTERMEDIATE

- SSI
- Percutaneous abscess drainage
- Overaggressive Mx of open abd wound

LATE

- FB
- Erosion by mesh

BASED ON LOCATION

INTERNAL

- Bilio-enteric
- Bilio-colic
- Gastro-colic
- Enterovesical
- Enterovaginal
- Colovesical
- Colovaginal

EXTERNAL

- Enterocutaneous
- Colocutaneous
- Enteroatmospheric

- Pharyngeal
- Esophageal
- Biliary
- Pancreatic
- Appendicular
- Gastric
- Duodenal
- Enteric
- Colic

BASED ON OUTPUT (PHYSIOLOGICAL)

- Low-output: < 200ml / 24h
- Intermediate Output: 200-500ml / 24h
- High Output: >500ml / 24h

By composition of output

- Gastric
- Enteric
- Colonic
- Biliary
- Pancreatic

Factors predicting FAILURE of spontaneous closure of fistulas

1) Distal obstruction
2) Local infection
3) High output
4) Very proximal bowel - Jejunal
5) Fistula characters
   - Multiple openings
   - Short length - < 2.5cm
   - Wide diameter - > 1cm
6) Epithelized tract
7) Presence of Foreign Body
8) Open abdomen
9) Tissue characters
   - Inflammed / Infected - IBD
   - Friable
   - Ischemic
   - Irradiated
   - Malignancy
10) Patient characters
    - Profound malnutrition
    - Profound immunosuppression
    - Alcohol
    - Tobacco use

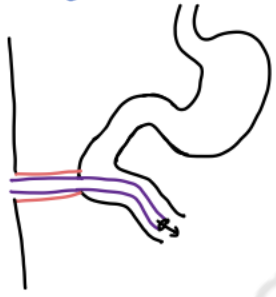
SurgeLight chandanasr@gmail.com

MANAGEMENT OF ENTEROCUTANEOUS FISTULAS

TABLE 16-8: APPROACH TO MANAGEMENT OF ENTEROCUTANEOUS FISTULAS

Phase	Goals	Time Course
Recognition/ stabilization	Resuscitation with crystalloid, colloid, or blood (Fluids) Control of sepsis with percutaneous or open drainage and antibiotics (Abx → exteriorize affected segments if possible) Electrolyte repletion (Electrolytes) Provision of nutrition → PN Control of fistula drainage → Antimotility, Infliximab in IBD, Fibrin glue, Gelfoam plug Commencement of local skin care and protection – NPWT	24-48 h
Investigation	Fistulogram to define anatomy and characteristics of fistula Other GI studies → to rule distal obstruction CT scan to define pathology Operative notes from prior surgery	7-10 d
Decision	Evaluate the likelihood of spontaneous closure Decide duration of trial of nonoperative management	10 d-6 wk When closure, unlikely or after 4-6 wk
Definitive management	Plan operative approach Refunctionalization of entire bowel, Adhesiolysis Resection of fistula with end-to-end anastomosis Secure abdominal closure Gastrostomy and jejunostomy	Surgical intervention at 3-6 mo after patient stabilized (3-6 months)
Postsurgical	Usual postoperative protocol Psychological and emotional support	Ensure access to ICU for management of potential complication Team approach to management facilitates recovery

- High output proximal fistulas – Fistuloclysis



enteral feeding tube is placed directly into the matured high output fistula

Enteral feeding requires atleast ≥4 ft functional intestine

Management of Abdominal Wall following ECF surgery

- No Preop fascial defect – 1° Closure
- Preop fascial defect (+)
  - Small (<5cm) → Primary fascial closure ± some relaxation
  - Large (>5cm) → Primary fascial closure ± Component separation ± Bioprosthesis
    Vascularised Flap closure
    Mesh – Absorbable (Biological / Non biological)

Surgeonsright chandanasri@gmail.com

PREDICTORS OF ECF RECURRENCE - post Op

Patient Factors

- Open abdomen
- Origin of Fistula - Small bowel > Large Bowel
- IBD
- "Frozen abdomen"
- Intra-abd infection

Surgical Factors

Timing - < 4 weeks
or
> 9 months

Multiple inadvertant enterotomies during re-operation

1° closure > Resection + Anastomosis

Stapled > Handsewn anastomosis

Mesh closure

SurgeLight chandanasri@gmail.com

MECKEL'S DIVERTICULUM

only true diverticulum of small intestine

Definition: a true diverticulum consisting of all 3 layers of the small intestine resulting due to the persistence of the vitellointestinal duct

PATHOPHYSIOLOGY: During the 8th week of gestation, the omphalomesenteric duct (vitellointestinal duct) normally undergoes obliteration

Incomplete obliteration → Meckel's diverticulum

→ Meckel's diverticulum is located on the antimesenteric border of the ileum
→ 45-60cm proximal to the ileocaecal valve
→ M:F :: 2:1
→ small bump/ long projection ± lout communication (fistula / persistent fibrous cord) ± umbilicus

→ Cells lining Meckel's diverticulum = pluripotent → heterotopic tissue (+)

- Gastric mucosa
- Pancreatic mucosa
- colonic mucosa

- endometriosis
- Brunner's glands
- Hepatobiliary tissues

RULE OF 2's
- 2% prevalence
- 2:1 male predominance
- 2 ft proximal to ICJ in adults
- ≤ 2y age - 50% are symptomatic within this age

CLINICAL PRESENTATION

- Asymptomatic - incidental discovery on imaging / laparotomy in most cases
- Lifetime risk of complications - 4-6%

1. BLEEDING → m/c presentation ~25-50% of pts ± complications
   m/c symptomatic presentation in <2y age
   → Manifests as: 1) Acute massive hemorrhage
   2) Anemia due to chronic bleeding
   3) Self limiting recurrent episodes.

- usual source of bleeding in Meckel's diverticulum - chronic acid-induced ulcer in the ileum adjacent to a meckel's diverticulum containing heterotopic gastric mucosa

2. INTESTINAL OBSTRUCTION - mechanisms

1) Volvulus of intestine around the fibrous band attaching the diverticulum to the umbilicus

2) Entrapment of intestine by mesodiverticular band (remnant of left vitelline artery → mesodiverticular band - tethers MD to ileal mesentery)

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3) Intussusception : diverticulum acting as a lead point (BROAD BASED DIVERTICULUM invaginates & is carried forward by peristalsis)
4) Stricture 2° to chronic diverticulitis
5) When MD forms a content of inguinal hernia (Littre's hernia) → incarceration of hernia → intestinal obstruction

3. Meckel's Diverticulitis - 10-20% of symptomatic presentation
   Perforation → Peritonitis
   mimics acute appendicitis
4. NEOPLASM → NET m/c neoplasm (77%)
   adenocarcinoma (11%)
   GIST (10%)
   Lymphoma (1%)

EVALUATION

- MD → usually incidental finding on imaging / endoscopy / surgery
- CT - low sensitivity
  - CT : Enterodynamics - 75% accuracy → can be done in acute setting
- RADIONUCLIDE SCANS : Na-Tc 99 M - positive only when diverticulum contains ectopic gastric mucosa (which is capable of tracer uptake)
  → GOOD ACCURACY IN PEDIATRIC AGE GROUP
  Sensitivity 85%
  Specificity 95%
  Adults - sensitivity falls to ~60% d/t ↓ presence of gastric mucosa
  SENSITIVITY can be enhanced by using Cimetidine
  ↓ peptic secretion in gastric mucosa
  ↓
  Retards the release & elimination of tracer from the gastric mucosa
- SPECT-CT
- Barium studies
- Double balloon endoscopy
- Mesenteric angiography - esp. in case of active bleeding

MANAGEMENT

SURGERY

- RESECTION OF DIVERTICULUM (V-shaped diverticulectomy)
- SEGMENTAL RESECTION OF ILEUM

- Asymptomatic → no consensus
  - generally resected - especially in <40y / longer than 2cm, fibrous band, evidence of heterotopic mucosa

SurgeLight chandanasri@gmail.com

MESENTERIC ISCHEMIA

Anatomy

3 Arteries contribute to Mesenteric Circulation → Rich Collateral network → Chronic ischemia well tolerated

1) Celiac trunk

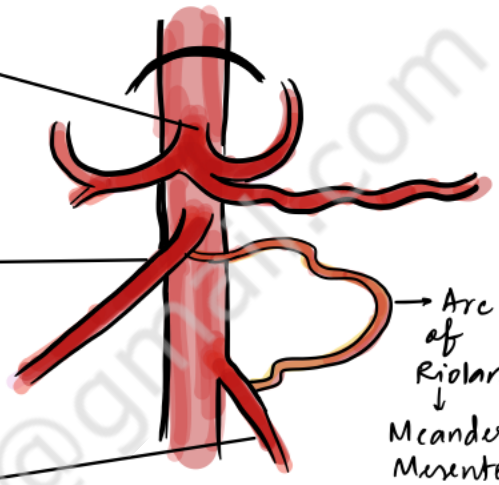
- Common Hepatic A
  - Gastric A
- Splenic A

- Supplies - Foregut (distal esophagus to duodenum)
  - Hepatobiliary system
  - Spleen

2) Superior mesenteric Artery

- Jejunal & ileal branches
- Ileocolic A
- Right colic A
- Middle Colic A

- Supplies Midgut
  - jejunum to mid colon



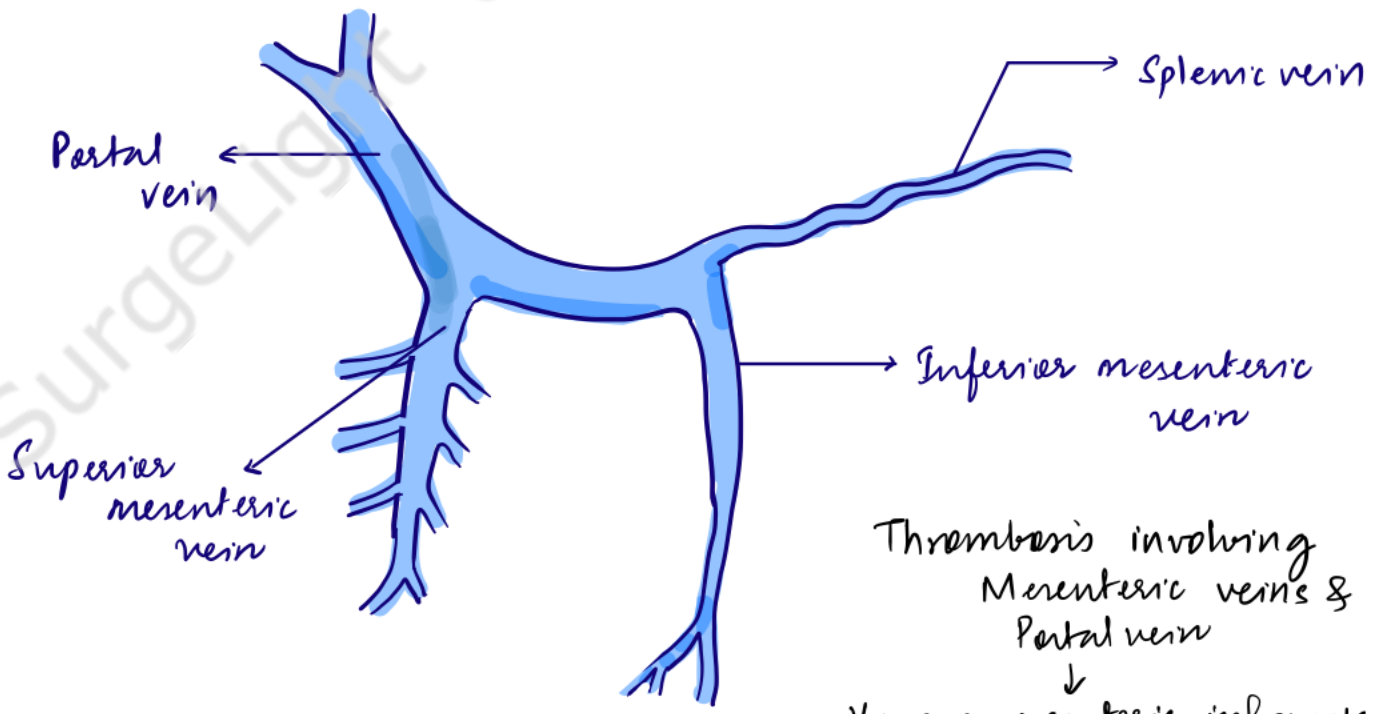
Arc of Riolan → Meandering Mesenteric artery

3) Inferior mesenteric Artery

- L. Colic A
- Sigmoid arteries
- Superior rectal A

- Supplies Hindgut
  - mid colon to rectum

VENOUS ANATOMY



Thrombosis involving Mesenteric veins & Portal vein → Venous mesenteric ischemia

SurgeLight chandanasri@gmail.com

Collaterals

1) Between CT & SMA - Superior & inferior pancreaticoduodenal arteries
2) Between SMA & IMA
   - Arc. of Riolan
   - Marginal A of Drummond
   - Un-named retroperitoneal vessels
3) Between IMA & internal iliac
   - Hypogastric As
   - Hemorrhoidal As

Regulation of Mesenteric blood flow

- Hormonal → Splanchnic vasodilators
- Neural → Enteral autonomic nerves

PATHOPHYSIOLOGY OF MESENTERIC ISCHEMIA

↓ Mesenteric blood flow

← Post prandial ↑ O_2 demand

Demands not met by GI collaterals

↓

- Anaerobic metabolism
- Acidosis

↓

Mucosal compromise → Full thickness necrosis

↓

PERFORATION

TYPES OF MESENTERIC ISCHEMIA

ARTERIAL

- ACUTE
  - EMBOLIC
  - THROMBOSIS
- CHRONIC
- NOMI

M/C involved vessel → SMA

↓

esp in acute mesenteric ischemia

VENOUS

- ACUTE MESENTERIC VENOUS THROMBOSIS → generally a/c bowel ischemia
- CHRONIC MESENTERIC VENOUS THROMBOSIS → Generally a/c Portal HTN Variceal complications

Causes

- Primary mesenteric thrombosis
  - Hypercoagulable states - Drugs
  - Malignancy
- Inflammatory conditions - 2°
  - Pancreatitis, Malignancy, Infections

surgeonlightchandrasi@gmail.com

ARTERIAL MESENTERIC ISCHEMIA

ACUTE MESENTERIC ISCHEMIA

EMBOLIC

emboli generally from a CARDIAC SOURCE (Eg: A.fib, MI & thrombus)

↓

lodge in mesenteric circulation

↓

m/c site - Middle colic artery origin - SMA

↓

mesenteric emboli

↓

'MENISCUS SIGN' on mesenteric arteriogram

THROMBOTIC

↓

Acute thrombosis occurs in sites with pre-existing mesenteric atherosclerosis

↓

m/c involves ORIGIN of SMA from abdominal aorta

↓

shows abrupt cutoff of SMA close to origin on arteriogram

CHRONIC MESENTERIC ISCHEMIA

Progressive luminal obliteration due to atherosclerosis (m/c)

↓

presentation is insidious 2/t extensive collaterals

CFs: 1) Post prandial abdominal pain

2) Food fear

3) Weight loss

Symptomatic when critical stenosis in ≥2 of 3 vessels

arteriogram - collaterals

NON-OCCLUSIVE MESENTERIC ISCHEMIA (NOMI)

characterised by a LOW FLOW STATE in otherwise NORMAL MESENTERIC ARTERIES

↓

Generally occurs in CRITICALLY ILL PATIENTS & HYPOTENSION ON VASOPRESSORS

↓

Splanchnic vasospasm

↓

Ischemia

arteriogram - vasospasm

Other causes of Mesenteric Ischemia

1) Median Arcuate ligament Syndrome / Celiac Artery Compression Syndrome

Compression of celiac trunk by MEDIAN ARCUATE LIGAMENT → address this before endovasc Rx - prefer open Rx

Rx - ARCUATE LIGAMENT DECOMPRESSION ± CELIAC PLEXUS SYMPATHECTOMY

2) Aortic Surgeries - ligation of IMA → Ischemic colitis

↳ especially when collaterals are poorly developed

3) Mesenteric arteritis

4) Radiation arteritis

5) Cholesterol emboli

Surgeright chandanasri@gmail.com

CLINICAL FEATURES

- Abdominal pain out of proportion to physical findings
  - ↳ in acute mesenteric ischemia
    - sudden onset of cramps
    - ↑/↓ bloody diarrhea (mucosal sloughing 2/t ischemia)
- Fever, nausea, vomiting, distension, tenderness, rebound, rigidity
  - → late signs → indicate bowel infarction + necrosis
- Chronic – insidious – postprandial pain

EVALUATION

- R/o other causes – Perforation, obstruction, pancreatitis etc.
- CBC – hemoconc, leucocytosis, METABOLIC ACIDOSIS
  - ↑ LACTATE
  - HYPERKALEMIA
  - AZOTEMIA
- X Ray – 'Pneumatosis intestinalis' / Gas in portal vein } ⇒ Infarcted bowel
  - Adynamic ileus + gasless abd
- USG – Duplex – ↑ Peak systolic velocity in SMA – stenosis
- Mesenteric Arteriography
  - Meniscus sign – embolus
  - Cut off – thrombus just beyond origin
  - Vasospasm – NOMI
  - ++ Collaterals – Chronic
  - ↓ DO NOT DO IF ACUTE SIGNS ⊕ → Priority is to open, explore & salvage bowel!
- Cardiac – ECG – A fib / MI
  - Echo – LV thrombus

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MANAGEMENT

- Resuscitate
- Systemic Anticoagulation ĩ Heparin
- Rx of Metabolic acidosis - $NaHCO_3$

SURGERY

1) ACUTE EMBOLIC MI

OPEN ABDOMEN → Explore

→ Identify SMA at root of small bowel mesentery
↓
Vascular clamp to control SMA
↓
transverse arteriotomy
↓
Thromboembolectomy ũ Fogarty's
↓
Close arteriotomy → restore flow

100-% O_2

→ Assess bowel viability
↓
R&A as needed

→ 2nd look procedure if doubtful

2) ACUTE THROMBOTIC MI

→ may require reconstructive procedure as there is pre-existing underlying atherosclerotic disease

→ BYPASS → Autologous: Saphenous vein graft preferred

→ Prosthetic graft avoided in presence of bowel ischemia d/t risk of infection

INFLOW VESSELS

- SUPRACELIAC ABDOMINAL AORTA → difficult, better results
  ↓ Atherosclerosis
- INFRARENAL AORTA → easier, poorer results
- ILIAC ARTERY

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3) CHRONIC MESENTERIC ISCHEMIA

↓

Elective Sx to revascularize mesenteric circulation

OPTIONS – Trans-aortic endarterectomy
Mesenteric artery bypass

→ For ostial lesions & distally patent CA/SMA

→ Grafts – both prosthetic grafts and autologous vein

4) MEDIAN ARCUATE LIGAMENT SD

- Release of ligamentous compression of CA
- Correct persistent stricture → bypass grafting

ENDOVASCULAR Rx

1) Chronic Mesenteric Ischemia: short segment occlusions

↓

Balloon angio + stent

↑

Recurrent Anastomotic stenosis

can be done prophylactically in pts undergoing aortic endovascular procedure, if mesenterics are stenotic ++

2) Acute Mesenteric Ischemia → Catheter directed thrombolysis

urokinase
rTPA

within 12h of symptom onset without acute abdomen

↓

indication for emergency Sx

3) NOMI → Cath directed selective mesenteric inj of vasodilators

PAPAVERINE
TOLAZOLINE

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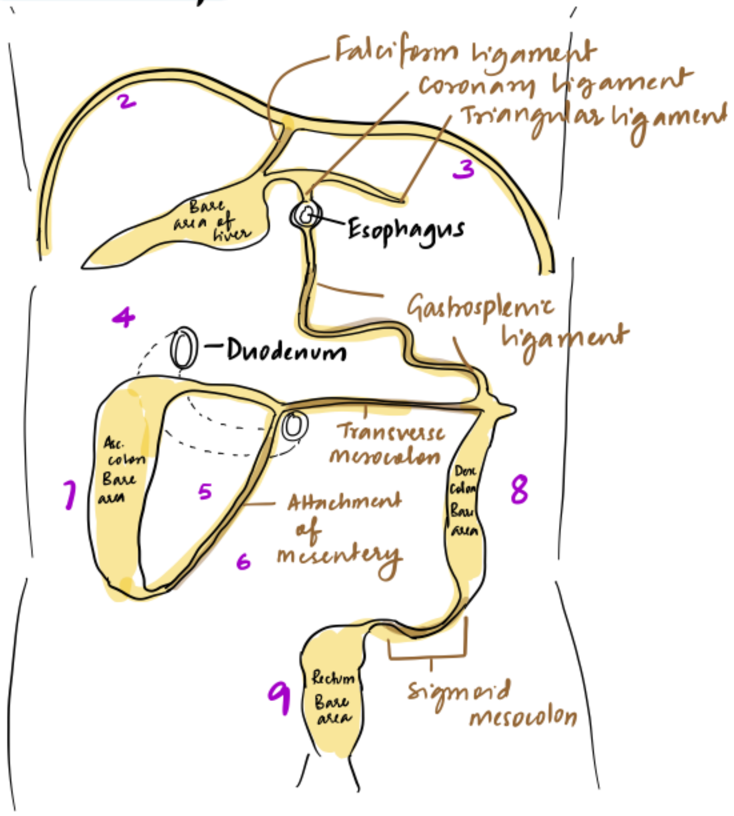
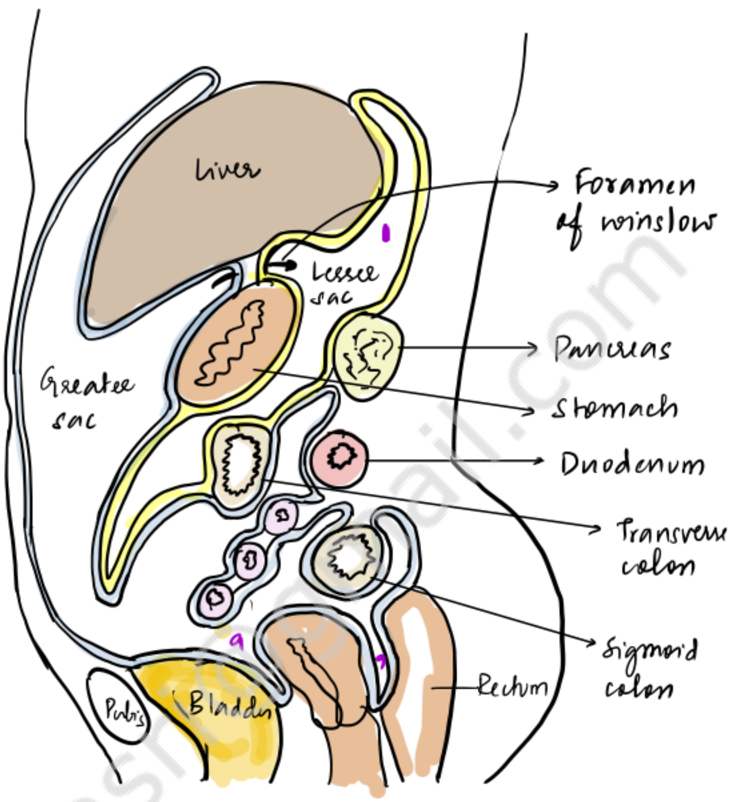
Peritoneal Spaces

Anatomy





12 (Ligaments + Mesenteries)

1) Coronary
2) Gastrohepatic
3) Hepatoduodenal
4) Falciform
5) Phrenicocolic
6) Gastrocolic
7) Duodenocolic
8) Gastrosplenic
9) Splenorenal
10) Small bowel mesentery
11) Transverse mesocolon
12) Sigmoid mesocolon

9 Potential Spaces

1) Lesser sac - Pseudocyst of pancreas
2) (R) Subphrenic
3) (L) Subphrenic { Subphrenic abscess L > R

Collectively known as "Subphrenic spaces"

4) Subhepatic - Hepatorenal pouch - most dependent in supine position
5) Supramesenteric
6) Inframesenteric
7) (R) Paracolic gutter → tracking - Valentino so
8) (L) Paracolic gutter
9) Pelvis - POD, R V pouch

Extraperitoneal Spaces

- (R) → (R) Perinephric space
- Middle → Bare area of liver
- (L) → (L) Perinephric space

