

PR ↑ common finding

ECG changes in electrolyte abnormalities seen in kidney disease

Hyperkalaemia	Hypokalaemia
<p>K ↑ </p>	<p>K ↓ </p>
Tall Tented T	T wave inversion
<p>STE (MI, acute pericarditis, variant A)</p>	ST depression
<p>P wave ⊖ (A-FIB, sick sinus syn, K ↑)</p>	<u>Pseudo-P</u> -pulmonale
QRS wide/broad > 100 msec	<u>P</u> rominent U wave
<p></p>	<p>Prolonged QU interval < 1.5 msec: diaphr. paralysis</p>
<p>DIASTOLIC CARDIAC ARREST → Cal. chloride 😊</p>	Rx: KCL + IVF

K⁺: 7.0 meq/L

K⁺: 8.0 meq/L

death:

most effective to lower K⁺

U delay of papillary muscles

- Rx:
1. IV b.i. Cal gluconate (ANTAGONISM)
 2. REGULAR insulin 10U + 25% - 50% D infusion
 → K⁺ ↓: 0.5 - 1 meq/hr [K⁺ INFLOX]
 3. Neb c salbutamol [K⁺ INFLOX]
 4. IV Lasix (KALIURIA)
 5. K⁺ binder: PATIPOMER
 6. Refractory K⁺: H.D

1. Cal. CARBONATE 😞

* Not routinely used in K ↑ ⇒ Na bicarbonate

Renal tubular acidosis

Type 1

Causes: SJOA REN and amphotericin B - α : H^+ excretion - β : HCO_3^- "

Mnemonic: Alpha can't acidify, Stones Fly and potassium says bye

DCT

Defect in alpha intercalated cells	Inability to acidify urine and urine pH > 5.5
Impaired function of H-K ATP ase	
Low urinary citrate and alkaline urine	<p>2. NEPHROCALCIOSIS</p> <p>3. KALIURIA : Hypokalemia</p>
Can't excrete H^+	Ca^{++}

Type 2

Causes: Fanconi syndrome, Wilson and multiple myeloma

- Impaired bicarbonate resorption from PCT

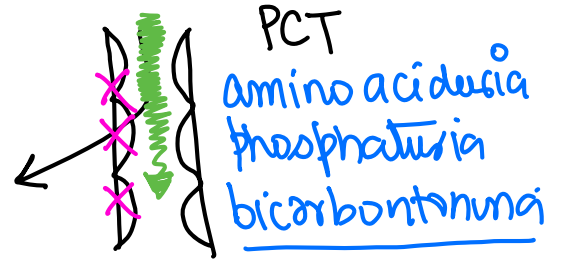
- Serum bicarbonate: <15 meq/L

- Salt loss occurs - increased distal sodium delivery - hyperaldosteronism and hypokalemia

65f: salt

AA
PO4

Salt loss



PCT
amino aciduria
phosphaturia
bicarbonaturia

Type 4

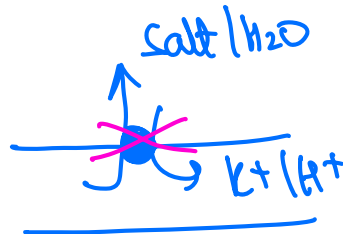
Cause: D. nephropathy

High sugar damages the JG apparatus plus tubulointerstitial fibrosis damages principal cells in collecting duct which is the site of aldosterone resistance

ENac: Resistance to aldosterone

Hyperkalaemia with inability to acidify urine

Renin \downarrow aldosterone \downarrow
principal cells: site of action of aldosterone
CD-DCT



\rightarrow impaired H^+ excretion
inability to acidify urine
 $\rightarrow \uparrow K^+$

all causes of RTA: NAGMA

(hrs) * acute onset : fast correction
 (days) * chronic onset : slow correction

Electrolytes

Oat cell ce lung, legionelle, Carcinoid Tumor

Hyponatremia

osmotic demyelination syn

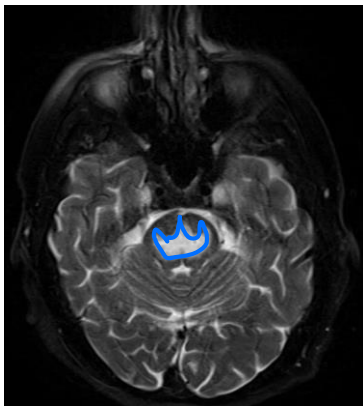
Fast correction of chronic hyponatremia leads to stroke like feature called ODS