Abscesses
Hematoma

Barnard's aphorism - Pus somewhere! Pus Nowhere else! Pus under the diaphragm

Hoover's sign: Spine deviates to the side of subphrenic abscess

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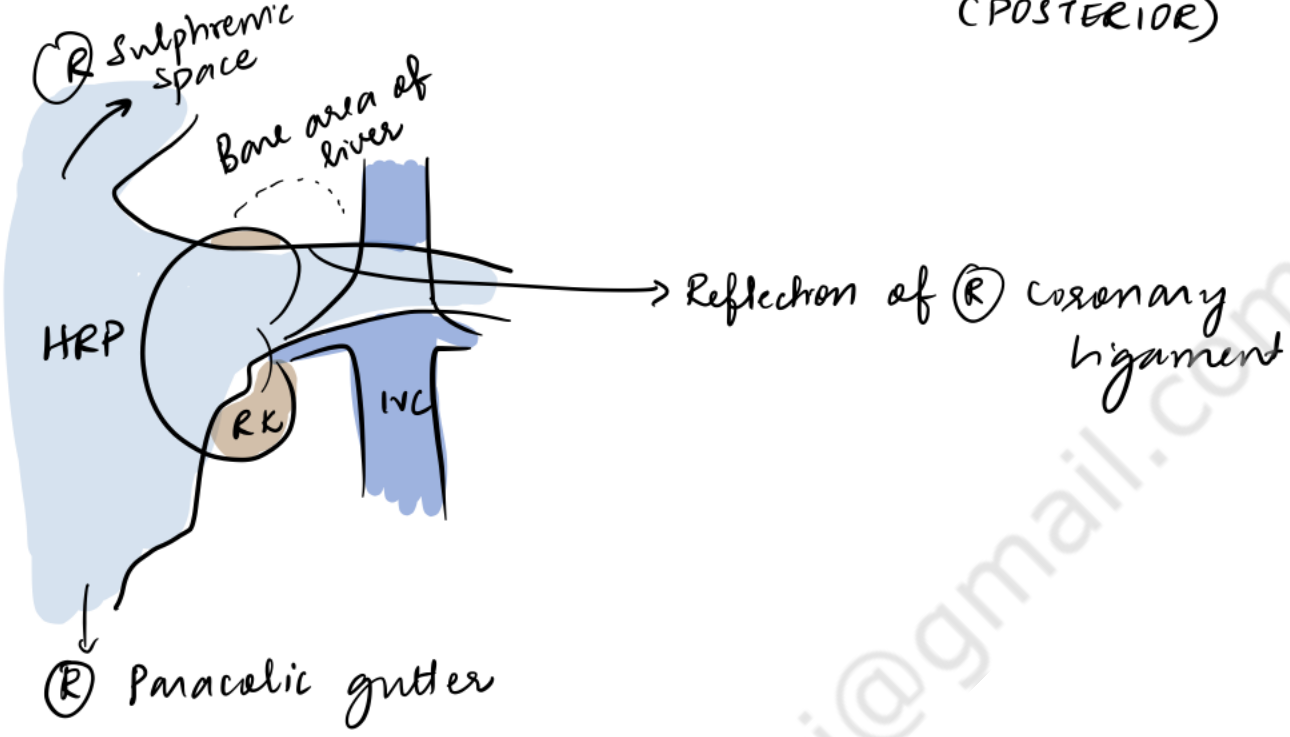
MORRISON'S POUCH / HEPATORENAL POUCH / SUBHEPATIC SPACE (POSTERIOR)



Boundaries

Inferiorly - hepatic flexure
Medially - 2nd part of duodenum
Superolaterally communicates with Ⓡ subphrenic space
Inferiorly, continues as Ⓡ paracolic gutter

Significance - most dependent part of the peritoneal cavity when supine

Surgeonlight.chandanasri@gmail.com

RETROPERITONEUM

ANATOMY

Retroperitoneal space – from diaphragm to pelvic floor (levator ani)
(T_{12}, 12th rib)
Anteriorly – peritoneum
Posteriorly – Posterior parietal wall of abdominal cavity
– Spine and paraspinal muscles

2 PARTS

LUMBAR FOSSA

From T_{12} to · base of sacrum
· iliac crest
· iliolumbar ligament

↓

Posterior wall is internally lined by fascia covering the quadratus lumborum & psoas major

Contents : Fatty areolar tissue
Adrenals
Kidneys
Ascending & descending colon
Duodenum
Ureters
Renal vessels & Gonadal vessels
IVC & Aorta

ILIAC FOSSA

Contiguous ε
– Lumbar fossa superiorly
– Lateral & anterior preperitoneal spaces
– Pelvis inferiorly

Posterior wall – iliacus muscle with its investing fascia

Contents
– Iliac vessels
– Ureters
– Genitofemoral nerve
– Gonadal vessels
– Iliac lymph nodes

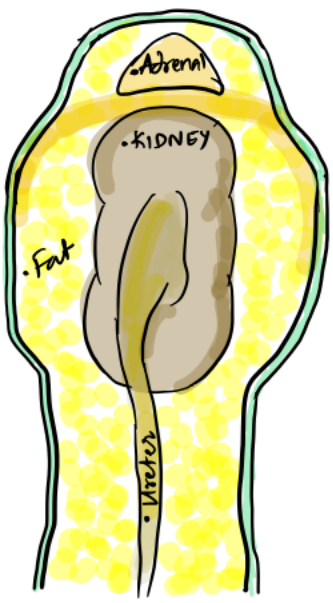
SPACES

ANTERIOR PARARENAL SPACE

Peritoneum

– Ascending and descending colon
– Duodenum
– Pancreas

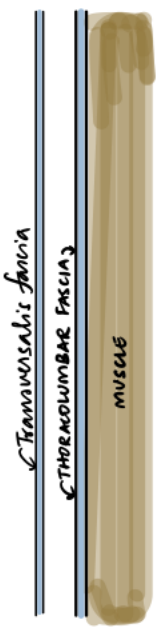
PERIRENAL SPACE



• IVC
• Aorta

POSTERIOR PARARENAL SPACE

↓
in continuity ε the preperitoneal space
↓
Contains fat



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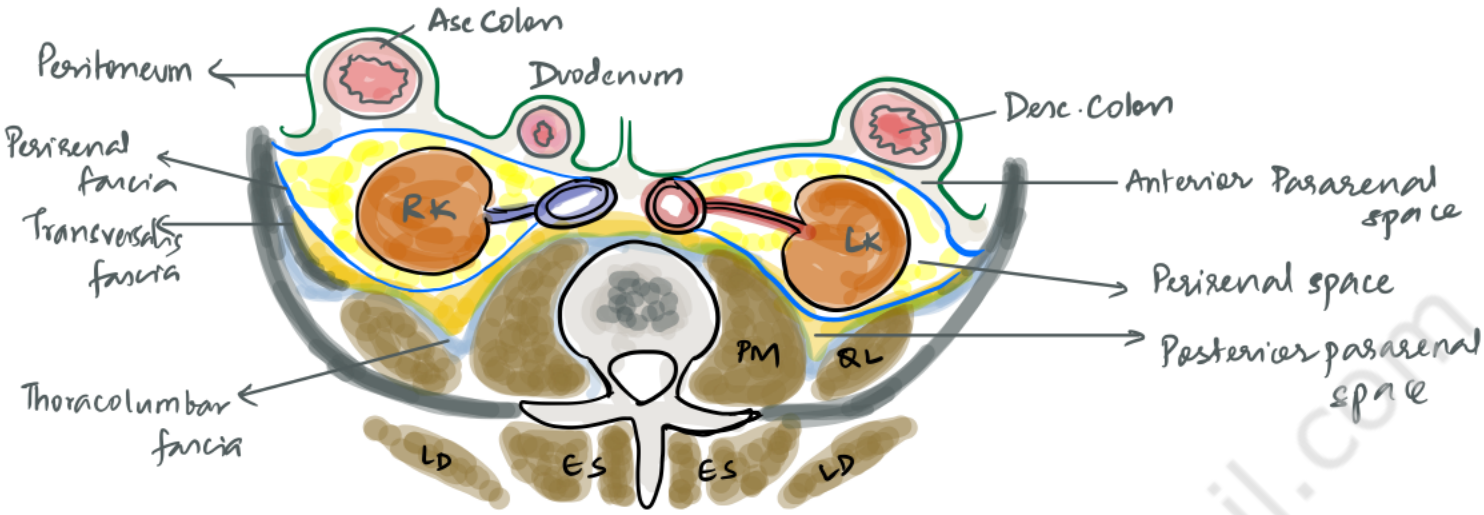
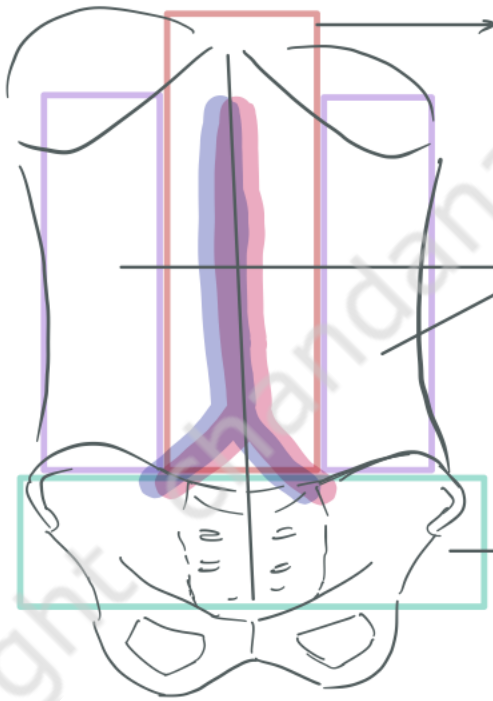


Zones of the Retroperitoneum

Significant in the context of trauma

Penetrating injuries in any zone → explore

Even Blunt injury in centromedial zone - explore



Zone I – Centromedial
- Abdominal aorta
- IVC
- Pancreas
- Duodenum

Bifurcation

Zone II – Lateral
- Kidneys & adrenals
- ureters
- Asc & Desc. colon

Zone III – Pelvic
- Pelvis
- Iliac vessels
- Rectosigmoid

Nerves of the retroperitoneum

1) Iliohypogastric
2) Ilioinguinal
3) Genitofemoral
4) Lateral femoral cutaneous
5) Obturator
6) Femoral

SurgeLight.friendamasri@gmail.com

RETROPERITONEAL LYMPHATICS

AORTIC GROUP
- Preaortic - near celiac A
- Retroaortic
- Para-aortic

CAVAL GROUP
Precaval
Retrocaval
Paracaval

Aortocaval

PELVIC GROUP
Common iliac
External iliac
Internal iliac / hypogastric
Obturator
Sacral

APPROACHES TO THE RETROPERITONEUM

① **Intraabdominal transperitoneal**

CATTELL MANEUVER (Cattell Braasch)
↓
for (R) sided structures
& INFRARENAL GREAT VESSELS

Incise lateral peritoneum along cecum, ascending colon & hepatix flexure
- Divide white line of Toldt
- Kocherization

Mobilise (R) sided structures anteromedially

MATTOX MANEUVER
↓
for (L) sided structures
& SUPRARENAL GREAT VESSELS

- Incise lateral peritoneum along sigmoid, descending colon & splenic flexure
- Divide white line of Toldt
- mobilize spleen, pancreatic tail, stomach
- Mobilise (L) sided structures anteromedially

② Posterior approach - preferable i/v/o ↓ post-op ileus
↓ intraabdominal adhesions
↓ Respiratory complications

③ Laparoscopic approach

④ Retroperitoneoscopic approach

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RETROPERITONEAL ABSCESS

Primary

↓

Of infection occurs due to hematogenous spread

↓

Systemic sepsis

↓

Monomicrobial

- Staph. aureus m/c

Secondary

↓

Due to infection in an adjacent organ

↓

m/c

Sources of infection

1) m/c - Renal - as a complication of urolithiasis - pyonephrosis
or UTI
Urological procedures
pyelonephritis
↓
Perinephric abscess
↓
spreads renal fascia
↓ bursts
tracks

47%

E. Coli
Proteus mirabilis
- Generally MONOMICROBIAL

2) GI - ~16%
Retrocaecal appendicular abscess
Diverticulitis
Crohn's Disease
Pancreatitis

E. Coli, Enterococci, Backeroides
↓
MULTIMICROBIAL

3) Spine / Bone infections - TB spine

4) Trauma, Complications of surgical procedures

Clinical features

A) OF SEPSIS - fever, malaise, weight loss

B) SPECIFIC TO LOCATION - Abdominal / flank pain - radiates to groin, labia, scrotum
Psoas abscess - referred pain - hip, thigh, knee
↳ can track upto thigh
LFCN — ON

Imaging - CECT

Rx

1) Abx
2) Drainage - Percutaneous / surgical
3) Rx of cause

SurgeLight drdanasri@gmail.com

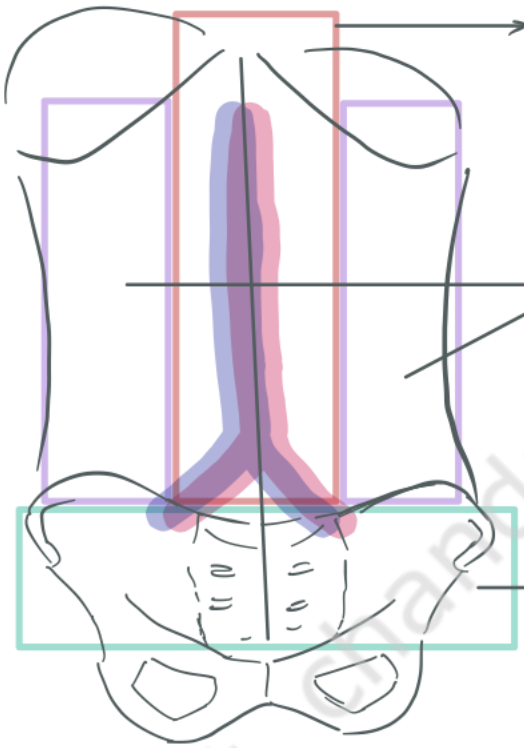
RETROPERITONEAL HEMATOMA

Causes

- Blunt / Penetrating trauma - Spleen, Kidney, vessels
- Abdominal aortic / visceral arterial aneurysms - rupture
- Acute / chronic anticoagulation / fibrinolytic therapy
- Blood dyscrasias

Evaluation

Zones:



Zone I - Centromedial
- Abdominal aorta
- IVC
- Pancreas
- Duodenum

Bifurcation

Zone II - Lateral
- Kidneys & adrenals
- ureters
- Asc & Desc. colon

Zone III - Pelvic
- Pelvis
- Iliac vessels
- Rectosigmoid

Centromedial / Zone I injuries i hematoma need surgical exploration as there is likelihood of injury to great vessels

Zone II - Predominantly renal source - explore in presence of H/S/O expanding hematoma

Zone III bleeding - usually results from pelvic # - should not be explored unless exsanguinating hemorrhage is obvious

SurgeLight drdanasri@gmail.com

RETROPERITONEAL FIBROSIS

- Rare disease - inflammation & fibrosis of retroperitoneal tissue

Primary / idiopathic (70%)

ORMOND'S DISEASE

- Immune mediated
- May be associated w other autoimmune conditions - ANKYLOSING SPONDYLITIS
  SLE
  WEGENER'S
  PAN
  IgG-4 disease
- HLA-DRB1-03 allele association
  Type 1 DM
  Myaesthenia gravis
  Hashimoto
- 2:1 - M:F
- 50-60y

Secondary (30%)

- Aortic aneurysms
- Pancreatitis
- Drugs
  - Ergot alkaloids
  - β blockers
  - Hydralazine
  - Methyl-dopa
- Malignancies
  - Lymphoma
  - Carcinoid
  - Sarcoma
  - Colorectal Ca
  - Breast Ca
- Infections - TB
- Radiation
- Retroperitoneal hematoma
- Surgery
- Tobacco use
- Asbestos exposure

Starts as chronic peri-aortitis → affects retroperitoneal tissue near the large arteries ⟶ expanding fibrosis - encases ureters
IVC
Aorta
Mesenteric vessels
Sympathetic nerves

B/L involvement - 70%

Clinical features - Pain, anorexia, wt. loss, fever
- URETERAL OBSTRUCTION - ↓ U/O, HUN
- Renal artery impingement - HTN
- Lower extremity edema, ↓ LL pulses - Iliac vessels involved
- New hydrocele - lymphatic obstruction
- New varicocele - Gonadal vessel involvement

Dx - ↑ESR, CRP
↑ANA

CECT - fibrotic RP tissue - similar attenuation to muscle
Contrast - degree & extent of encasement

Rx - Ureteral obstruction - URETEROLYSIS, STENTING → fail → surgery
IRPF - Rx - Corticosteroids / Tamoxifen / MTX / Azathioprine / Cyclophosphamide

SurgeLight chandanasri@gmail.com

RETROPERITONEAL TUMOR

- Extracapsular growth of a PRIMARY NEOPLASM of a RETROPERITONEAL ORGAN - Kidney, Adrenal, Colon, Pancreas
- Primary Germ cell neoplasms → from embryonic rest cells
  - ↑ FP, hCG, LDH, testicular examination
- LYMPHOMA from Retroperitoneal lymphatic system
  - 'B' symptoms, Other LN groups
- METASTASIS TO Retroperitoneal lymph nodes
- TUMOR of soft tissue of the Retroperitoneum
  Eg: Sarcoma, Desmoid tumor

m/c primary malignant neoplasm of retroperitoneum - SARCOMA

RETROPERITONEAL SARCOMA

m/c - Liposarcoma, Leiomyosarcoma

Risk factors: Radiation
NF-1 - Neurofibroma → malignant transformation → MPNST
Li fraumeni S°
Hereditary RB

Clinical features: Asymptomatic abdominal mass
pain
Weight loss
lower extremity edema / paraesthesia / paresis

Evaluation

HPE
Chest, abdominal, pelvic CT / MRI ± contrast

Staging

T
- Tx - cannot be assessed
- T0 - no e/o 1°
- T1 - < 5 cm
- T2 - 5-10 cm
- T3 - 10-15 cm
- T4 - > 15 cm

N
- N0 - no regional nodes
- N1 - regional nodal mets

M
- M0 - no distant metastasis
- M1 - Distant metastasis

G (Tumor diff, necrosis, mitotic count)
- Gx - can't be assessed
- G1 - TNM score 2-3
- G2 - 4-5
- G3 - 6, 7, 8

I A - T1 N0 M0 Gx, G1	II - T1 N0 M0 G2, G3	III A - T2 N0 M0 G2, G3
I B - T2,3,4 N0 M0 Gx, G1		III B - T3,4 N0 M0 G2, G3

IV - Any T, Any N, M1, Any G

Rx - Surgery ± IORT → RT / Chemo

Surgeonlight chandanasri@gmail.com

SHORT BOWEL SYNDROME

Definition: loss of critical bowel length required for the maintenance of enteral nutrition

- SBS occurs when there is **< 200cm** of small intestine remaining (<50-70%)
- Minimal length of small intestine required to prevent lifelong dependence on parenteral nutrition
  - **100 cm** - if colon is absent
  - **60 cm** with a completely functional colon

ETIOLOGY

Congenital
Intestinal atresia

Acquired
- Surgical resection of bowel

PEDIATRIC	ADULTS
necrotizing enterocolitis	Crohn's disease
Gastroschisis	Mesenteric ischemia
Midgut volvulus	Late stage small bowel obstruction
Complicated Meconium ileus	trauma
Extensive aganglionosis	Tumors

- Radiation enteritis
- Congenital villous atrophy
- Refractory sprue
- Chronic intestinal pseudoobstruction syndrome

PATHOPHYSIOLOGY

INTESTINAL ADAPTATION - mechanism of GI tract functional recovery that occurs in post-resection state of SBS

- Begins within 24 hr of significant intestinal resection and continues over a 2 year period
- 3 phases

ACUTE PHASE
1-3 m
- Dehydration
- Electrolyte imbalance
- Nutrional deficiency
- Hypergastrinemia
- Hyperbilirubinemia

Diarrhea, Malabsorption, Dysmotility

ADAPTIVE PHASE
1-2 y
- Enterocyte hyperplasia
- Villous hyperplasia
- ↑ Crypt depth

↑ surface area

Intestinal dilatation & lengthening

Requires luminal & parenteral nutrition to succeed

MAINTENANCE PHASE
long term
- Absorptive capacity peaks
- Nutritional & metabolic homeostasis by varying degrees of nutritional support

SurgeLight Chandana@gmail.com

POOR PROGNOSTIC INDICATORS IN SBS

- <200cm residual bowel
- Resection of Ileocaecal valve
- Distal small bowel resection
- Concomitant colonic resection
- Diseased remaining bowel

ANATOMIC SUBTYPES OF SBS

TYPE I
Jejunoileal anastomosis

- Resected: Most of jejunum
- Remnant: Ileum, ICV, Colon

- Impaired digestion
- Hypersecretion of acid
- Prognosis fair, but poor if <40cm of jejunum remains

DIARRHEA
↓
Best chance of recovery & good intestinal adaptation

- Need for permanent TPN / small bowel transplant VERY UNLIKELY

Rx: H_2 BLOCKERS / PPIs

TYPE II
Jejunocolic anastomosis

- Resected: Most of ileum
- Remnant: Jejunum, Colon

- Deficiencies in
  - Vit B_{12}
  - Bile salts
  - Fat
  - Fat soluble vitamins

Variable prognosis
Poor if <65cm jejunum remains

DIARRHEA
STEATORRHEA
↓
Loss of adaptive capacity of ileum
↓
Needs long term B_{12} & fat soluble vitamin supplementation

TYPE III
END JEJUNOSTOMY

Some jejunum retained, everything else resected

- Deficiencies in
  - Vit B_{12}
  - Bile salts
  - Fat
  - Fat soluble vitamins
  - Magnesium
  - Fluids
  - Electrolytes
  - Nutrients

DEHYDRATION
↑ OSTOMY OUTPUT
↓
Poorest prognosis
↓
Most likely to require permanent PN

Complications of SBS:

1) Small Bowel Bacterial Overgrowth (>10^5 CFU/ml in SI) → on endoscopic capture culture
   d/t altered GI structure & function
   Rx - Metronidazole, Probiotics
2) Catheter related infections
3) Liver disease - long term Parenteral nutrition - progressive intestinal failure
   CHOLESTASIS → STEATOSIS → FIBROSIS / CIRRHOSIS

SurgLight chaitanasri@gmail.com

MANAGEMENT

① MEDICAL MANAGEMENT

Goal:

Optimization & maintenance of
- Nutritional absorption
- Fluid & electrolyte imbalance
- Vitamin & trace element retention
- Nutritional & Weight maintenance

ACUTE PHASE
- IV fluid replacement
  - RL, 5D
- Replacement of water soluble vitamins & trace elements
- Rx of Gastric acid hypersecretion
  - H_2Bs / PPIs
- Octreotide (esp III)
- Antimotility - loperamide
- Cholestyramine for cholerheic diarrhea

ADAPTIVE PHASE

Enteral nutrition with a goal of 30-40 kCal/kg/day
- amino acids, carbs, medium chain triglycerides, glutamine

Parenteral nutrition

MAINTENANCE PHASE

Nutritional therapy

In PN dependent pts:

GLP-2 analogs
- TEDUGLUTIDE - promote villous height & crypt cell mass & help in reducing PN requirements

② SURGICAL MANAGEMENT

A. PRIMARY PREVENTION OF SBS

→ Avoid extensive bowel resection by early intervention in
- MESENTERIC ISCHEMIA / EMBOLISM / THROMBUS
- BOWEL OBSTRUCTION

→ Bowel conserving approach using strictureplasties in chronic relapsing and segmental bowel diseases like Crohn's

After resection - accurate documentation of bowel resected
- Presence / absence of ICJ

Mature stoma close to mucous fistula to avoid extensive adhesiolysis during relaparotomy

SurgeEight chandanasri@gmail.com

B. SURGICAL OPTIMIZATION OF REMNANT SMALL BOWEL

PROCEDURES TO IMPROVE INTESTINAL FUNCTION

to mitigate the superadded complications of stomas strictures and adhesions in a pt c̄ SBS

- stoma reversal
- strictureplasty
- adhesiolysis

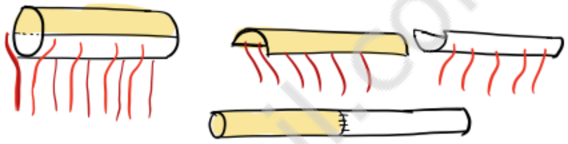
PROCEDURES TO PROLONG TRANSIT TIME

↑ transit time
↓
Improved absorption

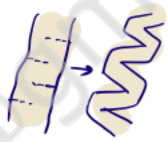
- Reversal of intestinal segment (5-15 cm) (antiperistaltic limb interposition)
- COLONIC INTERPOSITION (8-24 cm)
  - Isoperistaltic
  - Antiperistaltic
  - ∵ Colonic contractions are lower in frequency
- Intestinal tapering (comes at the cost of reduced total absorptive surface)

INTESTINAL LENGTHENING PROCEDURES

- Bianchi's LILT (longitudinal intestinal lengthening & tailoring)
  - principle - Bifurcated blood supply in 2 layers of mesentery



- STEP (Serial transverse enteroplasty)



- SILT (Spiral intestinal lengthening & tailoring)
- SMALL BOWEL TRANSPLANTATION
  in pts c̄ IFALD, PN failure, Recurrent CRBSIs

SurgeLight chandanasri@gmail.com

INTERNAL HERNIAS

Internal / Intra-abdominal hernias - visceral herniation through defects within the abdominal cavity

INTERNAL HERNIA DEFECTS

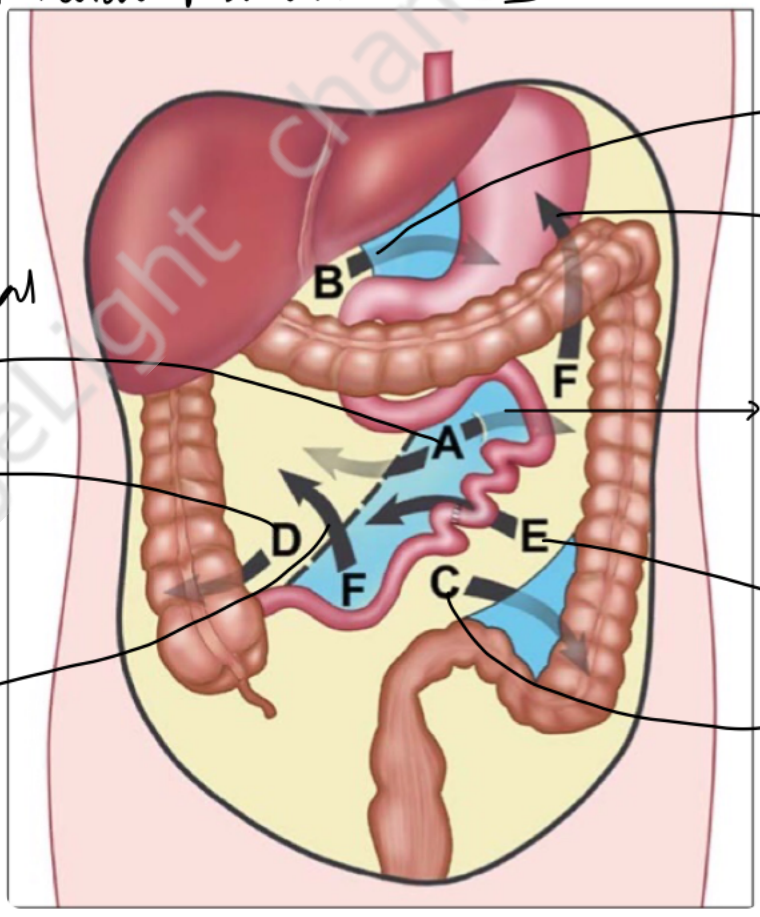
DEVELOPMENTAL / CONGENITAL

- DUE TO ABNORMAL MIDGUT ROTATION & FIXATION
  - PARADUODENAL / MESOCOLIC HERNIAS
    - (R) MESOCOLIC HERNIA
    - (L) MESOCOLIC HERNIA
- DUE TO ABNORMALLY LARGE INTERNAL FORAMINA / FOSSAE
  - FORAMEN OF WINSLOW HERNIA
  - PARAVESICAL HERNIA
    - ↳ not a true internal hernia
- ABNORMAL MESENTERIC DEFECTS
  - PERICECAL
  - INTER-SIGMOID

ACQUIRED

- Mesenteric defects created during
  - Bowel anastomosis
  - HJ
  - Roux-en-Y GB
    - 'PETERSON HERNIA'
- RETROANASTOMOTIC

[From 'Review of Internal Hernias' - Martin et al.



Foramen of Winslow Hernia

Peterson "A"

(R) Paraduodenal Hernia

(L) Paraduodenal - through a defect behind IMA near 4th part of duodenum

Paracecal

Retroanastomotic "A"

Trans mesenteric

Intersigmoid hernia

A = ACQUIRED

SurgeLight Chandanasri@gmail.com

PARADUODENAL HERNIA / MESOCOLIC HERNIA

- Small bowel herniates behind mesocolon
- Due to midgut malrotation

(R) MESOCOLIC / PARADUODENAL

→ Preartérial (duodenojejunal) limb of the midgut fails to rotate while post-arterial limb (Caecocolic loop) rotates

→ Defect in jejunal mesentery (WALDEYER FOSSA) behind SMA below 3rd part of duodenum

→ Most of the small bowel lies to the (R) of SMA between the 2 leaves of the ascending mesocolon

∴ SMA lies just medial to the neck of the sac

- Ileocolic, (R) colic, middle colic vessels lie on the anterior wall of the sac

Sac opened along lateral attachment of ascending mesocolon

(L) MESOCOLIC HERNIA

: Small bowel herniates through a defect between the IMA & the posterior attachment of the descending mesocolon - IN UTERO

The IMA forms the medial relation of the neck of the sac

Sac opened at the avascular plane just medial to IMA

→ M/C - ~75%

FORAMEN OF WINSLOW HERNIA

- small bowel > Caecum herniates through a large foramen of Winslow behind the free edge of the lesser omentum/ hepatoduodenal ligament

SurgeLight chandanasri@gmail.com

Clinical Features

1) Intestinal Obstruction - Acute / Subacute / Recurrent
2) Strangulation

- Often diagnosed intra-op

MANAGEMENT

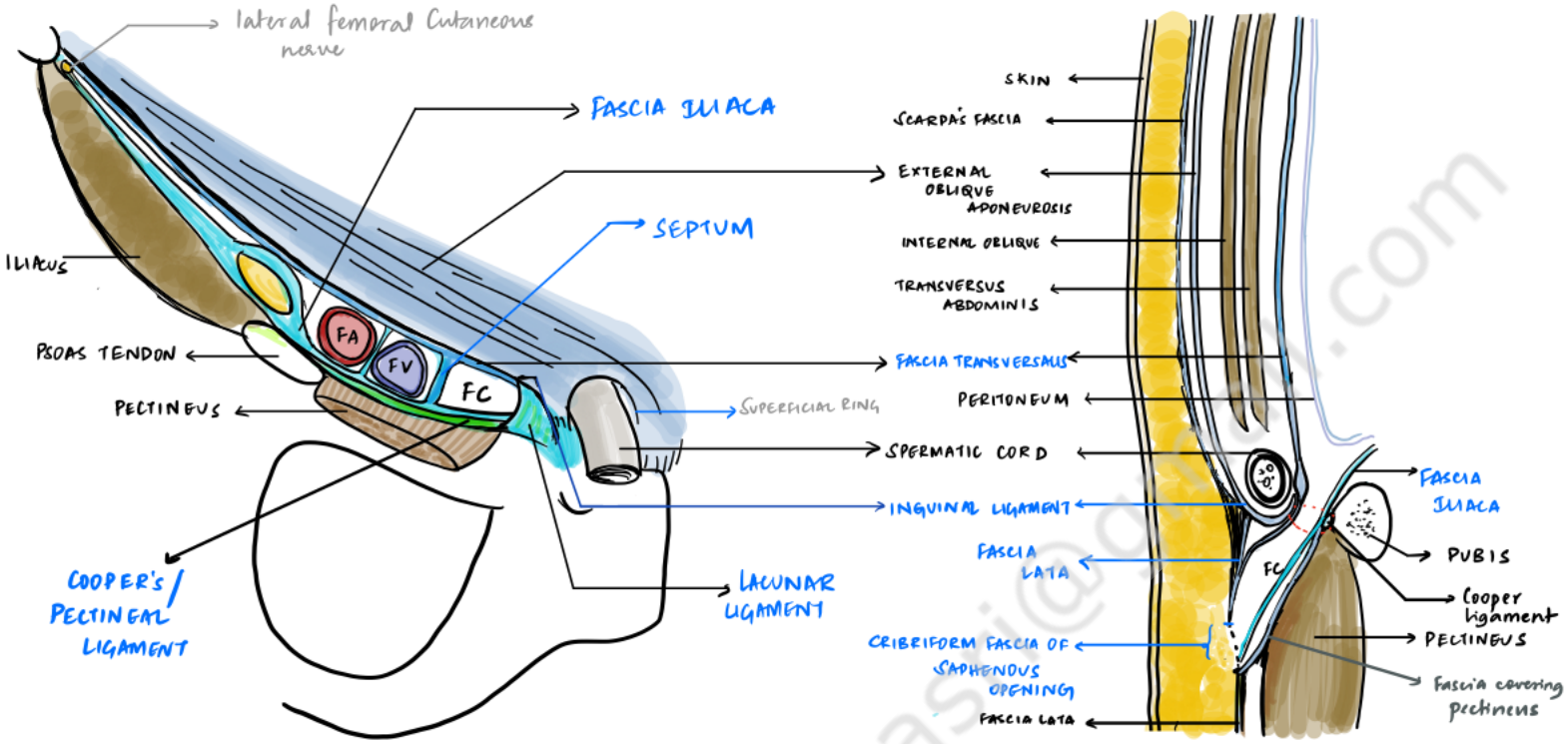
- Knowledge of anatomy and mechanisms important → whenever possible - reduction + defect closure
- (R) Mesocolic Hernia - Sac is opened along lateral attachment of ascending mesocolon
  ↓
  The (R) colon and cecum are reflected to the left
  ↓
  Assumes the position of NON ROTATION
- (L) Mesocolic hernia - incision of peritoneal attachments & adhesions along (R) side of IMV & delivery of herniated intestine from beneath IMV

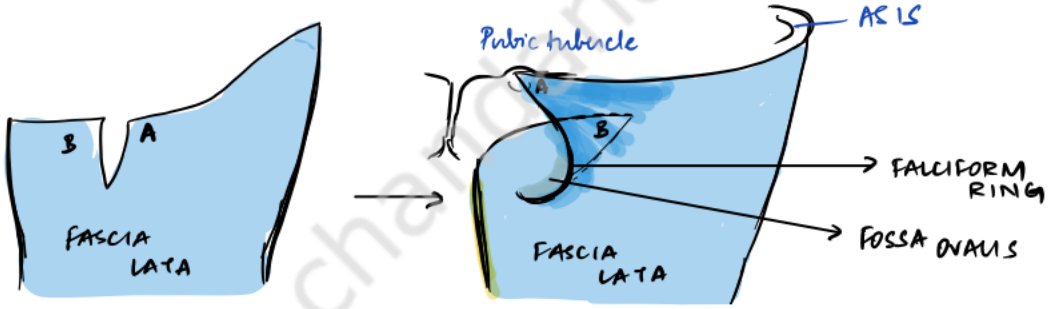
Pg 1084 - Sabiston 20e

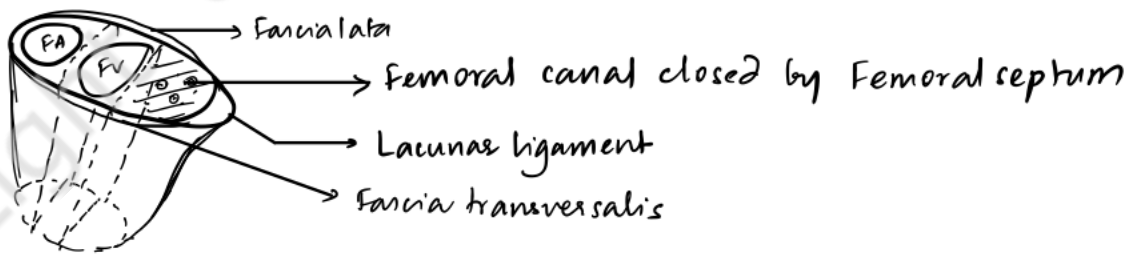
SunLight.chandanasri@gmail.com

FEMORAL HERNIA

ANATOMY OF FEMORAL CANAL







Femoral Canal

A space in the upper medial aspect of the thigh extending from the femoral ring above to the saphenous opening (Fossa ovalis) below

1.25 - 2cm in length

Femoral Sheath

Formed by 2 Fascial layers

→ Anterior layer - downward prolongation of FASCIA TRANSVERSALIS below inguinal ligament

→ Posterior layer - downward prolongation of FASCIA ILIACA behind femoral vessels

3 compartments ← Femoral canal, Femoral vein comp, Femoral A comp

Femoral ring

Upper limit of femoral sheath

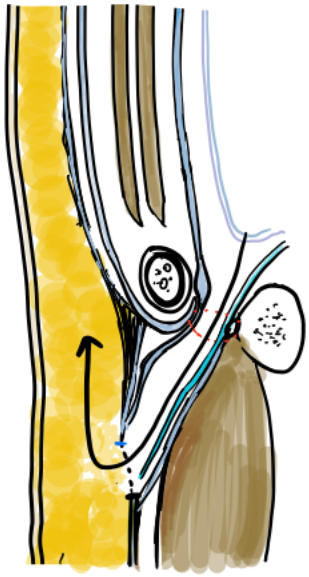
- MEDIALLY - LACUNAR LIGAMENT
- ANTERIORLY - ILIOPUBIC TRACT & INGUINAL LIGAMENT
- POSTERIORLY - COOPER'S LIGAMENT PECTINEUS MUSCLE
- LATERALLY - SEPTUM SEPARATING FEMORAL CANAL FROM FEMORAL VEIN

SurgeLit... chandnasi@gmail.com

Femoral Hernia

→ Abdominal contents pass through the femoral ring, traverse the femoral canal and exit through the saphenous opening

- Assumes a 'RETORT' shape



Course of Femoral Hernia

- Downwards upto the saphenous opening
- Upwards, once it exits the saphenous opening

Tendency of Femoral Hernia to extend upwards is attributed to

- Firm, unyielding falciform process
- Attachment of Scarpa's fascia to fascia lata just below saphenous opening
- Repeated flexion of thigh

COVERINGS OF FEMORAL HERNIA

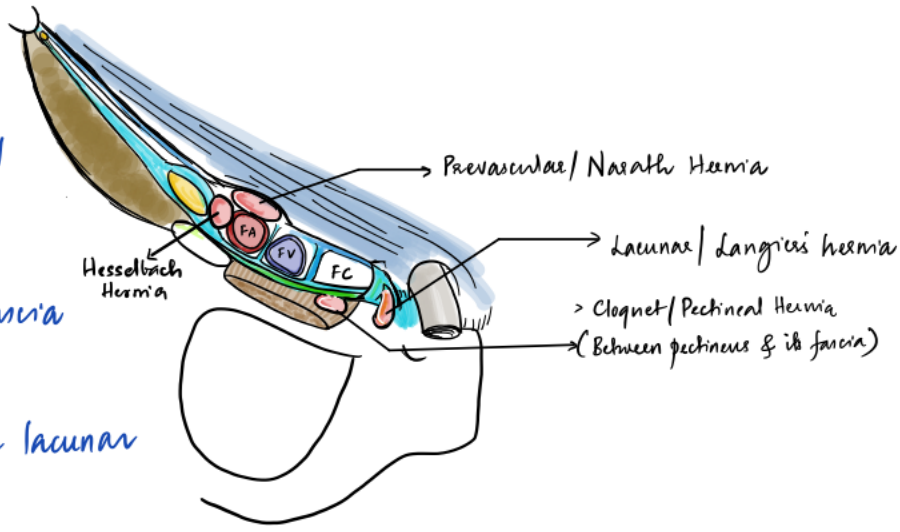
- Skin
- Superficial fascia
- Cribriform fascia
- Femoral septum & Fibrofatty tissue of femoral canal
- Fascia transversalis
- Peritoneum

RARE FORMS OF FEMORAL HERNIAE

1. PREVASCULAR / NARATH'S HERNIA:
   Hernia passes behind inguinal ligament in front of Femoral Artery
   Associated ε Congenital dislocation of hip

2. PECTINEAL / CLOQUET'S HERNIA:
   Hernia passes between pectineus & its fascia

3. LACUNAR / LANGIER'S Hernia:
   Hernia through a defect in the lacunar ligament



EPIDEMIOLOGY

- 3rd most common hernia, after inguinal & incisional hernia
- Women > Men
  → Pelvis, and therefore, femoral ring are wider in women, ↑ in multiparous ♀
- uncommon in children
- Majority >50y
- R : L :: 2 : 1 ; B/L in 20%

Surgeon.chandanasri@gmail.com

CLINICAL FEATURES

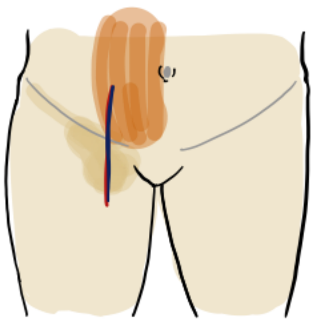
- Swelling below & lateral to pubic tubercle
  - Reducibility & cough impulse → less reliable signs of femoral hernia
    → d/t - adherence of contents
    - narrow neck of sac
- 40% present ĉ complication - obstruction | strangulation

Gaur sign - Distension of superficial epigastric &/or circumflex iliac vessels

SURGERIES FOR FEMORAL HERNIA

MC'EVEDY HIGH OPERATION

- Vertical incision over femoral canal ĉ upper portion ½ inch medial to linea semilunaris
- Anterior rectus sheath incised.
- Rectus abdominis retracted medially
- Fascia transversalis opened

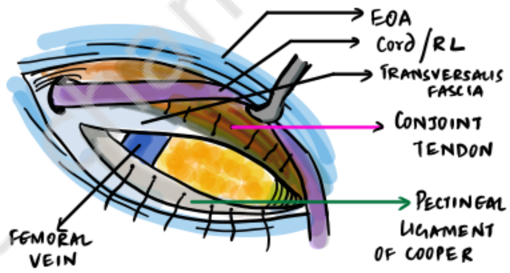


- Sac can be seen going underneath inguinal ligament
- It is dissected, opened and contents are assessed
- Peritoneal cavity can also be opened and assessed
- Femoral canal repaired from inside by approximating inguinal ligament to Cooper ligament without compromising femoral vein

PROCEDURE OF CHOICE FOR STRANGULATED FEMORAL HERNIA

LOTHEISEN OPERATION

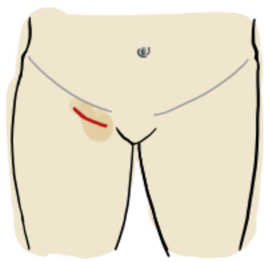
- Inguinal incision similar to inguinal hernia repair
- EOA opened, inguinal canal entered
- Cord structures retracted upwards

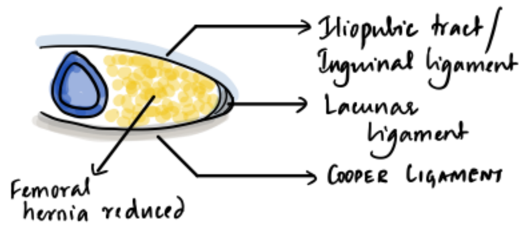


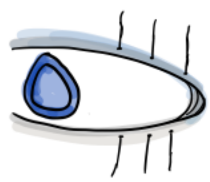
- Fascia transversalis incised
- Neck of the sac identified at the femoral ring
- Sac dissected, opened and reduced
- Femoral ring is obliterated by suturing the CONJOINT TENDON to pectineal ligament of Cooper

LOCKWOOD LOW OPERATION

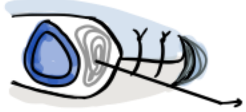
- Incision 1 cm below & parallel to the medial portion of inguinal ligament
- Sac isolated - freed upto neck & opened at fundus → care must be taken while incising lacunar ligament medially to free the sac [Aberrant obturator artery]







Interrupted sutures placed & tied from medial to lateral without compressing femoral vein



Remaining gap is reinforced with a plug of mesh

AK Henry - Lower abdominal incision for B/L Femoral Hernia

SurgeLight kandanasri@gmail.com

INFLAMMATORY BOWEL DISEASE - SURGICAL MANAGEMENT

Indications for Surgery in UC & CD

1) Acute onset of severe disease
   - Fulminant Colitis
   - Toxic Megacolon

2) Development of Disease Complications
   - Obstruction
   - Perforation
   - Complicated fistulas
   - Hemorrhage
   - Malignancy / Dysplasia

3) Failure of Medical therapy
   - Persistent symptoms despite long-term steroid use
   - Recurrence of symptoms when high-dose steroids are tapered
   - Drug induced complications

PRINCIPLES OF SURGERY

1) ULCERATIVE COLITIS

Procedure of choice = TOTAL PROCTOCOLECTOMY
- Rectum is always involved ∴ Resected
- UC - involvement is continuous ∴ Segmental resection is not justified

After total Proctocolectomy,
↓
Terminal ileum
- → ILEOSTOMY
  - → BROOK'S END ILEOSTOMY
  - → KOCH'S CONTINENT ILEOSTOMY
- → Ileal Pouch - Anal Anastomosis (IPAA)
  - → Pouch - J / S / H / W

SurgeLight chandanasri@gmail.com

2) CROHN'S DISEASE

Disease process is
- DISCONTINUOUS and SEGMENTAL
- RECTAL SPARING

∴ Preference is given to **BOWEL CONSERVATION**
- Only affected segments are operated usually
  1) Segmental resections
  2) Strictureplasties

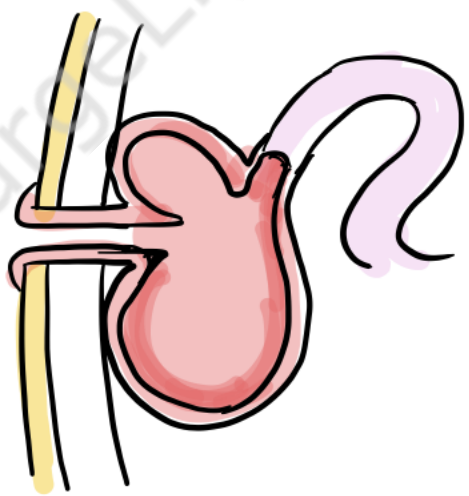
Extensive resections only i/c/o extensive involvement
No justification for prophylactic resection of unaffected bowel

Ileo-rectal anastomosis can be considered for reconstruction

- Ileal pouch
- Kochs continent Ileostomy

} NOT DONE in crohn's ↓ 2/t ↑ chances of involvement of ileum [unless care is very carefully selected]

KOCK POUCH / CONTINENT ILEOSTOMY



High volume, low pressure reservoirs created using terminal ileum

Continence mechanism
↓
Intussuscepted nipple valve

Allows fecal accumulation in reservoirs
↓
can be emptied at patients convenience at regular intervals by introducing a tube

SurgeLight chandanasrp@gmail.com

SURGERY FOR TOXIC MEGACOLON

A) Diversion Procedures

1) TURNBULL PROCEDURE - Multiple loop stomas to decompress & drain bowel
   - Loop ileostomy
   - Transverse colostomy
   - Sigmoid colostomy

2) LOOP ILEOSTOMY + COLONIC LAVAGE
   → through ileostomy, a malecot catheter is threaded into colon across ICJ
   - Colon lavaged c PEG fb abx irrigation

B) Total / Subtotal Colectomy c Hartmann Pouch or Mucous fistula
   ↓
   Proctectomy
   → generally avoided in emergencies & unstable pb as pelvic dissection is risky

---

RESTORATIVE PROCTOCOLECTOMY IN UC

Can be done as single / staged procedure

1) Single stage - TPC + IPAA
2) Double stage - TPC + IPAA + Proximal ileostomy
   fb - Ileostomy closure
3) Triple stage - TC + EI + Hartmann
   ↓
   fb - Completion proctectomy + IAA
   ↓
   EI closure