Symptoms usually appear 2-6 days after correction

Mild	Moderate <i>Corticospinal</i>	Severe <i>#</i>
Confusion	Quadripareisis	Locked-in syndrome
Dysarthria ✓	Spastic paralysis	Conscious but cannot move limbs
Dysphagia	Pseudobulbar palsy	

(CANT SPEAK, EAT, DRINK)

MRI findings of ODS shows trident sign



Q. 28-year-old male collapses at the finish line of a full marathon conducted in hot weather. He is confused and has a generalized tonic-clonic seizure in the emergency department. On examination: BP: 110/70 mmHg, HR: 96/min. History reveals he consumed excessive plain water during the race.

Labs show

- Serum sodium: 118 mEq/L
- Serum osmolality: low
- Urine osmolality: elevated

* Na⁺ ↓: excessive sweating

Acute onset Hyponatremia

What is the most appropriate immediate management?

- Fluid restriction and slow correction with normal saline
- 3% hypertonic saline bolus (100 mL over 10 minutes)
- 3% hypertonic saline infusion with slow correction @6 meq per 24 hours
- Demeclocycline therapy

CNS + features

✓ ? lung malignancy
 Q. A 62-year-old chronic smoker presents with fatigue, headache, and progressively increasing confusion for 10 days. He has lost 6 kg over the last 3 months. Examination reveals decreased breath sounds over the right upper lung field and disoriented patient.

Investigations:

- Serum sodium: 118 mEq/L
- Serum osmolality: Low
- Urine osmolality: High
- Urine sodium: Elevated
- Chest X-ray shows a right hilar mass

↓ L lung MASS : collapse lung segment

SIADH: d/t lung MASS

ADH ++: H₂O ++ Na ↓
 chronic Hyponatremie

What is the most appropriate management?

- 3% hypertonic saline bolus (100 mL over 10 minutes)
- 3% hypertonic saline slow correction not exceeding 6–8 mEq/L per 24 hours
- Rapid correction with normal saline infusion
- Demeclocycline therapy as immediate management

✓ Q. 55-year-old patient of lung cancer admitted for work up developed seizures. Labs shows Na= 120 meq/L, serum potassium = 4.0 meq/L, Calcium 10 mg/dl. Weight = 60 kg. Calculate the total correction to be given to this case over the next 24 hours?

- 180
- 360
- 600
- 720

Ch. Hyponatremia

$$(\text{desired value} - \text{actual value}) \times \text{TBW}$$

$$(126 - 120) \times \text{TBW}$$

$$6 \times 60 \times 0.5$$

TBW = wt x 0.6 ♂
 0.5 ♀


Total CORRECTION ⇒ (140 - 120) x 60 x 0.5
 20 x 30
 600 meq/l

Version 1


Version 2:

oat cell Ca lung, carcinoid Tumor
 legionelle
 meningitis, Encephalitis, brain abscess
 CEREBRAL Toxoplasmosis

Lab status in SIADH

Serum sodium $H_2O ++$	Low Euvolemic $Na \downarrow$
Serum osmolality $u. osm \propto \frac{1}{p. osm}$	Low Dilute \downarrow 
Urine osmolality	High CONCENTRATED \uparrow
Urine sodium $H_2O ++$	>40 meq/L due to ANF liberation) Rt atrium sketch \oplus : ANF levels \uparrow : natriuresis
Volume status Rx: -	Euvolemic 1. FLUID RESTRICTION 2. SALT CAPSULES 3. (NATRIE) VAPTANS

(if unconscious/unresponsive/seizures \oplus : 3% saline)

Electrolyte imbalance	Possible leading Cause of death	Intervention
Hyponatremia < 125	SEIZURES	3% saline $<$ bolus: A infusion: C
Hypernatremia > 158 <small>Elderly neonates</small>	SEIZURES	5% DEXTROSE $\frac{N}{2}$ in 5% D
Hypokalaemia < 1.5 meq	D. PARALYSIS 	KCL + IVF 1 amp = 10 ml kcl = 20 meq
Hyperkalaemia > 8.0 meq	Cardiac ARREST diastolic: Cal. gluconate	20 meq \rightarrow 0.25 meq/L \uparrow kcl
Hypocalcaemia < 7 mg/dL	LARYNGOSPASM: Cal gluconate	1st line: NS + lasix
Hypercalcemia > 13 mg	SYSTOLIC ARREST: $<$ (syptolic)	Ibandronate steroids
Hypermagnesemia > 10 meq	Asystole, bradycardia and Nm paralysis	Calcium gluconate iv
Hypomagnesemia	Ventricular arrhythmias and tetany like - carpedal spasm Torsades de pointes \uparrow	MgSO4 iv