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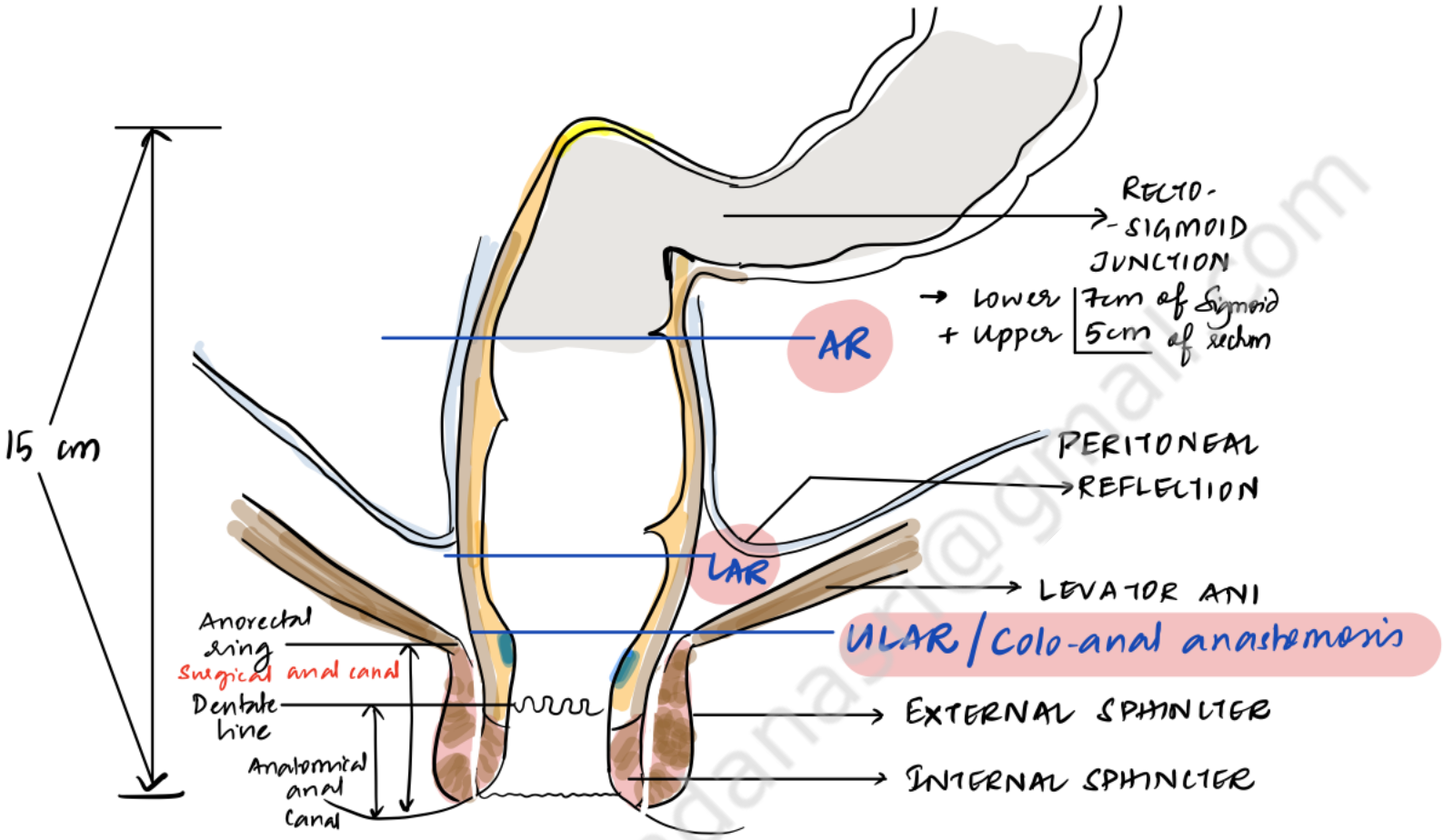
SURGICAL MANAGEMENT OF CA RECTUM

RESECTION PROCEDURES



Margins

- Generally, margins of 5cm on either side of the tumor are preferable
- Distal margin of 2cm → acceptable ONLY for tumors within 5cm of anal verge
  (some say 1cm)

Cancess with the lower edge ≤ 12cm from verge → total mesorectal excision is necessary

Extent of total mesorectal excision
→ minimum of 5cm distal to the lower edge of the tumor

Histological CRM in rectal cancer → ≥1mm

SurgeLight chandana@gmail.com

ANTERIOR RESECTION

- general term used to describe resection of the rectum FROM a purely ABDOMINAL APPROACH to the pelvis

HIGH ANTERIOR RESECTION: Resection of distal sigmoid + upper rectum
- Well above the pelvic peritoneal reflection (NOT DIVIDED)
- Primary colorectal anastomosis can be performed
- For tumors in proximal rectum / distal sigmoid (>12cm from verge)

LOW ANTERIOR RESECTION - Rectosigmoid is mobilized, pelvic peritoneum is opened, rectum is mobilized from sacral hollow
- Primary colorectal anastomosis lies below the level of peritoneal reflection
- for tumors in upper & middle rectum

ULTRA LOW ANTERIOR RESECTION - The distal limit of resection extends to the lower limit of rectum
COLOANAL anastomosis is performed
for tumors in lower rectum

ISR - INTERSPHINCTERIC RESECTION - distal dissection proceeds in the the intersphincteric plane - internal sphincter is removed in continuity ī rectum

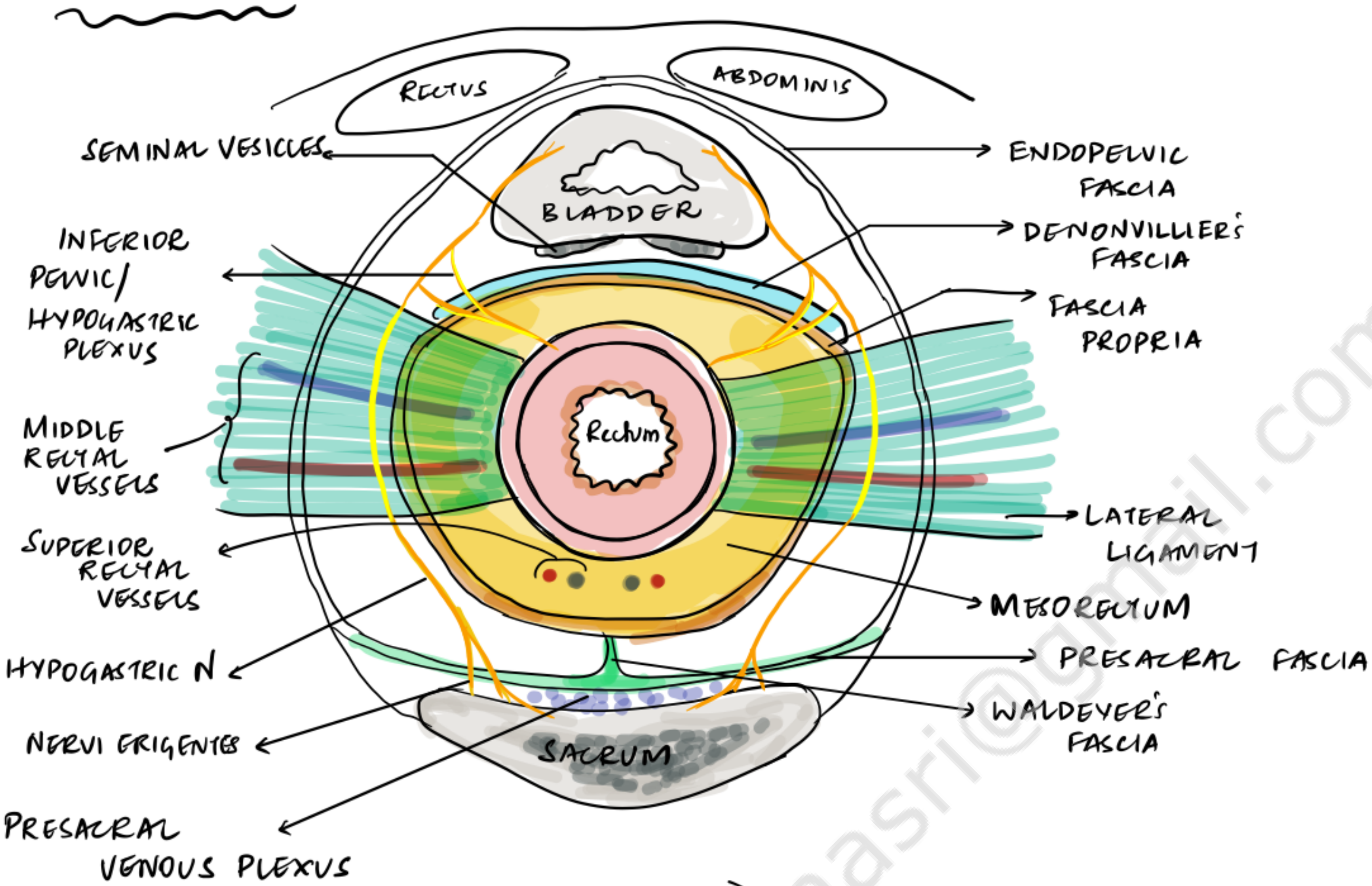
CONTINUITY RESTORED BY ANASTOMOSIS BETWEEN COLON & EXTERNAL SPHINCTER

ABDOMINOPELVIC RESECTION - removal of entire rectum, anal canal and anus + TME + construction of permanent descending / sigmoid colostomy

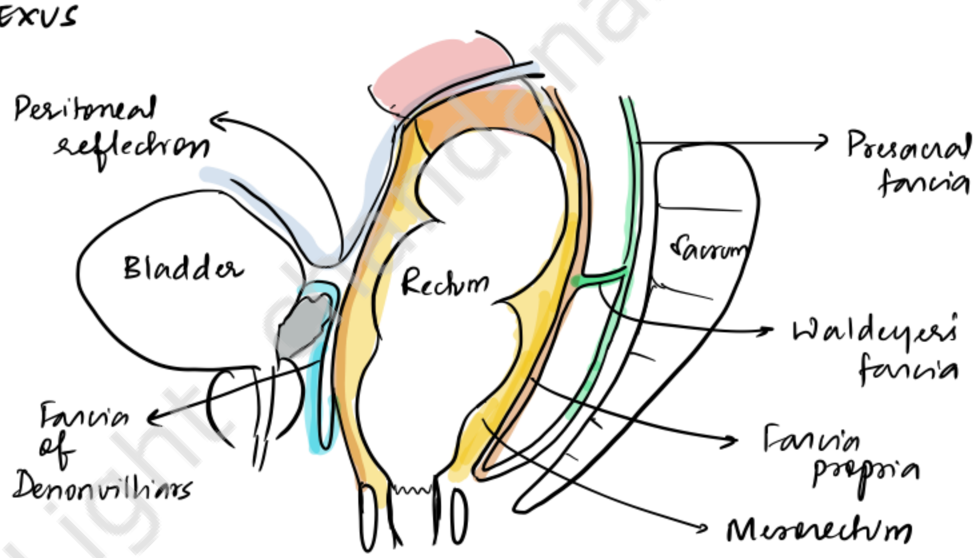
ELAPE - Extra Levator Abdominoperineal Excision:
↳ Levators, ischiorectal fossa & perianal skin are excised in continuity ī TME specimen to improve CRM quality

SurgeLight drandhasri@gmail.com

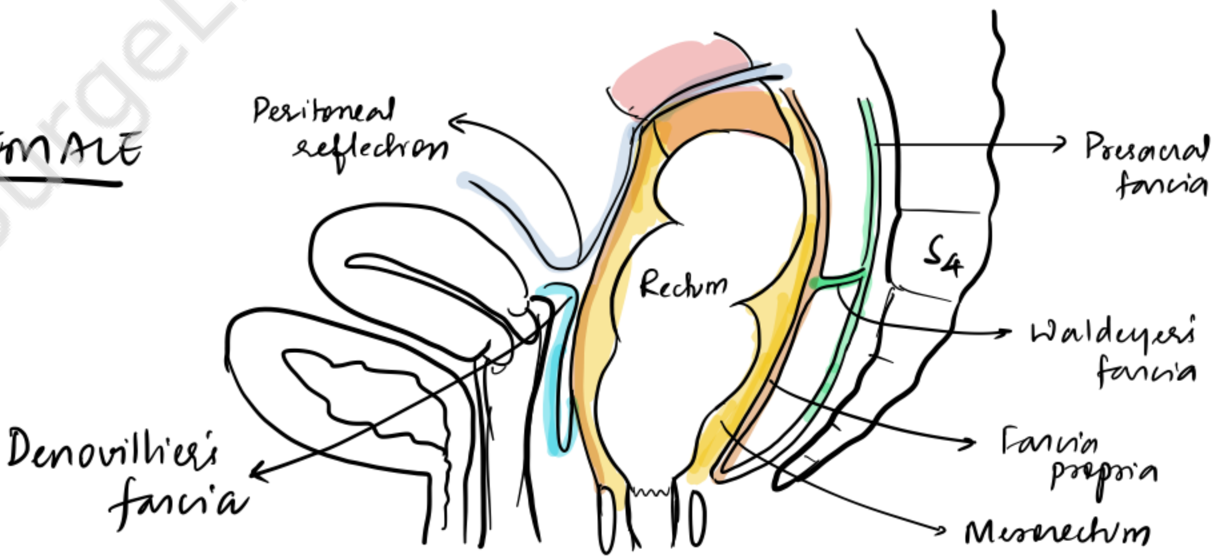
MESORECTUM



MALE



FEMALE



Mesorectum

- thick posteriorly
- thin on the sides
- Very thin anteriorly

A good mesorectal specimen shows:

1) - Cut edge of the peritoneal reflection
2) - Smooth shiny anterior surface of the middle third → Denonvilliers fascia / Rectogenital septum
3) Intact anorectal muscle in lower 1/3rd

GRADES OF TME

3 Complete - intact mesorectal plane, no defect >5mm, no coning

2 Near Complete - irregular mesorectal surface, moderate coning

1 Incomplete - thin mesorectum & defects - exposing muscularis propria

SurgeLight chandanasri@gmail.com

RECTAL PROLAPSE

Clinical entities classified under rectal prolapse

- Mucosal prolapse → only rectal mucosa / Partial
- Internal prolapse (intussusception) – does not protrude through anus
- Full thickness rectal prolapse → True rectal prolapse / Complete (Procidentia)
  ↓
  circumferential, full thickness - protrusion of the rectum through the anus

PATHOPHYSIOLOGY (Maingot)

1) Rectum and rectosigmoid junction have increased mobility off the sacrum
2) Descent of the rectosigmoid junction into the pelvis allows a funnel shaped intussusception into the rectum as the rectum attempts to expel itself
3) Dysfunctional pelvic floor and sphincter mechanism

PREDISPOSING FACTORS

→ Redundant rectosigmoid
→ Long rectal mesentery i poor rectal fixation
→ Deep pouch of Douglas
→ Children – may be a/c cystic fibrosis
  ↳ vertical orientation of the rectum
    mobility of the sigmoid colon
    Relative weakness of pelvic floor muscles
    Redundant rectal mucosa – poorly fixed to submucosa
→ Straining
  BPH
  Constipation
→ Diarrhea - explosive, amoebiasis, schistosomiasis
→ COPD, pertussis
→ Pelvic floor dysfunction
→ Pregnancy
→ Previous surgery

EPIDEMIOLOGY

F > M → 6 : 1
Children < 3 y
Elderly population

surgeonlight.chandanasri@gmail.com

Presentation

→ Mass per anum
- Protruding rectal mucosa
- Thick concentric mucosal rings
- Sulcus between anal canal & rectum
- Solitary rectal ulcer (SRUS) ↓ anal sphincter bone

→ associated pelvic organ prolapse
→ Pain ±
→ Fecal incontinence → anus is stretched and dilated by the protruding rectum - disrupting the sphincter mechanism
→ Irritant mucous secretion by externalised rectal mucosa

DDx - Hemorrhoids
Intussusception
Proctitis

EVALUATION

- Underlying conditions
- Co existent abnormalities in the colorectum
  - Barium enema
  - Colonoscopy

  Rigid proctosigmoidoscopy for SRUS
- Sphincter assessment
  - Anorectal manometry
    - ↓ resting pressure in internal sphincter
    - absence of anorectal inhibitory reflex
  - Colonic transit time - to determine need for colonic resection i/c/o constipation
  - Pudendal nerve terminal latency
  - Electromyography of puborectalis

- DEFECOGRAPHY - if diagnosis is doubtful
  - funnel that descends into the deep pelvis as the rectosigmoid descends
  - ↓
  - Ball valve obstruction at the anal canal
  - ↓
  - pushed onkside
  - Helps distinguish mucosal prolapse from full thickness prolapse

Surgeonsright.chandanasri@gmail.com

DEFECOGRAPHY GRADING SYSTEM

(N) - Rectum remains fixed to sacrum
sphincter relaxes
rectum empties

(R) - Rectocele

1 - Non relaxation of puborectalis
2 - Mild intussusception / mobility from sacrum
3 - Moderate intussusception
4 - Severe intussusception
5 - Prolapse

Defecography - Fluoroscopic defecography

- Oral contrast → to opacify small bowel → to detect enterocele
- Barium enema → to fill rectum
- Vaginal contrast → to visualise vaginal lumen
- Barium pill → taped to perineal body

Films taken with patient sitting on radiolucent commode

Can be coupled i Video defecography

Management

1) Non-operative management

- irreducible prolapse - repositioned ↓CA i hygroscopic antiedema measures
- treatment of constipation & diarrhea
- Biofeedback techniques in pelvic floor dysfunction

Stool softeners
Pelvic exercises - for constipation
Bulking agents - for incontinence

Complications of Surgery
1) Hypogastric nerve injury - incontinence - bladder dysfunction
2) Anastomotic leak / fistula
3) Bleeding from sacral venous plexus
4) Infection
5) Recurrence

SurgeLight chandanasr@gmail.com

SURGICAL MANAGEMENT

PERINEAL APPROACH

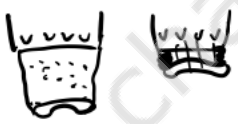
for older people with ↑ surgical morbidity

- ↓ surgical risk, but ↑ recurrence rate

- **ANAL ENCIRCLEMENT**
  - Thiersch wire
  - Non absorbable band is placed subcutaneously around the anus
    ↓
    Keeps the rectum from prolapsing by restricting the size of anal lumen

SUPRALEVATOR HIGH ENCIRCLEMENT can also be done

- **DELORME MUCOSAL SLEEVE RESECTION**
  - Circumferential incision made through mucosa of prolapsed rectum near dentate line.
    ↓
    Mucosa stripped upto the apex of prolapse
    ↓
    Denuded muscularis is plicated up
    ↓
    Mucosal edges are sutured together



- **ALTEMEIER PERINEAL RECTOSIGMOIDECTOMY**
  Full thickness circumferential incision in prolapsed rectum 1-2cm from dentate line
  ↓
  Prolapse delivered
  ↓
  Mesentery ligated
  ↓
  Bowel transected & handsewn/stapled to distal anal canal
  ± Plication of levator ani

- **WYATT PROCEDURE** - mesh rectopexy via perineal approach

- **MUCOSAL PROLAPSE** – Hemorrhoidectomy
  Goodsall ligature

ABDOMINAL APPROACH

for younger, healthier pts with ↑ life expectancy

- **ANTERIOR RESECTION** - if a/w constipation + redundant colon

- **MARLEX RECTOPEXY / RIPSTEIN PROCEDURE**
  - entire rectum is mobilized down to coccyx posteriorly, POD anteriorly, lateral ligaments laterally
  - Marlex mesh is fixed to presacral fascia & then partially (excluding anterior wall of rectum) wrapped around the rectum. Peritoneal reflections are sutured over the foreign body

- **SUTURE RECTOPEXY / GOUGHER RECTOPEXY**
  Rectum is fixed to presacral fascia with non-absorbable suture material

Fascia lata sling may also be used for rectopexy

- **RESECTION RECTOPEXY** – FRYKMAN GOLDBERG
  - Anterior resection + suture rectopexy

- **MOSCHOWITZ REPAIR**
  - for rectocele
  - Reduction of perineal hernia & closure of Cul-de-sac

- **VENTRAL RECTOPEXY**
  - dissection of anterior rectum down to pelvic floor
  - one end of mesh - anterior rectum
  - other end - anchored to sacral promontory

- **LAHAUT OPERATION** - a segment of mobilized rectosigmoid is fixed to the posterior rectus sheath

ABDOMINAL PROCEDURES MAY BE DONE BY
- Open surgery
- Laparoscopy
- Robotics

SurgeLight chardanash@gmail.com

STARR - for internal prolapse (intussusception) / Rectocele
↘ Stapled Transanal Rectal resection

CONSTIPATION Rx

- IBS-C
  - Bulk
  - Biofeedback
- Colonic transit type
  - Subtotal colectomy
  - Antegrade enema
  - Sacral nerve stimulation
  - Stoma
- ODS type
  - STARR
  - Botox
  - Rectopexy
  - Stoma

CONTINENCE MECHANISMS

Rectal compliance & capacitance (200-250ml)
Internal sphincter integrity
External sphincter & pelvic floor integrity
Rectoanal inhibitory reflex / Sampling reflex

Fecal incontinence - inability to withhold the passage of gas / liquid / stool → involuntary passage → when it is socially unacceptable

Rx fecal incontinence

Non Surgical
- Biofeedback

Surgical
- Bulking / Remodelling of Sphincter complex (Secca)
  - ↓
  - RFA to sphincters
  - ↓
  - Aseptic inflammation
  - ↓
  - Collagen deposition
  - ↓
  - fibrosis
  - ↓
  - tightening
- Injectable bulking agents
  - ↓
  - Hyaluronic acid
  - ↓
  - ↑ resting anal sphincter pressure by mass effect
- Sphincteroplasty
- Artificial bowel Sphincter
- Sacral Nerve Stimulation
- Stoma

SurgeLight chandanasri@gmail.com

SOLITARY RECTAL ULCER SYNDROME

Consequence of Pelvic floor outlet obstruction

Attempts to defecate against a CLOSED PELVIC FLOOR

↓

Rectum forms a 'funnel'

↓

Descent of the anterior rectal wall into the anal canal

↓

Ischemia & trauma to a portion of the anterior rectal wall (~10cm proximal to anal verge) which prolapses partially into anal canal during prolonged straining

↓

SOLITARY RECTAL ULCER

↓

Healing - incorporation of functioning mucosal glands beneath the new mucosal surface

↓

COLITIS CYSTICA PROFUNDA

↓

mucus production

collagen deposition

Presentation: h/o constipation
straining
digital maneuvers to evacuate rectum

- Bleeding
- Mucosal prolapse
- Hemorrhoids

'Anismus', 'Proctalgia fugax', 'Levator ani so'

DEFECOGRAPHY — Non relaxing puborectalis | ↑ Colorectal transit time

Rx - ↑ Fibre diet
Normal Bowel training - Biofeedback

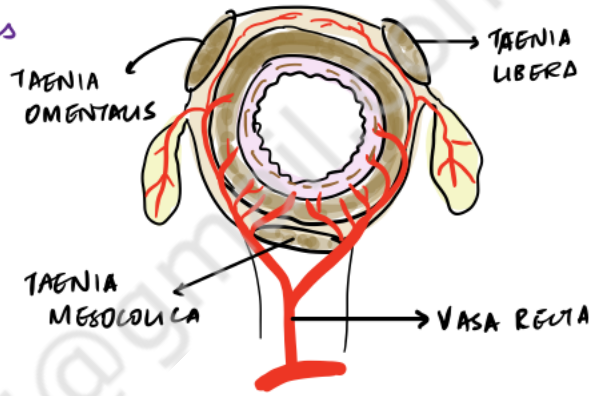
SurgeLight chandanasri@gmail.com

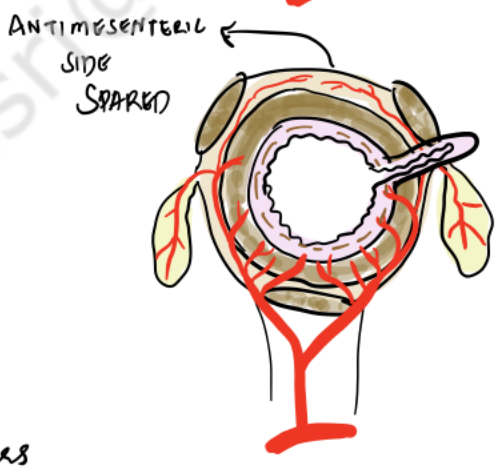
DIVERTICULAR DISEASE

Diverticula are abnormal outpouchings/sacs of colonic wall due to:
- ↑ intraluminal pressure
- disordered motility
- alterations in colonic structure
- ↓ dietary fibre

protrusion of mucosa & submucosa through muscle
- 'FALSE DIVERTICULA'