PRITCHARD Regimen: eclampsia: 14g \checkmark
 * 4g (20%) iv + 5g (50%) 1m in
 each buttock \checkmark

DTR \downarrow : most
 RR \downarrow : reliable
 u.o \downarrow : sign

Additional Notes

RTA

1	2	4
SJOGREN LAMB	WILSON expiry date: Tetracycline Fanconi syndrome	D. nephropathy
DCT # α intercalated cell	PCT # lining of PCT	CD # Principal cells ENac # ↷
INABILITY TO acidify urine - nephrocalcinosis - K ↓	Aminoaciduria KALIVRIA phosphaturia	INABILITY TO acidify urine - K ↑

HAGMA

- K: KETOACIDOSIS
- U: ATN, AGN, AKI, CKD
- L: LACTIC ACIDOSIS
- T: TOXINS

NAGMA

- D - diarrhea ←
- R - RTA 1, 2, 4
- F - FISTULA
- U - UTERED SIGMOIDOSTOMY
- S - (divert urine into colon)
- E -

Kt
⊖

$$\begin{aligned}
 &(\text{Na}^+) - (\text{Cl}^- + \text{HCO}_3^-) \\
 &(\text{140}) - (106 + 24) = 10 \text{ meq}
 \end{aligned}$$

* (N) AG = 6-12 meq/L

Additional Notes

① URINE : < 3 RBC/HPF in centrifuged specimen

HEMATURIA

> 5 RBC/HPF x 3 consecutive Reports

OR
> 100 RBC/HPF : single Report

* PAINFUL HEMATURIA : Nephrocalcinosis

Urine m/c: RBC ++

Ca Oxalate stones → envelope shaped

STRUVITE | STAG HORN | TRIPLE Poy: Mg NH₄ Poy

UREASE (+)



alkaline pH



* PROTEUS mirabilis



→ Hexagonal crystal

* PROVIDENCIA

CYSTINURIA

* IOC: Renal colic : NCCT Abdomen

CECT Abdo



= POLYURIA, URAEMIA features
S. Creatinine ↑

Renal stones

Rx: IVF

* ≤ 2 cm

⇒ E.S.W.L

> 2 cm

⇒ PCNL



Additional Notes

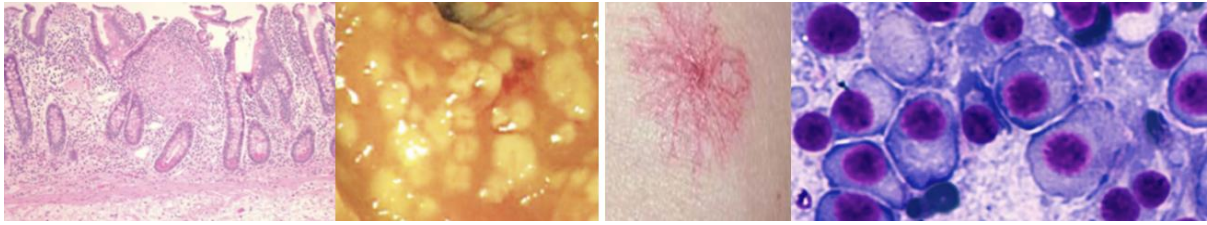



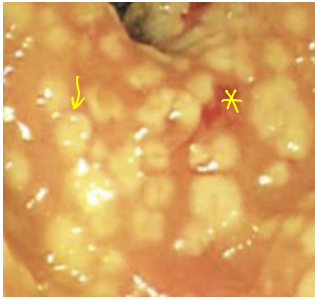
Additional Notes



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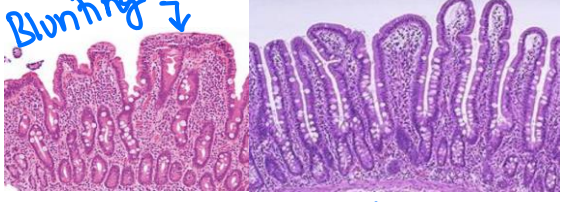
RR LIVER GIT system



PSEUDOMEMBRANOUS COLITIS	
<p>Cause: Overgrowth of <u>Cl. difficile</u> that produce toxins A (enterotoxin) & B (cytotoxin)</p> <p>Triggers</p> <ul style="list-style-type: none"> • Cephalosporins • Clindamycin • Fluoroquinolones • Ampicillin / Amoxicillin • Prolonged <u>PPI</u> use <p>Clinical features</p> <ol style="list-style-type: none"> 1. Profuse watery diarrhea 2. Abdominal pain, <u>fever</u> 3. Leukocytosis <u>TLC ↑</u> 4. Recent antibiotic exposure (oral or IV), often in a hospitalized or elderly patient. <p>Screening</p> <ol style="list-style-type: none"> * 1. Stool <u>GDH</u> antigen 2. Stool EIA for toxin A and B <p>IOC Stool <u>PCR for Toxin A, Toxin B gene</u></p> <p>Colonoscopy: Yellow-white raised plaques (pseudomembranes) with patchy distribution over erythematous mucosa in colon</p>	  <p style="text-align: right;"><u>Colonoscopy</u></p> <p>Rx: <u>MACROLIDE</u></p> <p>First episode: <u>Fidaxomicin</u></p> <p>Severe or relapsed PMC: <u>Vancomycin</u> <u>ORAL</u></p> <p><u>Multiple recurrences:</u> Faecal microbiota transplant</p> <p>m/c: Trophozoites ± ingested RBC : bacilli + PMN</p> <p>↓ Shigella ↓ E. Histolytica</p> <hr style="border: 1px solid blue;"/> <p style="background-color: yellow; border: 1px solid black; padding: 5px; display: inline-block;">blood in stool ⊕</p>

Pg. 159 Antibiotic induced colitis ⇒ Klebsiella oxytoca
 Post (Augmentin) ↓ (blood in stool)

Microvilli # : surface area ↓ : OSMOTIC DIARRHEA

CELIAC SPRUE		Barley, Rye, oats
 <p>Blunting ↓</p>		<p>BROW is CI wheat</p>
<p>IOC- IgA anti-tTG antibody = duodenum Jejunum</p>		<p>HLA DQ2 DQ8 : why?</p>
<p>* Anti endomysial antibody- Highest specificity</p>		<p>Rx ↓ Quinoa, Bajra, maize and rice with iron and Folic acid is given</p>
<p>If IgA deficient ↓</p> <ol style="list-style-type: none"> 1. IgG anti-tTG 2. Anti- Deamidated Gliadin Peptide 		<p>dimorphic Anemias</p>
		<p>Follow up required for</p> <ol style="list-style-type: none"> 1. T1 DM : Insulinopenia 2. DH * 3. EATL → lymphoma of gut death ENTEROPATHY associated T-cell lymphoma

MARSH classification

gold std: Small intestinal Mucosal Bx

* BEFORE: villous atrophy
AFTER : Regeneration of villi
exclusion of gluten

DERMATITIS HERPETIFORMIS

gluten allergy I

Teenager, PRURITIC PAPULES ± excoriation marks

WORK UP
- elbow
- knee

Skin Bx → Light microscopy = Papillary Tip microabscess
IF → Focal IgA deposits at D-E-J

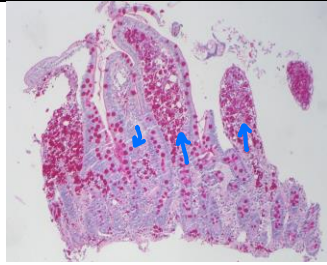
anti Epidermal Transglutaminase Alb

Rx ⇒ DAPSONE

IOC for malabsorption: 72-hour fecal fat estimation: > 7g fat (Sudan III stain)

fat malabs^N → BREATH TRIOLEIN TEST

Whipple disease



PAS+

Hepatocytes = α_1 AT ↓
 duodenal sample = Whipple
 BMA = lymphoblast ALL

Cause TROPHERYMI whipelli (bacteria)

1. Chronic diarrhea, steatorrhea, weight loss
2. Seronegative arthritis that precedes GI symptoms
3. Fever LG
4. Hyperpigmentation knuckles, IP joints