Formed on the mesenteric side of taeniae coli in the areas where colonic wall is pierced by vasa recta
- areas of relative weakness





RISK FACTORS
- older pts - hyperelastosis, altered collagen
- Immunocompromise
- Uremia
- PCKD
- Smoking, NSAID

m/c involves - SIGMOID COLON
DESCENDING COLON

Rectum - NOT INVOLVED

COMPLICATIONS
1) Infection - Abscess → m/c complication
2) Perforation
3) Bleeding
4) Fistula → colovesical, coloenteric, colocutaneous, colovaginal
5) Obstruction. 2/t chronic inflammation - rare
   - extrinsic compression by abscess

? Risk of cancer - uncertain

PRESENTATION
Asymptomatic until acute event
Incidental finding

ACUTE DIVERTICULITIS - LIQ pain, tenderness, Fever, tachycardia

HINCHEY CLASSIFICATION OF DIVERTICULITIS
Stage
- I
  - Ia - Pericolic inflammation
  - Ib + Pericolic abscess
- II
  - IIa - Diverticulitis + Retroperitoneal/Pelvic abscess
  - IIb - Diverticulitis + abscess (RP/Pelvic) + fistula
- III — Diverticulitis + Purulent Peritonitis
- IV — Diverticulitis + Fecal peritonitis

Sunaelight chandanasri@gmail.com

EVALUATION

- Indolent presentation - as chronic LLQ pain, bowel disturbances bleed PR

  → to ca colon

  CT, Colonoscopy, Barium enema

  ↓

  Diverticula - Saw tooth appearance

- Acute

  Blood inv - Leucocytosis

  Imaging - mucosal thickening
  edema
  irregularity
  contrast extravasation – fistula
  Abscess / collection ↘ perforation

  Fistula - MRI
  Cystoscopy
  Vaginogram

MANAGEMENT

A. ACUTE UNCOMPLICATED DIVERTICULITIS

Conservative - IV antibiotics

Ciplox + Metro

→ sufficient in 95%

B. ACUTE COMPLICATED DIVERTICULITIS

Abscess / Perforation / Severe Bleed → Surgery to control peritoneal sepsis → Lavage + Drain

Laparoscopic

(Uncontained)

Contained abscess → can be managed percutaneously

↓

Resection of affected segment (inflammed bowel)

Primary anastomosis

avoid if unstable

End colostomy + Hartmann

flb elective Total sigmoid colectomy

+ Resection of diverticular colon

↓ Recurrence ← COLORECTAL anastomosis

surgicallight.chandanasri@gmail.com

C. BLEEDING: Severe bleeding - rare

CT angio → Formal Mesenteric angio → Radiolabelled RBC / Tc 99m Sulfur colloid
Colonoscopy

Most cases resolve spontaneously ∴ conservative R
Rarely: angioembolisation → Sm

D. ELECTIVE SURGERY

FISTULAS — Resection + Colectomy
+ Anastomosis

- Medically fit patients
- Several proven attacks of acute diverticulitis
- Young pts (violent course)
- Immunocompromise

SurgeLight chandanasri@gmail.com

AMEBIASIS

Causative organism - Entameba histolytica
Entameba dispar

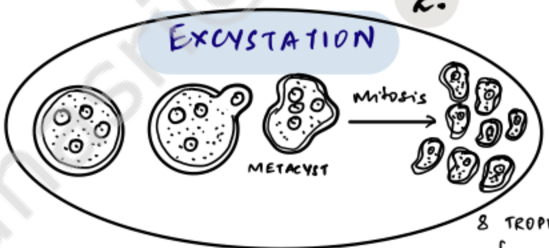
LIFE CYCLE

1.

MATURE QUADRINUCLEATE CYST
INFECTIVE FORM

passed in the feces of carriers → Feco-oral route → INGESTION → Cysts are resistant to acidic pH of stomach → SMALL INTESTINE alkaline pH

2. EXCYSTATION



8 TROPHOZOITES from one quadrinucleate cyst

3.

TROPHOZOITE / METACYST (VEGETATIVE FORM)
Actively motile

TROPHOZOITES pass into Large Intestine

Trophozoite invades the PORTAL CIRCULATION → Trapped in interlobular veins → Multiply in portal triads → LIVER

LIVER (m/c site of Extraintestinal amebiasis)

AMEBIC LIVER ABSCESS
(m/c Ⓡ lobe) - 80%
∵ Blood from SMV runs a more direct course in PV to the larger Ⓡ lobe

- ACROSS DIAPHRAGM → LUNGS, Pericardium
- SYSTEMIC CIRCULATION → LUNGS, BRAIN

4. ENCYSTATION

Binary fission → Cysts

Cysts → STOOL → INFECTIVE FORM
Can survive days to weeks in external environment

TROPHOZOITES → STOOL → NOT INFECTIVE
- Rapidly destroyed when passed out
- Even if ingested, destroyed by gastric acid

- Penetrate the Crypts of Lieberkuhn in colonic mucosa aided by tissue lytic substances - Proteolytic enzymes
AMEBIC CYSTIENE PROTEASE

↓

Penetrate submucosa → lytic necrosis

↓

"Abscess"

↓ Breaks down

Amebic ulcer → Flask shaped ulcers - Submucosal
- m/c in caecum
- 2nd m/c rectosigmoid

- Resolution
- Hemorrhage
- Perforation
- Peritonitis
- Pericecal/ Pericolic abscess
- Chronicity
  - Strictures
  - Granuloma / AMEBOMA

SurgeLight chandan...@gmail.com

CLINICAL FEATURES

INTESTINAL

- **AMEBIC COLITIS**
  - Gradual onset - symptoms present over 1-2 weeks
  - Crampy abdominal pain
  - Watery / Bloody diarrhea
  - Fever in 10-30% cases

(TENDERNESS IN LIF : Sir Philip Manson Bahr point)

- **CHRONIC AMEBIC COLITIS**
  - presentation is similar to IBD
  - Recurrent episodes of bloody diarrhea & vague abdominal discomfort

- **FULMINANT AMEBIC COLITIS**
  - Rare complication of amebic dysentery
  - Rapid onset of severe bloody diarrhea
    Severe abdominal pain
    Peritonitis
    Fever
  - Predisposing factors:
    Poor nutrition
    Pregnancy
    Corticosteroid use
    Very young age (<2y)
  - Perforation common
  - Toxic MEGACOLON may occur
  - Mortality >40%

- **AMEBOMA**
  - less common form of intestinal disease
  - formation of ANNULAR COLONIC GRANULOMATOUS REACTION to the organisms
  - usually presents as a palpable mass in RLQ
  - can mimic Ca Caecum

- **Amebic Appendicitis**
  - Very rare
  - in high prevalence areas

EXTRAINTESTINAL

- **AMEBIC LIVER ABSCESS**
  - Young adult males ↑ h/o living in or travelling to endemic area
  - ↑ / ↓ wt ± h/o antecedent amebic dysentery
  - Fever, night sweats, cough, abdominal pain
  - Tender hepatomegaly
    Bulging intercostal spaces
    Jaundice ±
  - Rupture - peritonitis

Abscesses are most common high in the diaphragmatic surface of the (R) lobe

Abscess contains chocolate coloured odorless ANCHOVY SAUCE-like fluid
↓
mixture of necrotic hepatic tissue & blood

Abscess is bacteriologically sterile
Abscess wall may / may not contain trophozoites

- **PULMONARY AMEBIASIS**
  - D/t transdiaphragmatic spread
  - Hepatopulmonary fistula - Anchovy sauce sputum

- **CEREBRAL AMEBIASIS** - abrupt onset & rapid progression death
  - D/t systemic spread

- **Cutaneous amebiasis**

Spotlight chandanasri@gmail.com

EVALUATION

INTESTINAL

1. STOOL EXAMINATION

- In stage of dysentery / colitis
  - TROPHOZOITES - hematophagous
- occult / frank blood
- Charcot-Leyden crystals
- PMNs

2. SIGMOIDOSCOPY / COLONOSCOPY

- visualisation of 'flask-shaped' or 'collar-stud' undermined ulcers in the rectosigmoid colon / caecum
- Biopsy / Scrapings may show trophozoites
- should be differentiated from inflammatory bowel disease

LIVER ABSCESS

1) CXR - Elevated (R) hemidiaphragm
   Subdiaphragmatic air-fluid levels
   Pleural effusion
   Consolidating infiltrates
2) USG - hypoechoic collection
3) CT - more sensitive
   also delineates contiguous organ extension
4) Aspiration
   For microscopy and culture

SEROLOGICAL TESTS FOR AMEBIASIS

1) Indirect Hemagglutination
2) Latex agglutination
3) Immunoelectrophoresis
4) Counter-immuno electrophoresis
5) Amebic gel diffusion test
6) Complement fixation
7) Indirect Immunofluorescence assay
8) ELISA → m/c

Serum antibodies appear 7-10 d after infection & may persist for years

Surgelight.chandanasri@gmail.com

MANAGEMENT

INTESTINAL

Luminal amoebicides

PAROMOMYCIN
DILOXANIDE FUROATE - 500mg TID x 10days
IODOQUINOL

Systemic amoebicides

Metronidazole, Tinidazole } Niboimidazoles
Dehydroemetine ⟶ esp in fulminant colitis

SURGERY is reserved for:

- Toxic Megacolon
- Severe hemorrhage
- Perforation

→ Indications for emergency surgery
↓
RESECTION & EXTERIORIZATION

- Amoeboma which does not regress after medical therapy must be treated i colonic resection in order to rule out colonic malignancy

LIVER ABSCESS

Uncomplicated Liver abscess
↓
Treat Medically

Both Amebic Liver abscess + colitis
↓
SYSTEMIC AMEBICIDES
Niboimidazoles
- Metronidazole - 750mg TID x 1 month
or IV x 5-10 days
↓
Followed by 10 days of luminal amebicide
DILOXANIDE FUROATE

Radiological resolution can take 3-9m

INDICATIONS FOR PERCUTANEOUS DRAINAGE

1. Deterioration in clinical condition despite adequate treatment
2. Bacterial superinfection
3. High risk of rupture
   Old age
   Size >5cm

INDICATIONS FOR SURGICAL DRAINAGE

1. Ruptured abscess
2. Impending rupture
3. Inadequate catheter drainage

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PARASTOMAL HERNIAS

- Incisional hernia at the site of an intestinal stoma

RISK FACTORS FOR DEVELOPMENT OF PARASTOMAL HERNIA

A. Specific

- Colostomy > Ileostomy > Ureterostomy
- Stomas brought out via primary incision, other incisions in vicinity
- Stomas brought out lateral to lateral border of rectus abdominis > better ← Transrectus stoma
- Large fascial defect created during surgery
- Emergency surgery, intestinal edema
- Devascularisation / Denervation of anterior abdominal wall

B. Post op factors

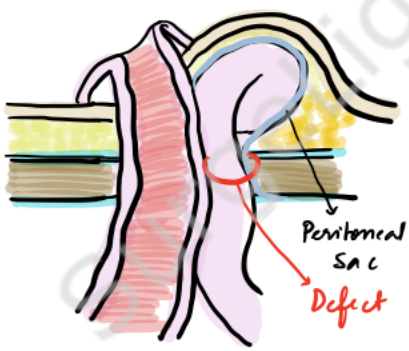
- Parastomal infections
- RT
- Ascites / distension

C. General

- Obesity
- Advanced age
- Malignancy
- Steroid use
- Debility
- COPD

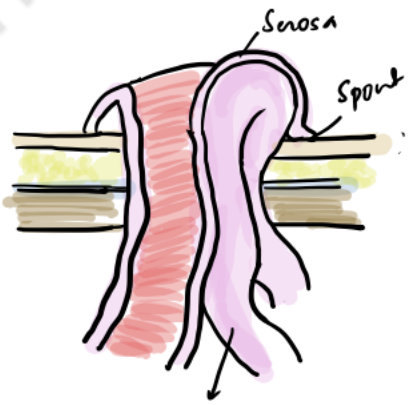
TYPES

TRUE PARASTOMAL HERNIA



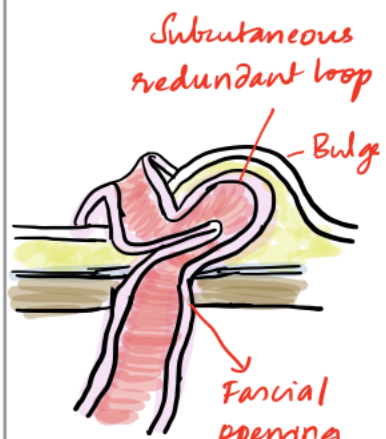
90% - m/c

INTRASTOMAL HERNIA



Bowel loop herniates into the space between the serosal surface of stoma & spout

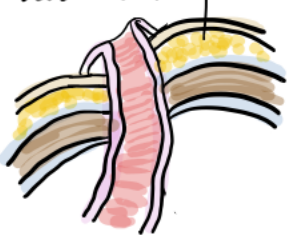
SUBCUTANEOUS PROLAPSE



PSEUDOHERNIA

No hernia

Diffuse bulge 2/t weak abd. wall



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Presentation

- Bulge around the stoma - cough impulse, widened fascia
- Pain
- Pouching difficulties
- Complications - Incarceration, Strangulation, Obstruction, Fistulization, Perforation, Ischemia of stoma → Indications for surgery ↓ esp in a permanent stoma

Evaluation

CT may be done if there are diagnostic difficulties

MANAGEMENT

Non operative management if asymptomatic/mildly symptomatic

SURGERY

APPROACHES

Simple local Repair

- Peristomal incision ('L' shaped) ↓ Reduce the sac ↓ Clear the anterior fascia ↓ Narrow the fascial opening around stoma
- Laparotomy incision ↓ Extraperitoneal flap ↓ Narrow the opening of stoma after reducing hernia

Stoma relocation

Laparotomy ↓ Adhesiolysis ↓ Fashion a new stoma elsewhere ↓ Repair original stoma site ± / + mesh

Prosthetic Repair

EXTRAFASCIAL

Peristomal incision ↓ Dissect & identify hernial sac ↓ Reduce hernia ↓ Reinforce fascial defect & extrafascial mesh

INTRAPERITONEAL (can be done laparoscopically)

1) SUGARBAKER: Laparotomy ↓ Hernia reduced from within peritoneal cavity. Defect re-inforced using INTRAPERITONEAL MESH & a tunnel for bowel
2) KEYHOLE METHOD

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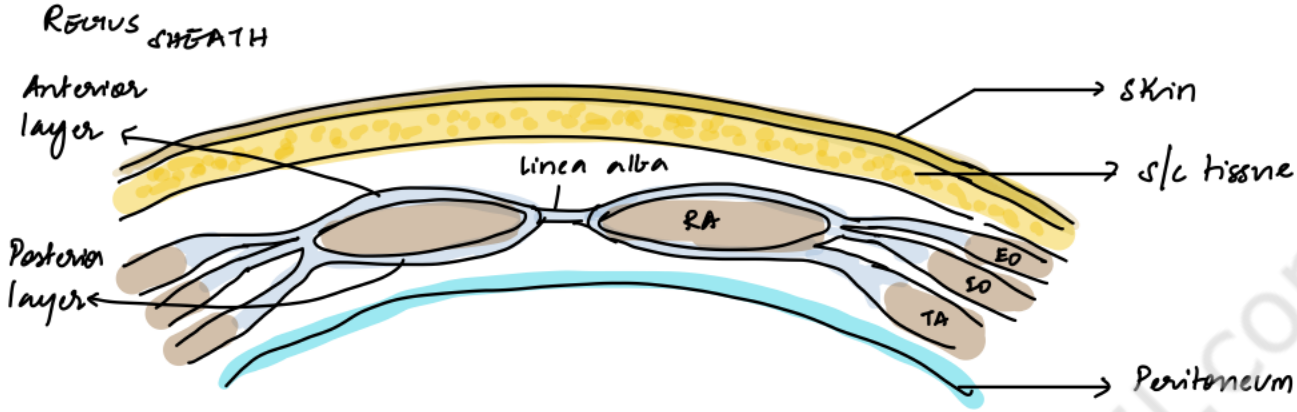
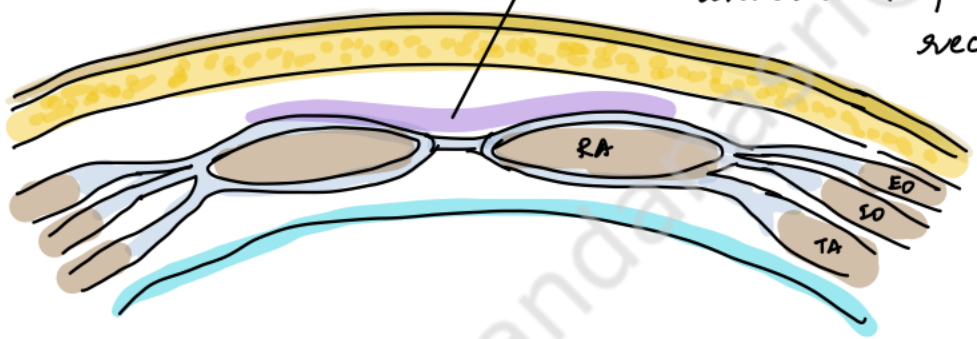
PLANES OF MESH PLACEMENT IN VENTRAL HERNIA REPAIR



1) ONLAY - also called OVERLAY

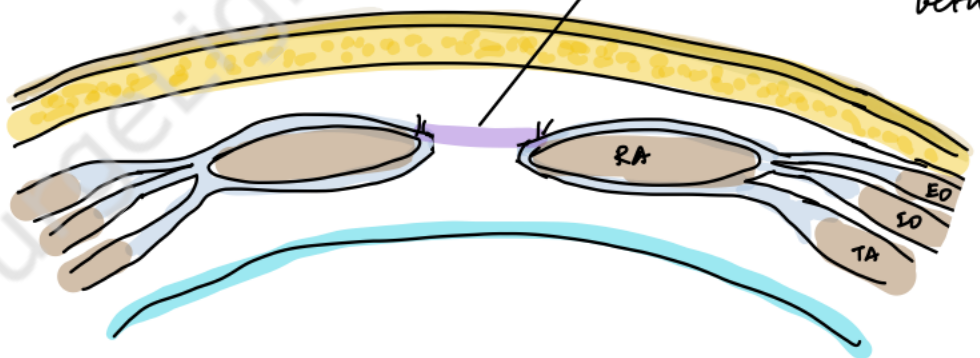
Safe & easy



in subcutaneous plane

Seroma (+)
SSI (+)
↑ Recurrence

2) INLAY



[No attempt is made to primarily repair the defect before placing mesh]

↑ Recurrence
Seroma
SSI

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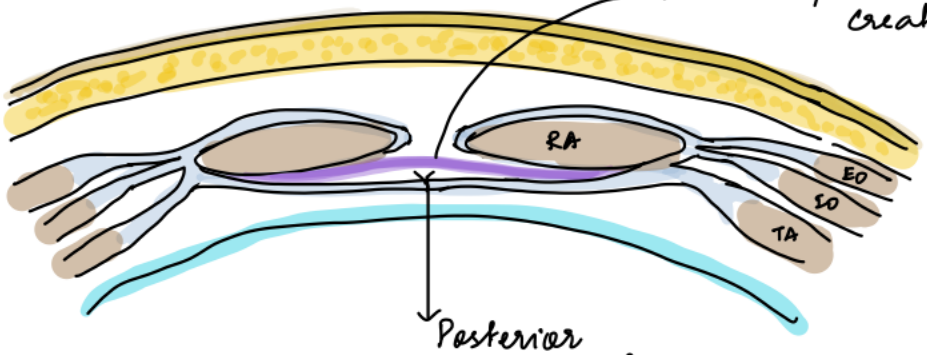
3) SUBLAY → most confusing term d/t book-to-book discrepancy!

1) RETRORECTUS / RETROMUSCULAR SUBLAY



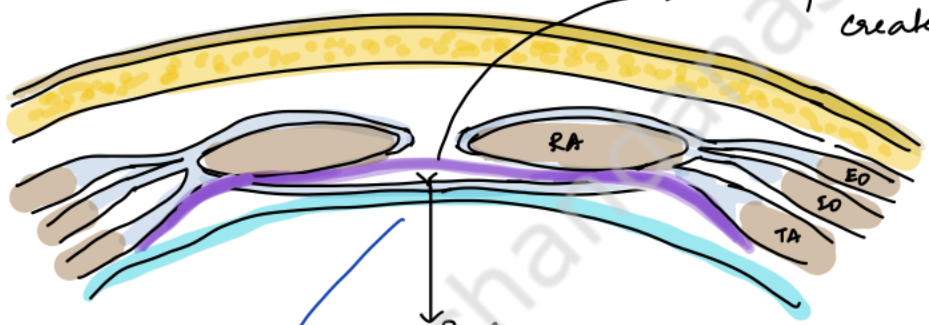
→ Mesh placed in the plane created between rectus muscle and posterior layer of rectus sheath

Posterior sheath reapproximated in midline

↓
↓ Seroma
↓ SSI
↓ Recurrence

- Chronic pain d/t ↑ dissection

2) PREPERITONEAL SUBLAY
- also (confusingly) called EXTRAPERITONEAL OVERLAY!



→ Mesh placed in the plane created between rectus muscle and posterior layer of rectus sheath

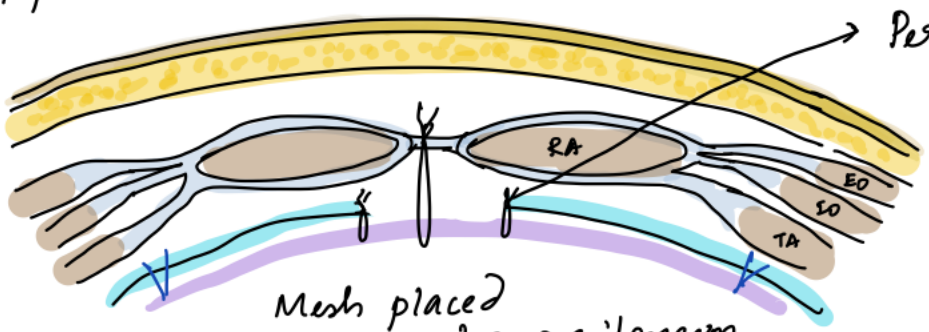
↓ plane extends laterally beyond the lateral border of rectus muscle

Posterior sheath and peritoneum reapproximated in midline

Below arcuate line, posterior layer is deficient, so mesh lies in PREPERITONEAL PLANE → Preperitoneal repair

---

4) UNDERLAY / INTRAPERITONGAL UNDERLAY



→ Peritoneal edge of hernia sac

Mesh placed under peritoneum

↑ IAP
↓
Positively reinforces abd wall
↳ Best ventral repair

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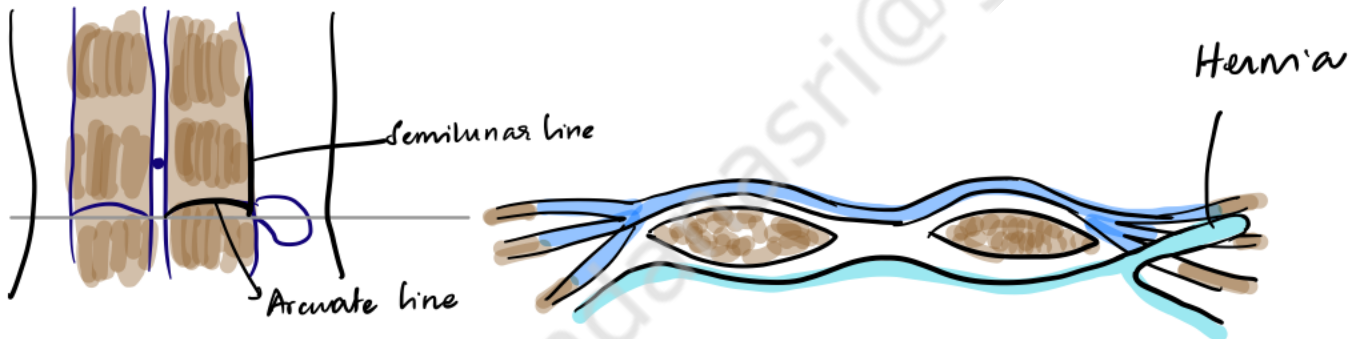
UNUSUAL HERNIAS

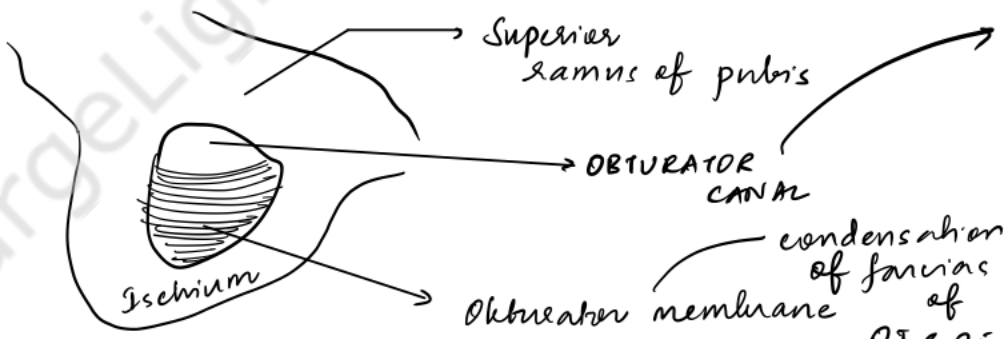
SPIGELIAN HERNIA

- occurs through the Spigelian fascia - between rectus muscle & semilunar line
  at or below arcuate line
- inherent weakness 2/t absence of posterior rectus sheath
- often interparietal - hernia sac dissects posterior to EOA
- small - bulge may not be obvious
- 4 - 7th decade
- Narrow neck - risk of incarceration



Repair - transverse incision over sac - dissect, reduce, excise/
invaginate
repair - shaping
prosthesis

OBTURATOR HERNIA



Obturator region - medial portion of upper thigh

external opening - under the pectineus - medial to femoral vein

℞ - Reduction & repair via POSTERIOR APPROACH

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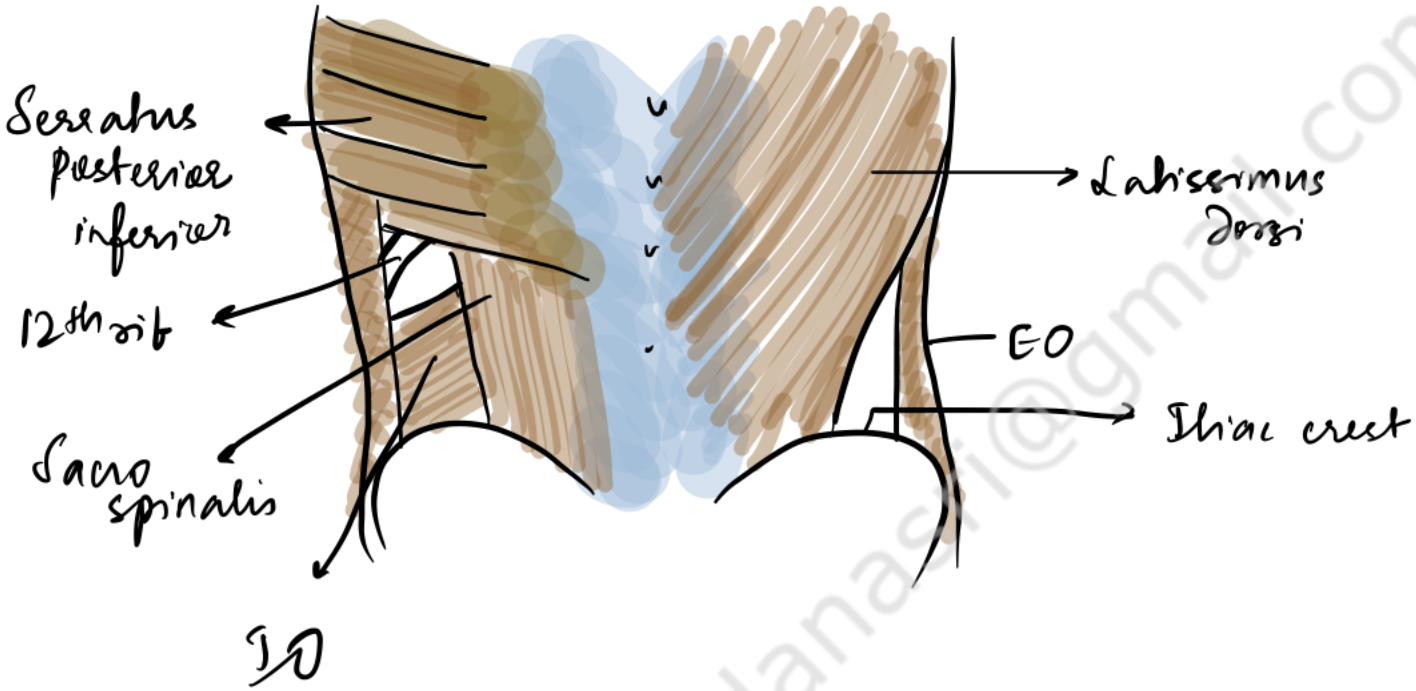
LUMBAR HERNIA

- Superior Lumbar triangle — GRYNFELT – MEC
- Inferior Lumbar triangle — PETIT



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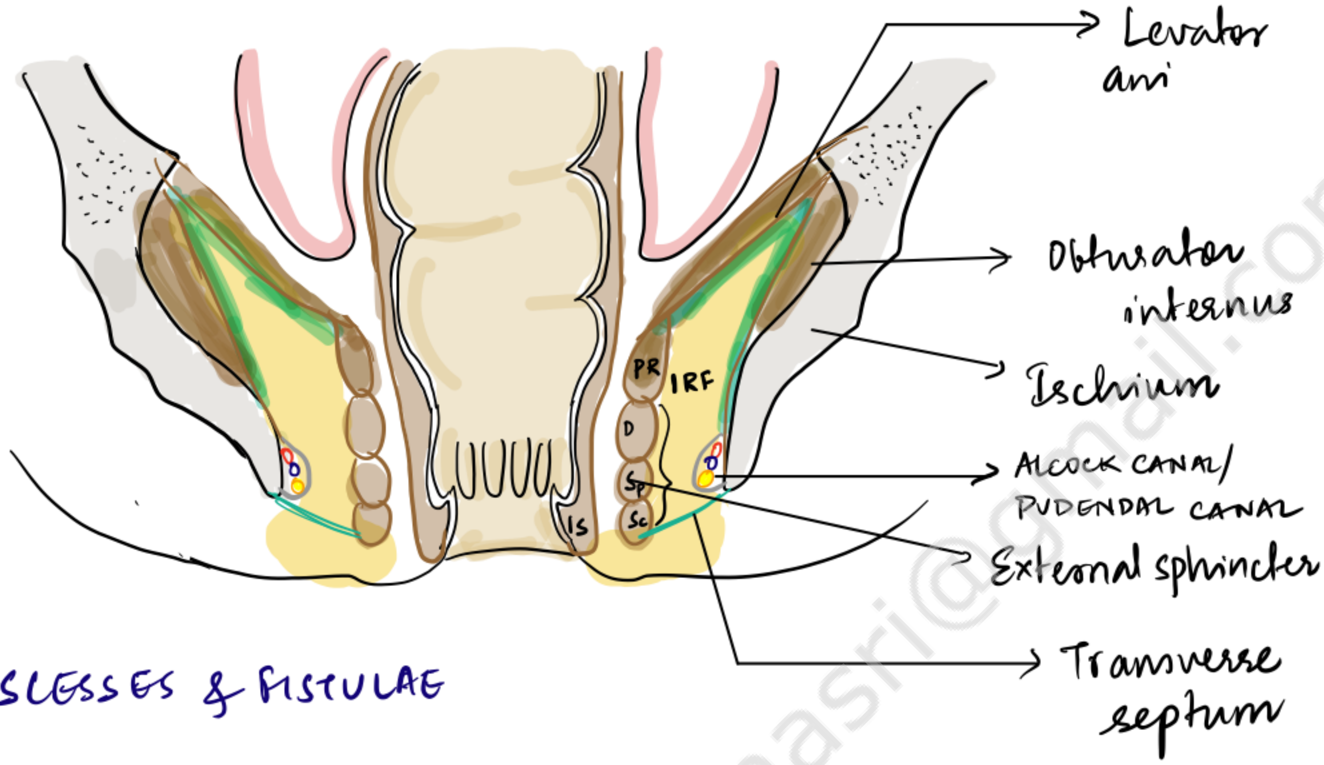
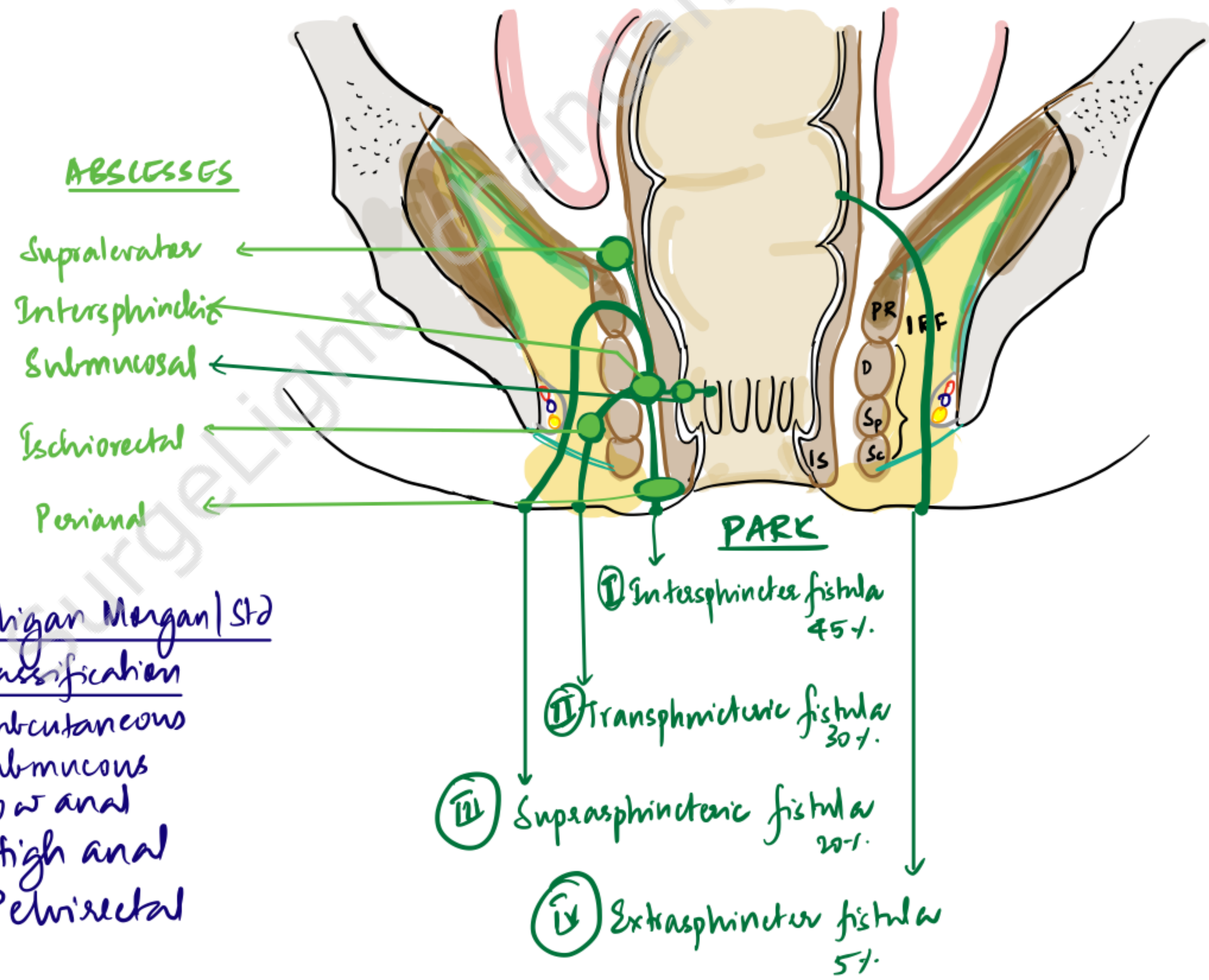
ANORECTAL SEPSIS

SURGICAL ANATOMY



ABSCESSES & FISTULAE



Milligan Morgan | Std Classification
- Subcutaneous
- Submucous
- Low anal
- High anal
- Pelvirectal

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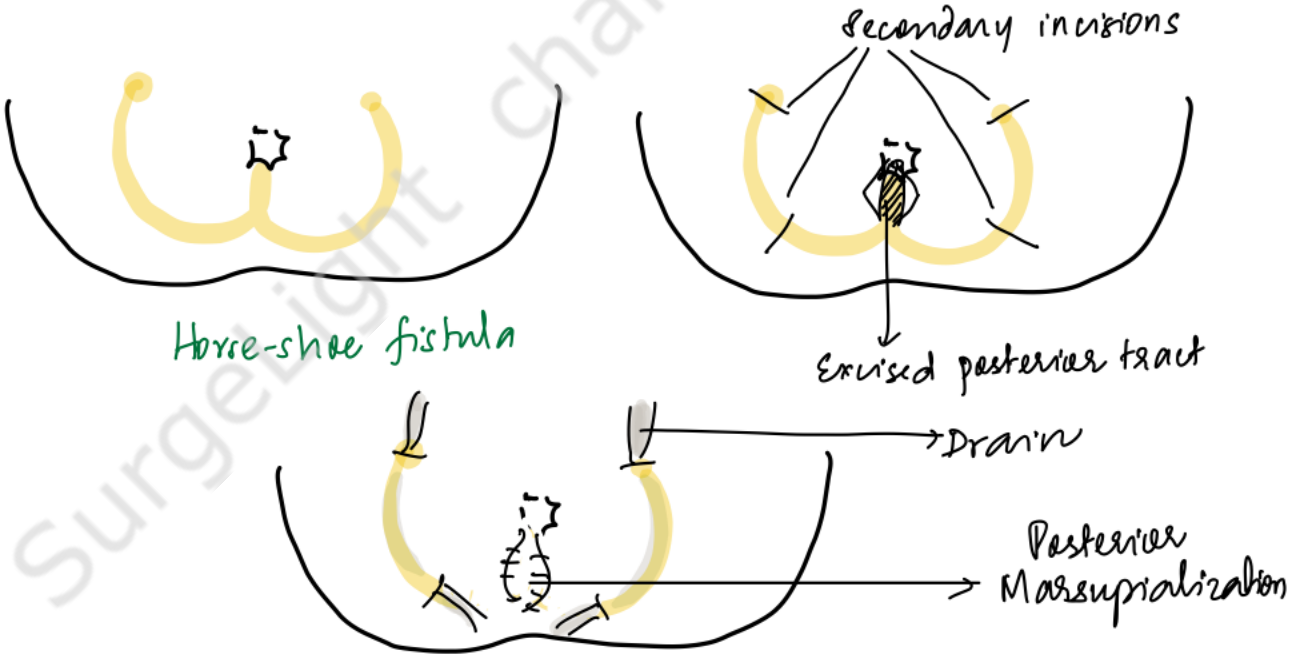
Procedures for Fistula

1) Fistulotomy
2) Fistulectomy
3) Horseshoe fistula - Hanley procedure
4) LIFT - Ligation of Interesphincteric Fistula tract
5) VAAFT - Video Assisted Anal Fistula Tract Ligation
6) Gluing
7) Endorectal advancement flap - for high anal fistulas
8) Setons < Cutting / Draining
9) Anal Fistula Plug repair
10) Colostomy

HANLEY PROCEDURE



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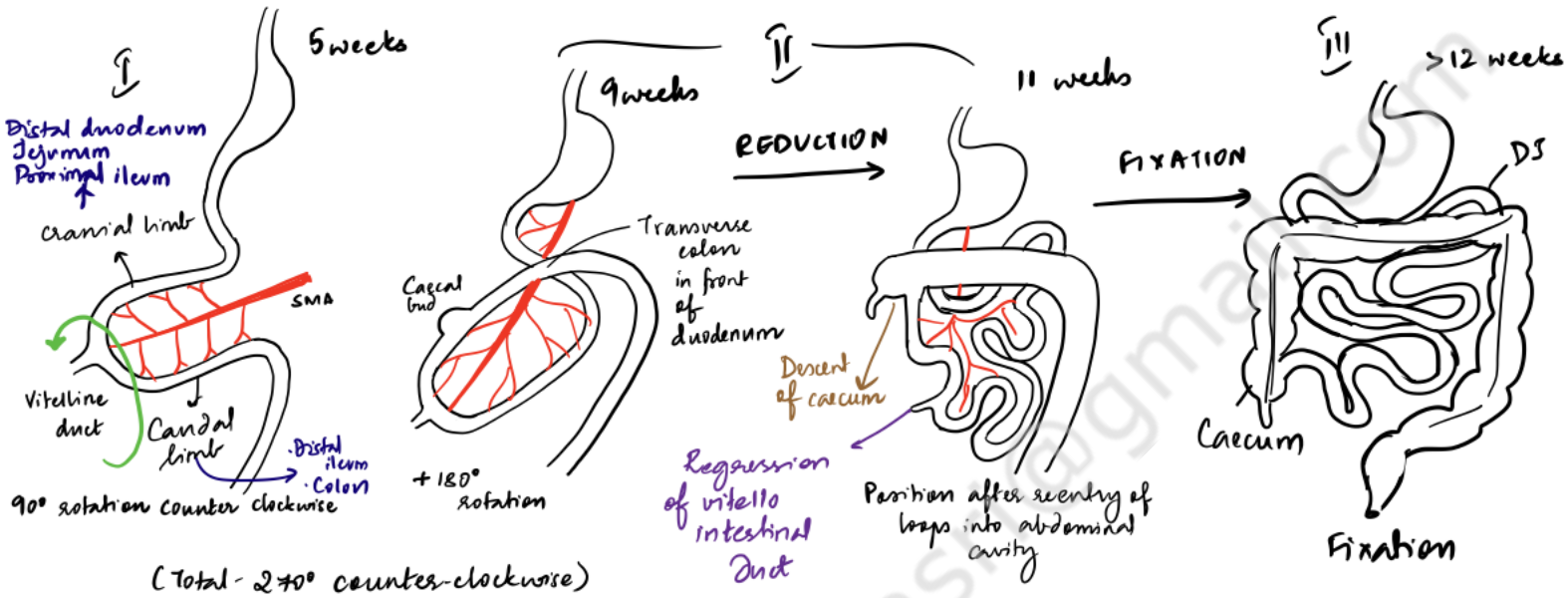
MALROTATION OF GUT

Definition: a group of abnormalities resulting from abnormal rotation and fixation of the intestine

EMBRYOLOGICAL BASIS



(Total - 270° counter-clockwise)

- The endoderm of the yolk sac forms the primitive gut - 2/t FOLDING OF THE EMBRYO cranially and caudally - 4th week of gestation

  Endoderm → epithelial lining of intestines
  Splanchnic mesoderm → other layers, mesentery
  Neuro-ectoderm → Nerves

- Physiological herniation of midgut through the umbilicus - 5th week
- Endodermal proliferation and temporary occlusion of lumen - 5th week
- Growth & expansion of mesoderm, apoptosis of ectoderm → recanalisation - 7th week
- Intestine returns to abdominal cavity after rotation → ~10th week - 12th week

Stage I: 4-10 weeks - Physiological herniation of midgut

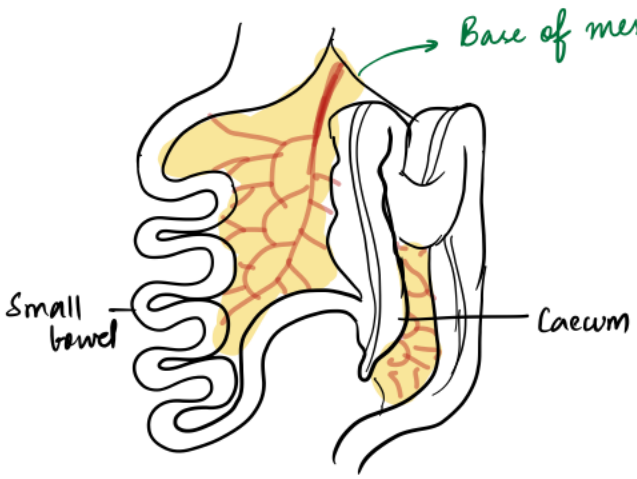
Stage II - 10-12 weeks (Reduction) - Migrates back into abdomen - small bowel first
Caecocolic loop last
Caecum initially lies to left → 270° CCR → Right

Stage III - Fixation - of duodenojejunal loop to (L), caecum to RLQ

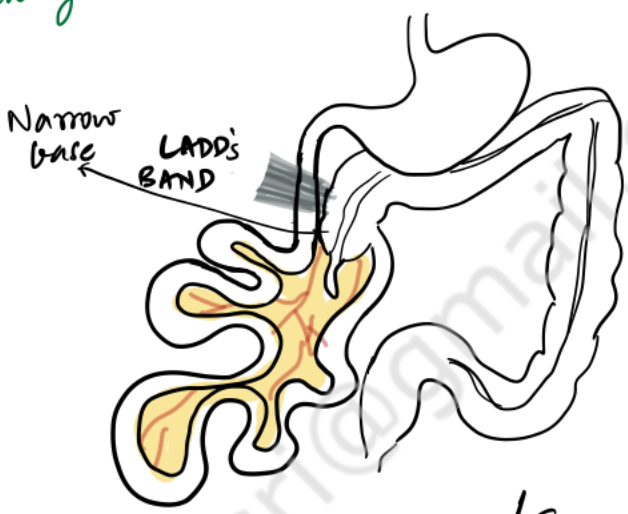
SurgeLight chandanasagar@gmail.com

DISORDERS

1) Non rotation - arrest in stage I → No rotation of both
   - Duodenojejunal limb
   - Caecocolic limb
   - Duodenojejunal flexure does not lie to the left of SMA
   - Caecum does not lie in RLQ
   - If base of mesentery is fairly narrow, only then → volvulus



NON-ROTATION



MALROTATION / Incomplete Rotation

2) Incomplete rotation - arrest in stage II

(1) - Duodenal obstruction
↓
D/t Peritoneal bands → LADD's BAND running from caecum to mesentery
↓
compression of 3rd part of duodenum

(2) Midgut volvulus - due to narrow mesenteric base - twists in clockwise direction

(3) Internal hernia - if duodenojejunal loop does not rotate but caecocolic loop rotates and gets fixed -
Most of the small bowel is trapped in the mesentery of large bowel
↓
(R) MESOCOLIC HERNIA / PARADUODENAL HERNIA

3) Incomplete fixation - arrest in stage III

Hernial pouches form when mesentery of the (R) & (L) colon and the duodenum do not become fixed retroperitoneally

(L) MESOCOLIC HERNIA - migration of small intestine in (L) upper quadrant (→ due to non-fixation of descending mesocolon) between IMA & posterior parietal attachment

SurgeLight - chandanasri@gmail.com

CLINICAL PRESENTATION

1. Acute Midgut volvulus
2. Chronic Midgut volvulus
3. Acute Duodenal obstruction
4. Internal herniation

ACUTE MIDGUT VOLVULUS

- 1st year of life
- Acute onset bilious emesis

X Ray - Dilated bowel ± paucity of distal gas

CHRONIC MIDGUT VOLVULUS

- intermittent / partial twisting
  ↓
  LYMPHATIC / VENOUS OBSTRUCTION
- recurrent abdominal pain
- Malabsorption syndromes
- Altered bowel habits
- GERD

ACUTE DUODENAL OBSTRUCTION

- Ladd's bands
- infancy to preschool age

INTERNAL HERNIATION

(R) & (L) Mesocolic hernias
Intermittent abdominal pain
Hematochezia, Hemorrhoids, dilated abdominal wall veins

Investigations

1) CBC - sepsis. ↑/s/o NEC
   - ↓ Hb - GI bleed
2) ABG - Metabolic acidosis
3) E/E - Hyperkalemia
4) Coagulation profile - abnormal → DIC
5) EXR Abdomen - distended stomach & proximal duodenum
6) UGI contrast series (in stable pts)
   (N) - DJF (L) of midline, above pylorus
   Typical - DJF (R) of midline / absent
   Atypical - DJF in midline / left of midline - below pylorus } Malrotation
   Abrupt tapering of contrast → volvulus
7) Lower GI series - rarely indicated - Caecal localization
8) USG Abd - 100% sensitivity in expert hands - inversion of SMA & SMV
   Fixed midline bowel loops
   Duodenal dilatation ± distal tapering
9) CT - coiling of SMV around SMA → volvulus