IOC: Duodenal biopsy

- * -PAS-positive macrophages in lamina propria showing rod shaped bacilli
- PCR on blood sample

Rx:

1. Ceftriaxone iv 2 wks
2. TMP SMX : 6-12 mths

Complications

- CNS cognitive decline
- CVS: culture negative endocarditis (Aortic > mitral)

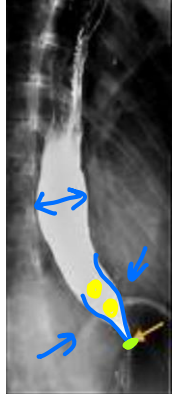
STERILE

CHAGAS: Mega ♡ (Dum), Mega colon, Mega esophagus

T. CRUZI: Reduviid bug: ROMANA SIGN

DISEASES OF ESOPHAGUS

Achalasia cardia



Ba swallow *

Shows smooth tapering defect

Bird beak
Pencil Tip

Sigmoid esophagus

Pathogenesis

APERISTALSIS, LES Tone ↑

Degeneration of inhibitory neurons (NO & VIP-producing) in the myenteric (Auerbach) plexus of the esophagus

Most common cause?

AUTOIMMUNITY

UGIE shows

☼ PUCKERING of lower esophagus

IOC

High resolution manometry :

LES Tone ↑↑

Chicago classification

Type 1	Type 2	Type 3
Absent peristalsis	-Absent peristalsis	Premature / spastic distal contractions
Incomplete LES relaxation	-Incomplete LES relaxation	
	-Uniform pressurization of entire esophagus	

Type 2 Best response to pneumatic dilatation and

Type 3 worst prognosis

Surgery of choice

↓ Tc

1. Heller's myotomy: Longitudinal division of LES muscle fibers to relieve + obstruction

2. Partial fundoplication (Dor or Toupet): Added to prevent postoperative GERD

Effective for all Chicago types, especially Type I & II

Young ♀

: Progressive dysphagia
* dysphagia (liquids > solids)
more

- * Halitosis
- * Regurgitation of food items
- * R. pneumoniae episodes
- * Chest pain ±

	<p>Other procedures</p> <ol style="list-style-type: none"> 3. PD 4. Botulinum toxin inj 5. CCB
--	---

* Pseudo achalasia → carcinoma stomach
 ↓
 Auerbach plexus #
 Achalasia

* Ba swallow — ZENKER DIVERTICULUM
 — HIATUS HERNIA

* Crohn's D — Ileum #
 — IOC: WCE > MR enterography (STRICTURES)
 — DOC: Budesonide, Prednisone, Mesalamine, INFLIXIMAB
 ✓ Halbmik → PERIANAL FISTULA
 ↓
 ileal release

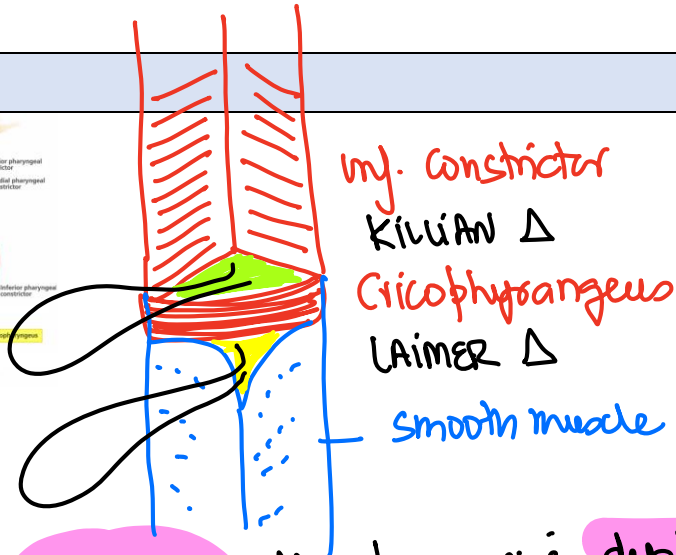
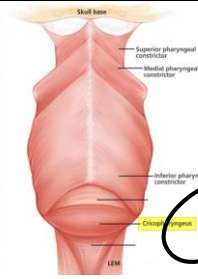
* UC — Rectum
 — IOC: Colonoscopy
 — DOC: MESALAMINE + Budesonide enema