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MANAGEMENT

1) Stabilize patient
   - NG tube
   - IV access
   - Correction of fluid & electrolyte deficits
   - BROAD SPECTRUM ANTIBIOTICS
     - Acute → Immediate Surgery
     - Stable → Investigate

2) Surgery

LADD PROCEDURE

1) Reduction of volvulus - detorsion in counter-clockwise manner
   → Place warm towel & observe
   → Assess viability, resect gangrenous segments
2) Division of Ladd bands
   - usually extend across 3rd part of duodenum from ascending colon to the (R) UR posterior aspect
3) Mesenteric base widened by mobilizing caecum
4) Incidental appendicectomy
5) Bowel loops are replaced into the abdomen
   - Small bowel on (R) side
   - Caecum & large bowel on left side

Laparoscopic LADD procedure may also be done

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MECONIUM ILEUS

Neonatal intestinal obstruction resulting from impaction of abnormally thick & tenacious meconium in the distal small bowel (ileum)

Earliest clinical manifestation of CYSTIC FIBROSIS

80-90% Meconium Ileus pts → Cystic Fibrosis
The rest - Preterm infants c h/o maternal tocolysis

Meconium - first stool of newborn
formed during intrauterine life
INTESTINAL EPITHELIAL CELLS, BILE, SUCCUS ENTERICUS, MUCUS, LANUGO, AMNIOTIC FLUID
olive green
evacuated within 24-48 hrs

TYPES

SIMPLE

Thickened sticky meconium obstructs terminal ileum

- Proximal bowel dilatation
  Bowel wall thickening
  Congestion
- Colon - narrow & empty
  'MICROCOLON' - underused colon

COMPLICATED

- Bowel necrosis, perforation, peritonitis
  pseudocyst formation, intraperitoneal calcifications, ascites, volvulus, stenosis, atresia

PSEUDOCYST - walling off of the extruded meconium
- cystic mass c rim calcification

CLINICAL FEATURES

Classical triad - Abdominal distension
Bilious vomiting
Failure to pass meconium

INVESTIGATIONS - In utero

- Maternal polyhydramnios
- Dilated bowel loops

1) Plain X-ray - air filled bowel loops
No air - fluid levels
Soap bubble / Neuhauser sign

Complicated - pseudocyst
USG - snowstorm

2) Contrast enema - microcolon
(N) / dilated colon = Meconium plug sd)
- meconium pellets in terminal ileum

Also helps in meconium evacuation

Done after adequate resuscitation / hydration

Surgeonlightchandanachari@gmail.com

MANAGEMENT OF MECONIUM ILEUS

- Resuscitation – IV fluids
  R + A
  IV antibiotics

- Non-operative management
  CONTRAST ENEMA – Gastrografin + N-acetyl cysteine
  - Gastrografin → hyperosmolar → fluid shifts into bowel & helps liquefy the meconium
  - N-acetyl cysteine → Breaks disulphide bonds in meconium

- SURGERY – Indications
  - Persistent / worsening abdominal distension / obstruction
  - Complicated Meconium ileus

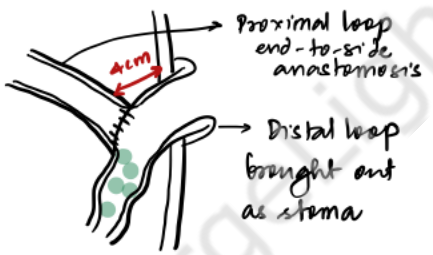
PROCEDURES

1) Enterotomy + decompression
   Enterotomy on antimesenteric border of dilated ileum
   Instillation of dilute NAC / saline → evacuation
   transverse closure of enterotomy
   Appendectomy → HPE for ganglion cells, f/s/o cystic fibrosis
   Post op anal dilatation + rectal irrigation

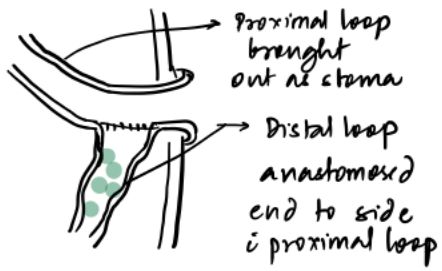
2) Enterostomy i/i out tube + irrigation

3) Resection and enterostomy

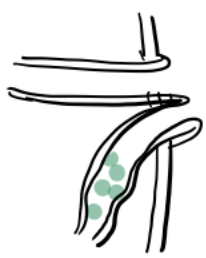
BISHOP KOOP PROCEDURE



SANTULLI & BLANC PROCEDURE



MICKULICZ PROCEDURE



4) Resection & anastomosis – can be done in case favorable cases

Complications

SBS
Stoma related complications

Surgeonlight.chandrasri@gmail.com

OMPHALOCELE AND GASTROSCHISIS
(Neonatal anterior abdominal wall Defects)

Omphalocele

- Central defect of anterior abdominal wall
- Results from failure of return of herniated midgut into abdominal cavity by 11 weeks of gestation
- Defect size >4cm
  (<4cm defect → Hernia of umbilical cord)
- Defect - umbilical ring
- Sac - outer layer - amnion
  inner layer - peritoneum
- ASSOCIATIONS
  - BECKWITH WIEDMANN SYNDROME
    Gigantism
    Macroglossia
    Umbilical Defect - Hernia / Omphalocele
    Visceromegaly i pancreatic islet cell hyperplasia
    Genitourinary abnormalities
    ↑ incidence of Wilms' tumor, liver tumors & adrenocortical neoplasms
- PENTALOGY OF CANTRELL
  1) Omphalocele
  2) Cleft sternum
  3) Anterior (retrosternal) Diaphragmatic hernia of Morgagni
  4) Absent pericardium
  5) Cardiac defects - Ectopia Cordis, VSD

℞ - Resuscitation
Prevention of hypothermia
Preservation of intact sac
DEFECT CLOSURE
- Primary for small/medium defects
- Prosthetic patch closure
- Skin flap closure
- Placement of silo for sequential reduction & staged closure

Promotion of sac epithelialization by applying MEBROMIN & BETADINE

Gastroschisis

- Lateral defect - just to the (RIGHT) of the umbilical cord at the site of the obliterated (R) umbilical vein
- Due to defective ingrowth of mesoderm or impaired midline fusion → weakness of body wall

Defect >4cm, (R) of umbilical cord

NO sac

Direct exposure of herniated intestine to amniotic fluid
↓
Intestine is thickened, edematous & foreshortened

ASSOCIATION: Intestinal Atresia
Undescended testes (10 - 20%)

MANAGEMENT

- Prevention and correction of dehydration
- Place infant in a warm saline bag upto the nipple line
- Antibiotics
- Surgery
  - Primary closure
  - Prosthetic patch closure
  - Biomaterial substitutes
  - Silo placement
- TPN until regainment of bowel function
  - Short bowel Syndrome possible

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HIRSCHSPRUNG'S DISEASE

Developmental disorder characterised by an absence of ganglion cells in the myenteric (Auerbach) and submucosal (Meissner's plexus)

EMBRYOLOGICAL BASIS

Craniocaudal migration of ganglion cells of bowel begins - 12th week

↓

DEFICIENT MIGRATION

(Earlier the migration stops, longer the aganglionic part of distal bowel)

Neurogenic parasympathetic abnormality

↓

Aganglionic bowel is unable to relax and the propulsive wave stops at the proximal end of the aganglion segment

↓

Muscular spasm of distal colon and internal sphincter

↓

FUNCTIONAL OBSTRUCTION

↓

DILATATION OF BOWEL PROXIMAL TO AGANGLIONIC SEGMENT

(Dilated bowel = (N) bowel)

EPIDEMIOLOGY

- 1 in 3000 - 5000 live births
- M : F :: 4 : 1
- Familial - 7-10%
- RET proto-oncogene mutations

A/c Down's Syndrome

CLASSIFICATION

1) Rectosigmoid	75-80%
2) Long Colonic	10%
3) Total colonic	10%
4) Jejuno ileal	<5%

Presentation

1) m/c in neonatal period
2) Abdominal distension, bilious vomiting, failure to pass meconium delay
3) Poor feeding / constipation
4) Enterocolitis → most dreaded complication, m/c/c of death in HD
   Abdominal distension, foul-smelling stools, fever, sepsis

Surgeon Light Chandanasri@gmail.com

EVALUATION

- Suggestive history & findings
- Contrast enema - Megacolon
  Transitional zone showing difference in caliber
  [Suggestive - if rectum is narrower than sigmoid]
- Anorectal manometry
  - Missing rectoanal inhibitory reflex
- Rectal biopsy
  No ganglion cells
  Thick nerve trunks
  IHC : acetylcholinesterase, Calretinin immunostaining } ↑ activity in Lamina propria & mucosa

MANAGEMENT

Stable patients : mild symptoms
Continue oral feeds
Daily emptying of bowel : saline enemas

↓

Plan for Surgery

SURGERY

Emergency - Bowel diversion for
- Fulminant enterocolitis
- long segment / total colonic aganglionosis not fit for immediate surgery

↓

COLOSTOMY

Levelling colostomy to a bowel segment above the transition zone : normal innervation and caliber

↓ (Confirm : frozen section)

can subsequently be pulled through without the need to confirm ganglion status

↓

BRIDGE COLOSTOMY
(Both stoma barrels should be separated by fascial/skin bridge)

SurgeLight chandanasri@gmail.com

DEFINITIVE PROCEDURES

① DUHAMEL PROCEDURE

- Aganglionic rectal stump is left in place
- Ganglionated normal colon is pulled behind the stump.
- Stapler is introduced into the anus ī one limb in aganglionic stump and other in the ganglionated colon

↓ Fixed

Neorectum

empties normally d/t posterior patch of normal bowel

② SWENSEN PROCEDURE (RECTOSIGMOIDECTOMY)

Aganglionic bowel is **REMOVED** down to the level of the internal sphincter

↓

Coloanal anastomosis

③ SOAVE PROCEDURE (MUCOSECTOMY)

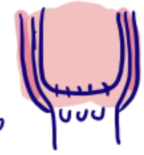
Endorectal mucosal dissection within the aganglionic distal rectum

↓

ganglionated Ⓝ colon pulled down through the remnant muscular cuff

↓

Coloanal anastomosis



- Can be performed by lap
- Can be performed transanally

Popular because colostomy can be avoided

④ DE-LA-TORRE OPERATION (TRANSANAL)

Through circumferential incision above dentate line → mucosal dissection upto the peritoneal reflection

↓

Posterior myotomy in muscular cuff

↓

Pull through of aganglionic bowel out of anus

↓

Resection at transition zone

↓

Ⓝ Colon sutured to the resected site at dentate line

⑤ REHBEIN ANTERIOR RESECTION

↓

After resecting the aganglionic segment, the anastomosis is performed **abdominally** using EEA or handsewn technique

---

SPHINCTEROMYOTOMY - indicated in patients ī internal sphincter achalasia after an operation

HYPOGANGLIONOSIS / INTESTINAL NEURONAL DYSPLASIA → presents similar to HD
- management is controversial

SurgeLight chandanasri@gmail.com

ESOPHAGEAL ATRESIA – TRACHEO-ESOPHAGEAL FISTULA

Definition:

Esophageal atresia is a congenital discontinuity resulting in proximal esophageal obstruction

TEF - Abnormal fistulous communication between esophagus & trachea
- can occur alone / in combination

EMBRYOLOGIC BASIS

• During 4th week of gestation
- foregut begins to differentiate into ventral respiratory part and dorsal esophageal part
↓
ventral respiratory part separates from the esophagus by the formation of lateral tracheoesophageal folds (some say cranial & caudal) which fuse in the midline to form TRACHEO-ESOPHAGEAL SEPTUM
↓
Incomplete fusion / abnormal tracheo-esophageal folds → ABNORMAL COMMUNICATION BETWEEN TRACHEA & ESOPHAGUS

• May develop due to disturbances in epithelial proliferation & apoptosis

EPIDEMIOLOGY

1) 1 in 2500–3000 live births
2) M : F : 1·26 : 1
3) Risk of 2nd child c̄ EA/TEF – 2% → 20% c̄ multiple cases
4) RR in twins 2·26
5) Environmental factors
   - Methimazole in early pregnancy
   - Maternal diabetes
   - Thalidomide exposure
6) Chromosomal anomalies (6-10%)
   - TRISOMY 18
   - TRISOMY 21
7) ASSOCIATIONS

VACTERL -
- Vertebral anomalies – thoracic
- Anorectal anomalies
- Cardiac anomalies
- TE – TEF
- Renal anomalies
- Limb anomalies – Radial dysplasia

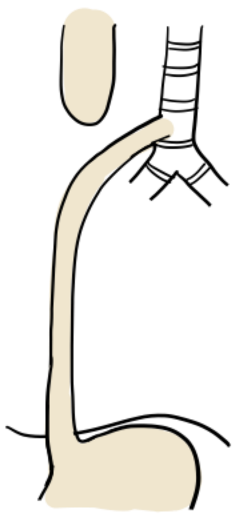
- Trisomy 21
- Duodenal / Lower intestinal atresia
- Tracheomalacia

CHARGE -
Coloboma iris, Heart defects, Atresia of choanae, Retarded development, Genital hypoplasia, EA-TEF

SurgeLight chandanasri@gmail.com

TYPES (A – E) LADD & GROSS
F = Congenital stenosis

VOGT – I – III c < $\overline{II}$ – $\overline{III}$ c = A → D (H fistula not incl)
I → aplasia



EA + Distal TEF

• LADD & GROSS – 'C'

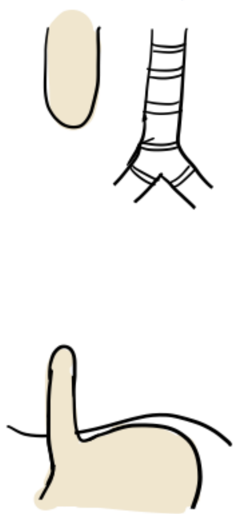
• m/c type ← common

• 85%

• Proximal esophagus
  - dilated
  - thick wall
  - descends into the superior mediastinum upto T_3 or T_4

• Distal esophagus
  - enters trachea posteriorly at Carina or 1-2cm higher

• Distance between esophageal ends is variable



Pure EA

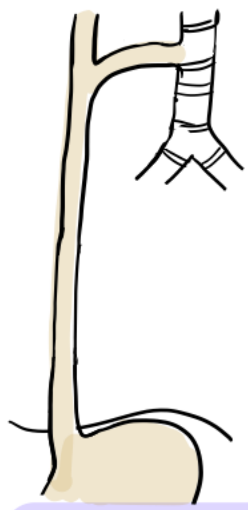
• LADD & GROSS – (A)

~7%

Proximal esophagus
  - ends blindly at the level of azygos vein in posterior mediastinum

• Distal esophagus
  - short stump suspended by fibrous band

• Large Distance between esophageal ends



H – type / No EA

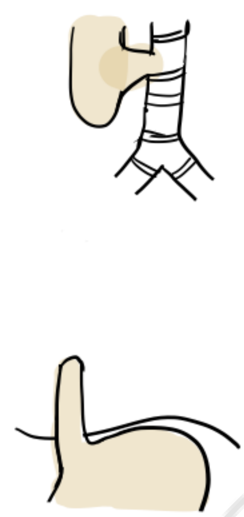
• LADD & GROSS – 'E'

4%

Fistula starts from membranous trachea and runs caudad to esophagus

• Short fistula of variable diameter

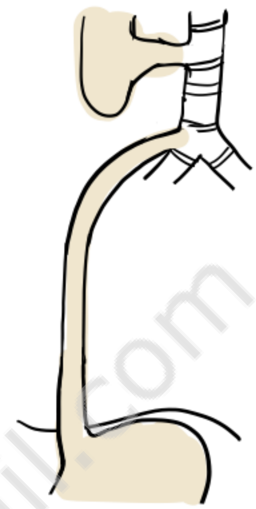
• Situated at thoracic inlet / neck



Proximal Fistula

• LADD & GROSS – 'B'

2%



Proximal + Distal Fistula

• LADD & GROSS – 'D'
D for Double

< 1%

EVALUATION

• Antenatal – Polyhydramnios in 2nd half of pregnancy
  - Fluid shifting in the upper pouch
  - Paucity of fluid in stomach & intestine

• Post-natal – drooling of saliva
  dyspnea
  cyanotic attacks

Air in the bowel below diaphragm
⇒ distal communication
if not – pure atresia / proximal fistula

REPLOGLE tube helps approximate length of upper pouch

Evaluate for other malformations

used of contrast discouraged – risk of aspiration

surgelight.chandanasri@gmail.com

Management

- Immediate oro/naso-esophageal insertion of Replogle tube
  → continuous/ intermittent aspiration of saliva to prevent aspiration
- Nurse in propped up position
- Intubation and ventilation i/c/o severe respiratory distress
  severe pneumonia
  severe associated malformations

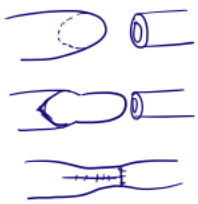
ET-tube must go beyond fistulous opening

SURGERY - After proper resuscitation

Open extrapleural approach is prefered ; Thoracoscopic repair may also be done

(R) Dorso-lateral thoracotomy (i/c/o (N) aortic arch ; (L) thoracotomy for (R) arch)
division of azygous vein
Proximal & distal ends dissected & mobilized
Fistulous tract divided and defect repaired

Attempt Esophago-esophagostomy
↓
Lengthening procedures- circular/spiral myotomy
mucosal-muscular flap



in case of long gap

- proximal pouch may be tagged to prevertebral fascia
- Cervical esophagostomy + gastrostomy
- gastric transposition
- Colonic interposition

Waterston Prognostic Classification

	Birth weight			Survival
I	>1.5 kg		No Cardiac malformation	97%
II	<1.5 kg	(or)	Cardiac malformation	60%
III	<1.5kg	+	Cardiac malformation	22%

Complications

- Aspiration - Pulmonary distress
- Anastomotic leak - mediastinitis

} early

Esophagotracheal fistula
Anastomotic strictures
Tracheomalacia

} Late

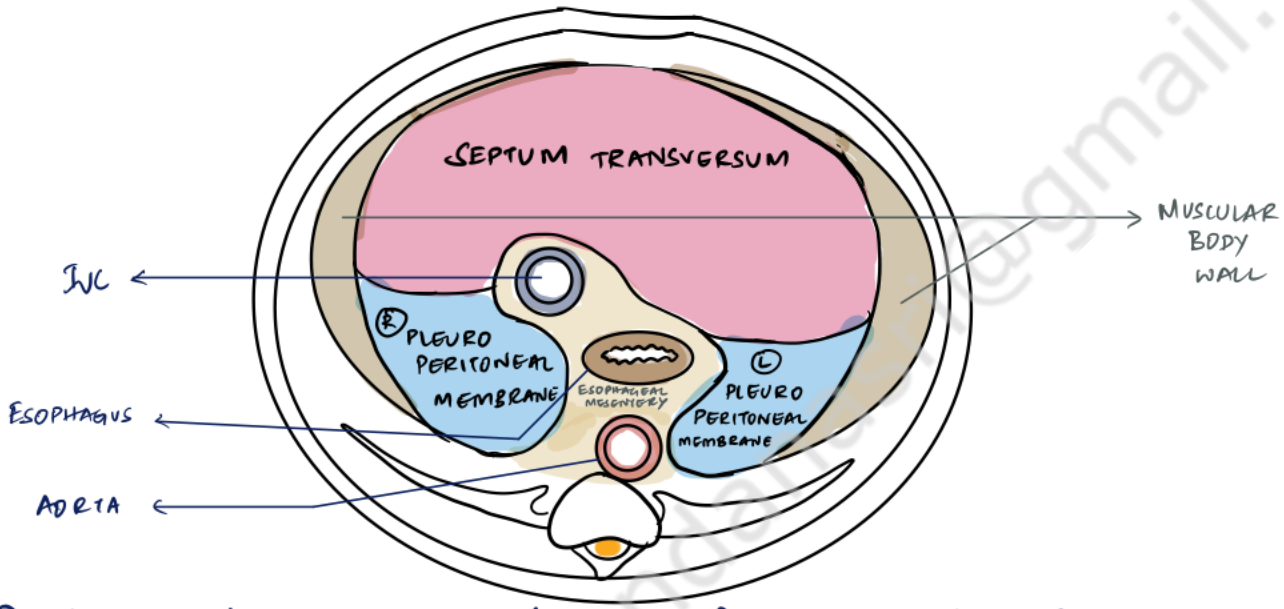
Surgeonlightanandanasri@gmail.com

CONGENITAL DIAPHRAGMATIC HERNIA

EMBRYOLOGICAL BASIS

The diaphragm is a musculotendinous partition that contains 4 embryonic components.

1) PLEUROPERITONEAL MEMBRANES
2) SEPTUM TRANSVERSUM
3) DORSAL MESENTERY OF ESOPHAGUS
4) MUSCULAR INGROWTH OF LATERAL BODY WALLS



- Diaphragmatic precursors begin to form in 4th week
- By 8th week, pleuroperitoneal membrane fully forms → ® first

Failure of complete formation of pleuroperitoneal membrane → CDH

If closure of pleuroperitoneal canal has not occurred by the time the midgut returns to the abdomen, the viscera will be misplaced into the ipsilateral thoracic cavity

↓

With this abnormal positioning, the midgut cannot undergo normal fixation ⟶ MALROTATION

DIAPHRAGMATIC HERNIA → Abdominal contents relocate into the thoracic cavity

↓

'Space occupying lesion'

↓

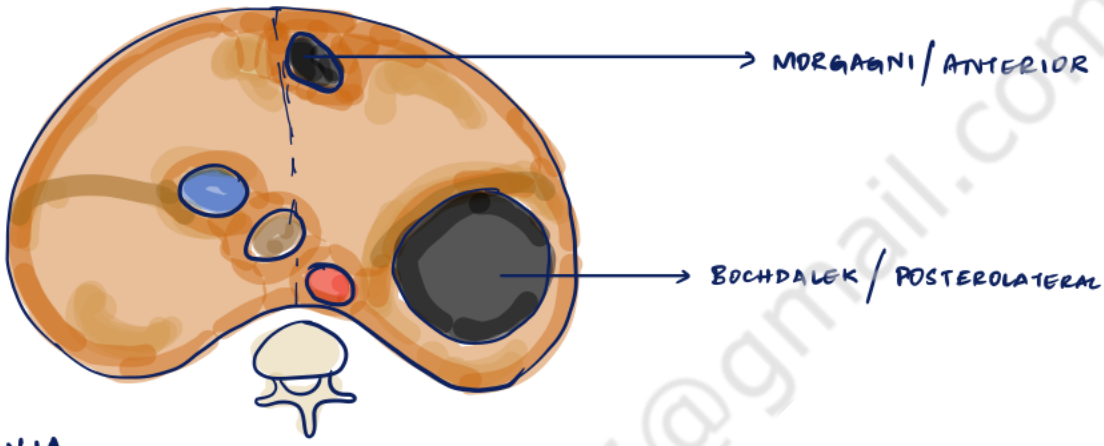
- Detrimental effects on fetal lung development → PULMONARY HYPOPLASIA
- Persistence of fetal circulation → ® to Ⓛ shunt → Worsening hypoxia

SurgeLight.chandan@gmail.com

EPIDEMIOLOGY

1) 1 in 2000 to 5000 live births
2) Left sided defects (85%.) > (R)
3) Overall survival rates 70-90%.

CLASSIFICATION



BOCHDALEK HERNIA

- Posterolateral defect of variable size (2-4cm to complete diaphragmatic agenesis)
- Majority (80%.) are seen on the Left side
- (R) sided defects (15%.) - rare - have very poor prognosis due to severe pulmonary hypertension / pulmonary hypoplasia
  may be associated with anomalous hepatic venous drainage
- Bilateral defects (5%.) very rare - invariably fatal

MORGAGNI HERNIA

- <2%.
- located anteromedially at the junction of septum transversum and antero lateral muscular body wall
- most often asymptomatic as amount of pulmonary compression is minimal - discovered incidentally as anterior mediastinal mass.
- typically, herniation of TRANSVERSE COLON into anterior mediastinum covered by a hernial sac - rarely incarceration / strangulation

ASSOCIATED : PENTALOGY OF CANTRELL

- Epigastric omphalocele
- Anterior CDH
- Sternal cleft
- VSD
- Ectopia cordis

Other associated anomalies - 20-40%. - Cardiac (20-40%) - VSD, TOF, TGV, CoA
Beckwith Wiedemann - limb (30%.) - shortening, costovertebral defects
Tracheobronchial stenosis, Esophageal atresia, cleft palate

Sunbright chauhanasri@gmail.com

EVALUATION

PRENATAL DIAGNOSIS

USG - Fetal stomach in thorax
Polyhydramnios – 75%. d/t GI obstruction
Liver herniation → poor prognosis
Mediastinal shift
Size of ipsilateral lung → Lung head ratio (<1 → poor survival)

↓ f/s/o CDH → search for associated anomalies
Karyotyping ± CVS

↓ Plan delivery at equipped tertiary care hospital

Fetal MRI

POSTNATAL DIAGNOSIS

Clinical presentation depends on degree of pulmonary hypoplasia & PHTN

Respiratory Distress - Pallor
Cyanosis
Grunting
Sternal retractions

CXR → loops of bowel in thorax

NG tube → helps determine position of stomach

Echo for cardiac anomalies
pulmonary hypertension

Ⓡ sided defect - CT/MRI for hepatopulmonary fusion
vascular anatomy of mediastinum & liver

Differential Diagnosis:

CONGENITAL CYSTIC ADENOMATOID MALFORMATION
BRONCHOGENIC CYST
DIAPHRAGMATIC EVENTERATION
CYSTIC TERATOMA

SurgeLightchandrasi@gmail.com

MANAGEMENT

- **Medical Optimization:**
  No rationale behind immediate reduction of hernia to improve lung function as lungs are HYPOPLASTIC (not atelectatic)
- Resuscitation - prompt ETT →
  (Bag & mask ventilation must be avoided → bowel distension → worsens ventilation)
  PREVIOUSLY - aggressive hyperventilation & induced alkalosis
  currently - Permissive hypercapnia (as long as pH > 7.2)
- NG decompression
- Ventilatory adjunct: HFO, ECMO
- Timing of repair
  ≥ 48 hr of STABILIZATION

SURGERY

Open approach

↓
Subcostal incision on side of defect
↓
Reduction of hernia by gentle downward traction
↓
DEFECT

- **Small / medium** → Primary closure
- **Large** → Prosthesis
  - GORE-TEX
  - Biologic
  - Absorbable

VERY LARGE DEFECT ± LOSS OF DOMAIN
↓
Silastic sheet between fascial edges
↓
temporising measure
↓
Ventral hernia
↓
Delayed closure

Thoracoscopic approach

- Improved cosmesis
- Improved surgical field visualization
- Avoidance of thoracotomy associated musculoskeletal deformities

MANAGEMENT OF MORGAGNI HERNIA

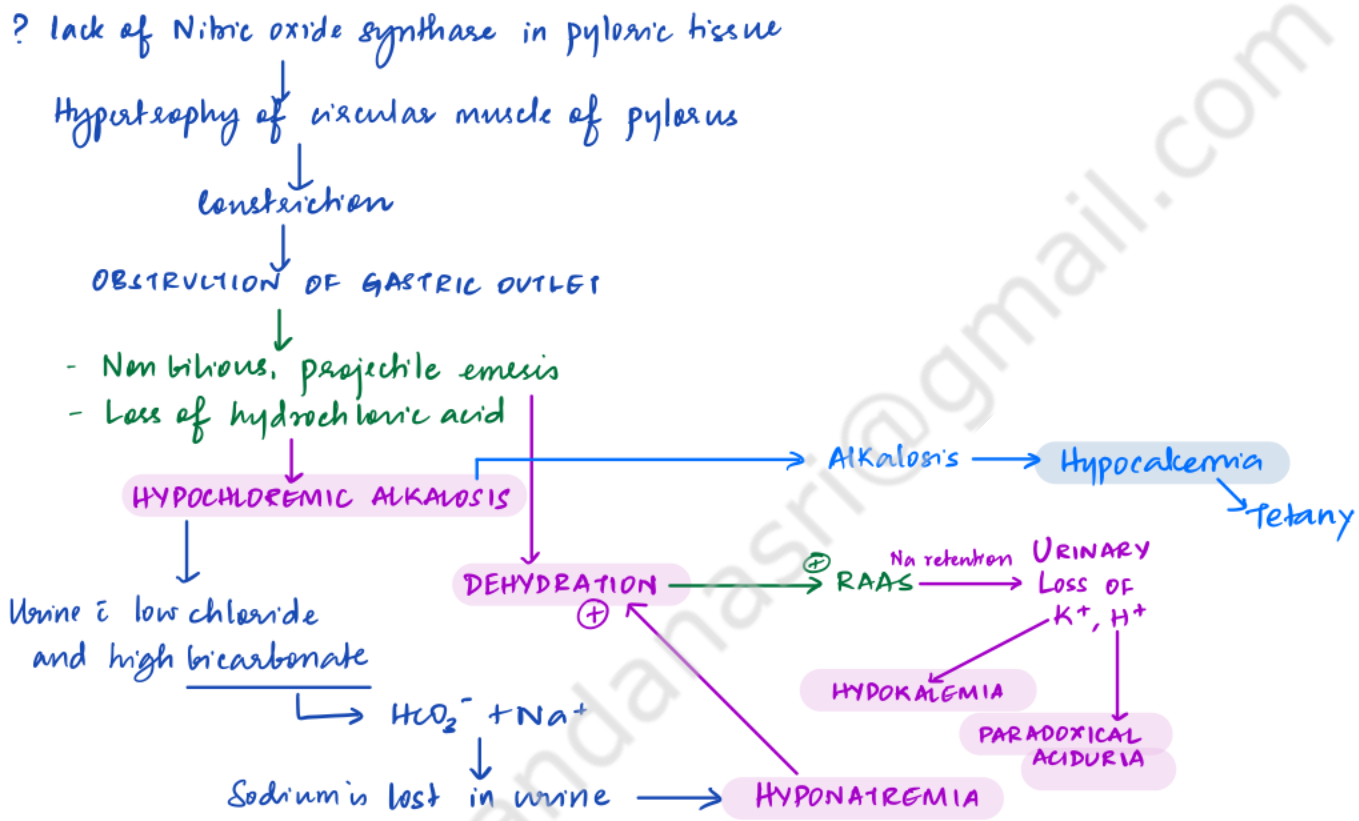
- Repair is recommended in children
- Asymptomatic hernia in adults may be observed
- Hernia sac - dense adhesions to pericardium

Surgelight chandanasri@gmail.com

INFANTILE HYPERTROPHIC PYLORIC STENOSIS

- Disease of newborns
- 1 in 300-900 live births
- 2-8 weeks of life
- M:F :: 4:1

PATHOPHYSIOLOGY



CLINICAL PRESENTATION

1) Progressively worsening non-bilious emesis
   - ↑ frequency
   - forceful
   - projectile
2) Visible gastric peristalsis (L → R)
   - LUQ epigastrium
3) Palpable 'olive' in epigastrium

EVALUATION

1) Chest X-ray → enlarged gastric bubble
2) USG → Pyloric muscle thickness >3-4mm / Pyloric length >15-18mm
   - NARROWED PYLORIC CHANNEL - "String sign"
   - FOLDING OF RUGAE - "Double track sign"
   - PYLORIC BULGE INTO ANTRUM - "pyloric beak/shoulder sign"
3) ABG & Electrolytes

SurgeLight chandahasri@gmail.com

MANAGEMENT

- **MEDICAL STABILIZATION**

Dehydration, Acid-Base & Electrolyte imbalance → MUST BE CORRECTED PRE-OP TO PREVENT POST-OP APNEA

- Initial rehydration with NS (10-20ml/kg bolus) until urine output is established
↓
- Correction of Hypokalemia by (D_5/ $\frac{1}{2}$NS + 20-30meq/L KCl)
↓
Repeat until alkalosis normalises
(Serum bicarbonate < 30meq/L)

MECHANISM OF APNEA
Metabolic alkalosis
↓ Decreased Respiratory Drive
Compensatory retention of respiratory CO_2
BY HYPOVENTILATION & APNEA

- **SURGERY**

RAMSTEDT PYLOROMYOTOMY
- Open
  - RUQ incision
  - Periumbilical incision
- Laparoscopic

Hypertrophied pylorus is incised from gastroduodenal junction to just beyond the hypertrophic mass
↓
incision is deepened by splitting muscle fibres
↓
INTACT MUCOSA SHOULD BULGE BEYOND THE DIVIDED MUSCLE EDGES

Complications:
- Perforation
- Incomplete pyloromyotomy

Feeds resumed 6-8 hrs post-op

SurgeLight chandanasri@gmail.com

ANORECTAL MALFORMATIONS

Definition: A spectrum of congenital malformations in which the anus fails to open normally onto the perineum

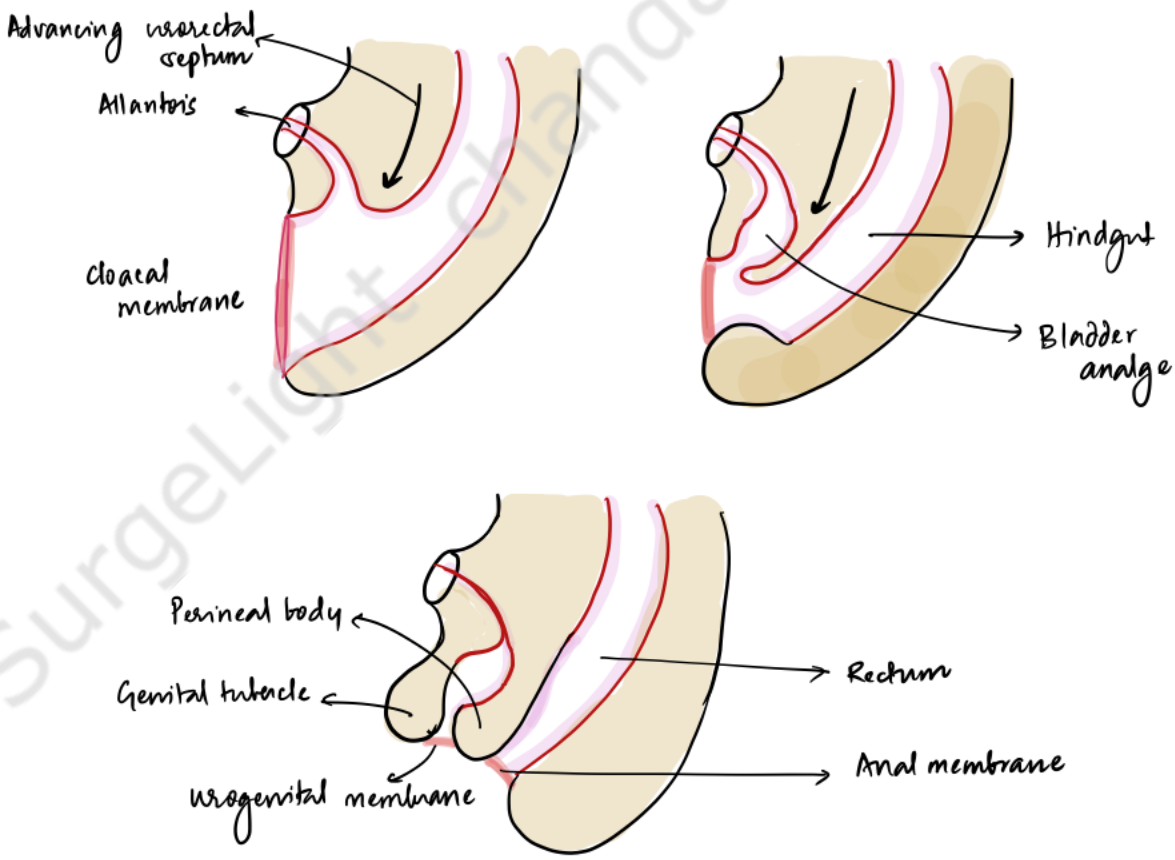
EMBRYOLOGICAL BASIS:

- Urorectal septum moves caudally to divide the cloaca into
  - Anterior urogenital sinus
  - Posterior anorectum

Failure of this septum to form → Fistula between bowel and
- Vagina ♀
- urinary tract – ♂

- Complete / partial failure of anal membrane (part of cloacal membrane) to resorb

→ ANAL MEMBRANE / STENOSIS (IMPERFORATE)



SurgeLight chandanasri@gmail.com

TYPES

WINGSPREAD INTERNATIONAL CLASSIFICATION

	MALE	FEMALE
HIGH (Rectum ends supralevator)	Anorectal Agenesis ī fistula < Rectovesical, Recto prostatic ī out fistula Rectal atresia	Anorectal agenesis ī fistula - Recto vaginal ī out fistula Rectal atresia
INTERMEDIATE (Rectum ends within Levator)	Recto bulbar fistula Anal agenesis ī out fistula	Rectovaginal fistula Recto vestibular fistula Anal agenesis ī out fistula
LOW (Rectum ends infralevator)	Perineal fistula (Anocutaneous) Anal stenosis	Anovestibular fistula Anterior ectopic anus (Anocutaneous) Anal stenosis

PENA CLASSIFICATION

	MALE	FEMALE
WITH FISTULA	Rectovesical fistula Recto urethral fistula < Prostatic, Bulbar Perineal fistula	Persistent Cloaca <3cm common channel >3cm common channel Vestibular fistula Perineal fistula
WITHOUT FISTULA	• Imperforate anus without fistula • Rectal atresia	• Imperforate anus without fistula • Rectal atresia

SurgeLight chandanasri@gmail.com

FEATURES OF THE TYPES OF ANORECTAL MALFORMATIONS

MALES

1) PERINEAL FISTULA
- usually a/i median bar (BUCKET HANDLE)
- Opens usually along / at the tip of median bar
- May open on either side of median bar
- Distal rectum is partially surrounded by sphincter complex

2) RECTOBULBAR FISTULA
- Relatively long common wall b/w urethra & rectum
- Voluntary sphincters well developed
- Visible pit at anal site

3) RECTOPROSTATIC FISTULA
- Voluntary sphincters - more hypoplastic
- Sacral dysraphism ⊕
- Pit ±, Flat bottom ⇒ sphincter hypoplasia

4) RECTOVESICAL FISTULA
- usually opens into bladder neck
- Common wall is small
- Severe sacral dysraphism
- Flat bottom; Very poor voluntary sphincter

NO FISTULA

5) ANORECTAL ANOMALY : NO FISTULA
- Rectum ends blindly
- Well formed anal pit
- Well developed voluntary sphincters
- Typical anomaly in Down's Syndrome

6) RECTAL ATRESIA
- 2° to ? Intrauterine vascular accident
- Normal looking anus ending blindly above dentate line
- Normal voluntary sphincter muscles
- Proximal blind bowel connected to blind ending anus by short distance of fibrous tissue / membrane

FEMALES

1) PERINEAL FISTULA
- Between anal site and vestibule (ant median bar
- More anterior fistula opening NOT in sphincter complex
- Perineal groove between bowel outlet & vestibule

2) ANTERIOR PERINEAL ANUS
- (N) / moderately stenotic anus situated anteriorly, just behind vestibule
- (N) sphincter - posterolaterally
- poor sphincter - anteriorly

3) RECTOVESTIBULAR FISTULA
- opens in posterior fourchette of vestibule behind hymen - 2-4 cm common wall
- well developed voluntary sphincters
- a/i vaginal anomalies

4) RECTOVAGINAL FISTULA
very rare

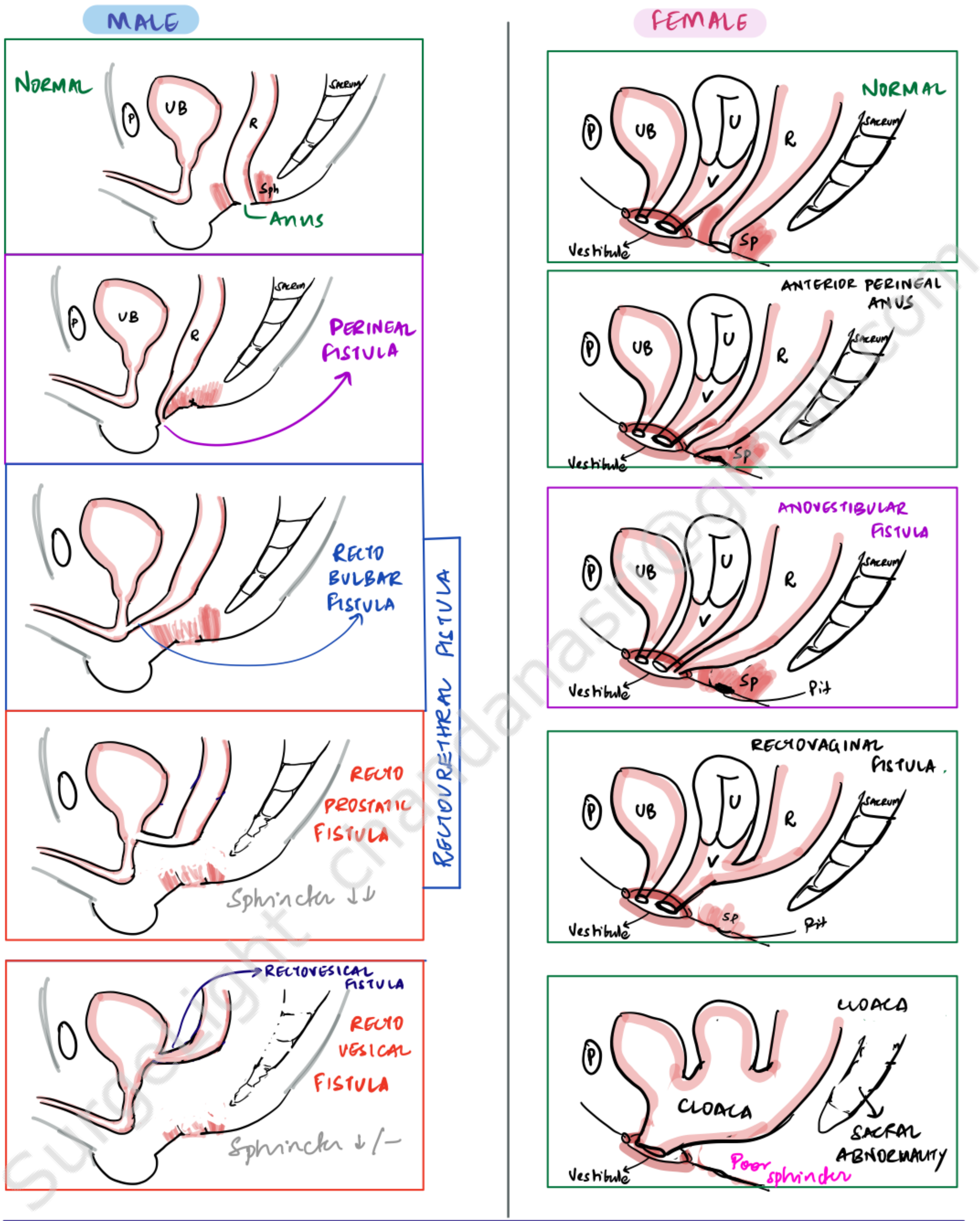
5) CLOACA
- Common channel for urinary tract, vagina & rectum → length 1-5 cm
  ↓
- can drain anywhere - but m/c - inf post. vagina
- long common channel ⇒ Poor sphincters
- can be a/i obstructive uropathy
- a/i mullerian anomalies
- a/i severe sacral anomalies

NO FISTULA

6) A.R.M WITHOUT FISTULA
7) RECTAL ATRESIA

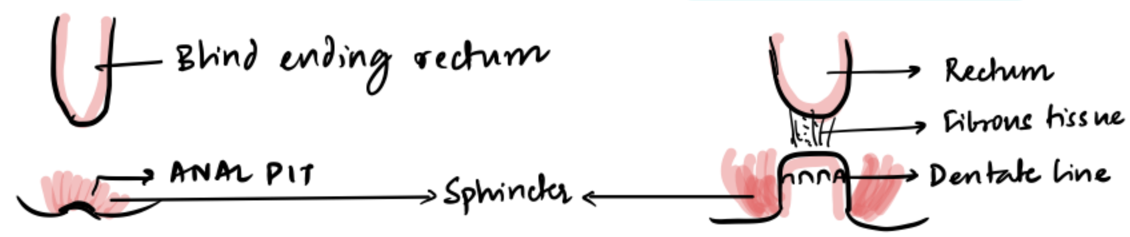
} Similar to males

Surgeonlight chandanasr@gmail.com



Common to both genders

IMPERFORATE ANUS | RECTAL ATRESIA



Surgeright chandanasri@gmail.com

EPIDEMIOLOGY

1) 1 in 5000 live births
2) Males are affected more (58%)
3) Males have more severe malformations than females
4) May be Syndromic / Non Syndromic ARM

- VACTERL
- Klippel Feil S°
- Trisomies - 21, 13, 18
- Cat-Eye S°
- Hirschsprung disease
- Opitz syndrome etc.

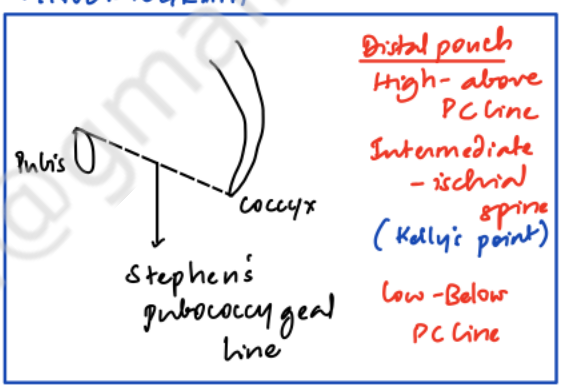
Associations
- Urogenital anomalies
- Skeletal anomalies
- Gastrointestinal anomalies
- Cardiovascular anomalies
- CNS anomalies
- Overall association - ~65%

EVALUATION

- Detailed physical examination - Perineal inspection
- Rule out associated anomalies

[Spine, Sacrum - USG
Renal USG
Urinalysis
R/O FA-TEF
2D Echo]

- INVERTOGRAM



- MURUGASSU - Sitting position
Needle introduced into rectal pouch → meconium aspirated → contrast injected → X-Ray

Historically, (Wangenstein) invertogram was done ; coin taped to anal dimple → causes discomfort to the baby

18-24 hrs later (Before that, rectum is collapsed - so false High ARM)

CROSS TABLE LATERAL RADIOGRAPH

± Perineal USG ± CT/MRI for sphincter anatomy

Rectal gas distal to coccyx
- Stable
- No other significant anomalies
→ PSARP ē/ēout covering stoma

Rectal gas proximal to coccyx (Rectal atresia >1cm gap)
→ + Meconium in urine (Rectourethral / Rectovesical fistula)
Abnormal Sacrum, Flat bottom (Cloaca, Vestibular fistula)
→ COLOSTOMY — End colostomy ē mucous fistula — Sigmoid/descending colon
→ Later, distal cologram (3-8 wks)
→ Delineate distal anatomy

SurgeLight.chandanasri@gmail.com

DEFINITIVE SURGERIES - PROCEDURES

Low ARM - Incisional of Anal membrane
Anovestibuloplasty
Anoplasty
→ Anal dilatation

PSARP - Posterior Sagittal Ano Recto Plasty

PSARVUP - Posterior Sagittal Ano Recto Vagino Urethro Plasty

TUM - Total Urogenital Mobilization

MALES

1) PERINEAL FISTULA
Minimal PSARP / Cutback

2) RECTOURETHRAL FISTULA
PSARP

3) RECTOVESICAL FISTULA
PSARP ± Laparotomy

4) ANORECTAL ANOMALY : NO FISTULA
PSARP

5) RECTAL ATRESIA
PSARP

FEMALES

1) PERINEAL FISTULA
Limited PSARP

2) ANTERIOR PERINEAL ANUS
Limited PSARP

3) RECTOVESTIBULAR FISTULA
Limited PSARP

4) CLOACA
PSARVUP ± Laparotomy + TUM

PSARP - determining sphincter location by electrical stimulation
Posterior midline incision from coccyx to anal pit
identifying & dividing fistula
Mobilising rectum
Anoplasty
↓
Serial anal dilatations

SurgeLight chandanasri@gmail.com