✓ SEVERITY UC: TRUE LOVE & WITT'S CRITERIA

I.B.D = LA FEVER, bleeding, ESR ↑ CRP +

* STOOL/fecal: CALPROTECTIN (IBD vs IBS)

Zenker diverticulum



* Ba swallow
↓
outpouching +

70 - 80 yrs: Non progressive **dephagie**
 Halitosis, Regurg^N, pneumonia ±
 wt loss

Surgery

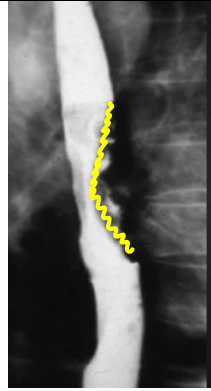
* 1st inv: UGIE

1. Dohlman procedure (Endoscopic diverticulotomy) : > 4 cm
2. Z POEM

↓
ZENKER

* ZENKER DEGENERATION
 TYPHOID (skeletal muscle)

Carcinoma esophagus : SCC : middle 11



SHOULDERING SIGN: irregular filling defect

50-70yr: dysphagia progressive

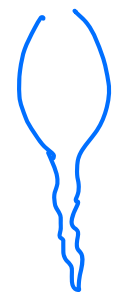
CACHEXIA (Solids > liquids)

Halitosis, Regurg^N, R. pneumoniae epis

WORK-UP: **UGIE**, HR manometry

↓ ba swallow, ba meal follow those

- UGIE + Bx PUNCH
- CT abdomen: LIVER
- Rx: surgical notes



Rat Tail app

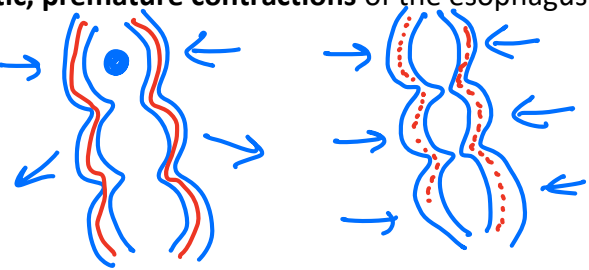
SEROSA absent in esophagus



CORK SCREW

DIFFUSE Esophageal spasm: idiopathic

Simultaneous, non-peristaltic, premature contractions of the esophagus with normal LES relaxation.



Jack hammer esophagus

Hypercontractile esophagus characterized by very high-amplitude, prolonged peristaltic contractions on HRM

* **CHEST PAIN** : Esophageal Angina^o

young ♀ : ****** intermittent episodes of chest pain at Rest
dysphagia, Regurgitation of food items

Inv : High Resolution O. manometry:

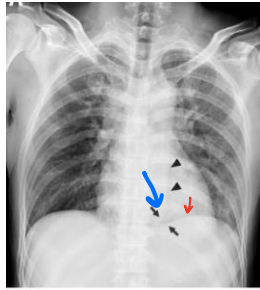
LES Tone ↑
achalasia (spastic)

Non-peristaltic contractions
DES

Rx: Anticholinergics, Tab Isorbide mononitrate
Amlodipine

↳ Peda edema, Reflex Tachycardia

Alcoholic develops multiple episodes of vomiting and retching followed by development of chest pain. CXR is shown First differential diagnosis



1. Mallory Weiss

do Hematemesis: submucosal Tear LES
 Cardia → LES: self-limiting
 Source: left gastric A

MACLEER TRIAD: Perforation of esophagus

Vomiting
 Chest pain chemical mediastinitis
 Pneumo-mediastinum → XRC
 Continuous diaphragm sign

2. Boerhaave syndrome

LOC: CT chest + gastrografin
 CONTRAST oral

3. Bleeding esophageal varices

↓
 TERLIPRESSIN

Rx: THORACOTOMY
 (Right sided)

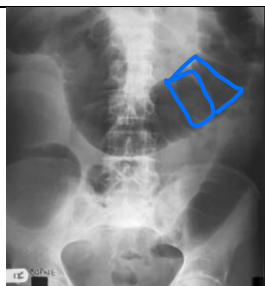


gas under diaphragm
 moose track sign

Pneumoperitoneum
 Why? - Hollow viscus PERFORATION
 ↑
 DU: D₁: ANTERIOR
 ↑
 H. PYLORI omentum
 ↓
 Rx: Exploratory lap + Graham patch



20yr ♀: colicky pain
 bilious vomiting
 XR Abdo: multiple dilated bowel loops
 Δ: SBO
 Why? - Adhesions, bands
 Ascariasis



60yr ♂: Abdo. distention
 Obstipation: cont per flatul
 H/o constipation NR laxatives
 XRay: Haustrations, dilated bowel loops
 Δ: LBO
 Why?: ? malignancy

neonate ⇒ day 2: bilious vomiting
 XRay Abdo: double bubble sign: DA: Rx: D-D
 XRay " : Triple bubble " : JA: Rx:

HEPATOLOGY

MC of acute viral hepatitis (adults)	HEV
Virus with highest chances of progression to chronicity <u> </u>	HCV
MC of chronic viral hepatitis (Total no of cases)	HBV
MC viral of Fulminant hepatitis	HDV + HBV (Superinfection) ↳ 20% chances
MCC of Fulminant hepatitis in pregnancy *	HEV ↳ 30% chances
MCC of hepatocellular carcinoma	Hep B > Hep C
DOC for Hepatitis B	TENOFOVIR, ENTECAVIR (HIV + HBV)
DOC for hepatitis C	SOFOBUVIR + VELPATASVIR NS-B NS-A
DOC for hepatitis D	↳ α- INTERFERON: immunity booster
MCC of OLT (orthoptic)	↳ MASH/NAFLD > Hep C Metabolic dysfunction associated steato Hepatitis

Steatosis:
T₁ ++
in hepatocytes

* HBV = 90% → spontaneous Resolution
* HCV = 90% CONVERSION To ch. hepatitis

≥ 5% Hepatocytes steatosis: fatty liver



Hepatitis B

→ ss/ds DNA virus :



ground glass Hepatocytes obesity, DM

Antigens

MCC Tx route: SEXUAL

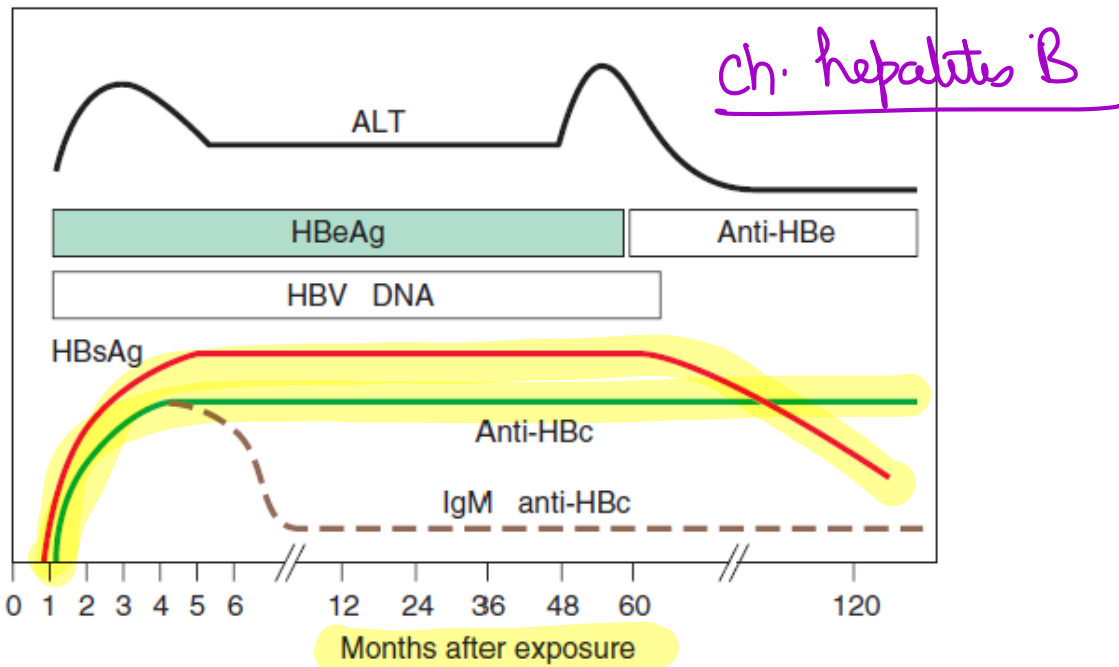
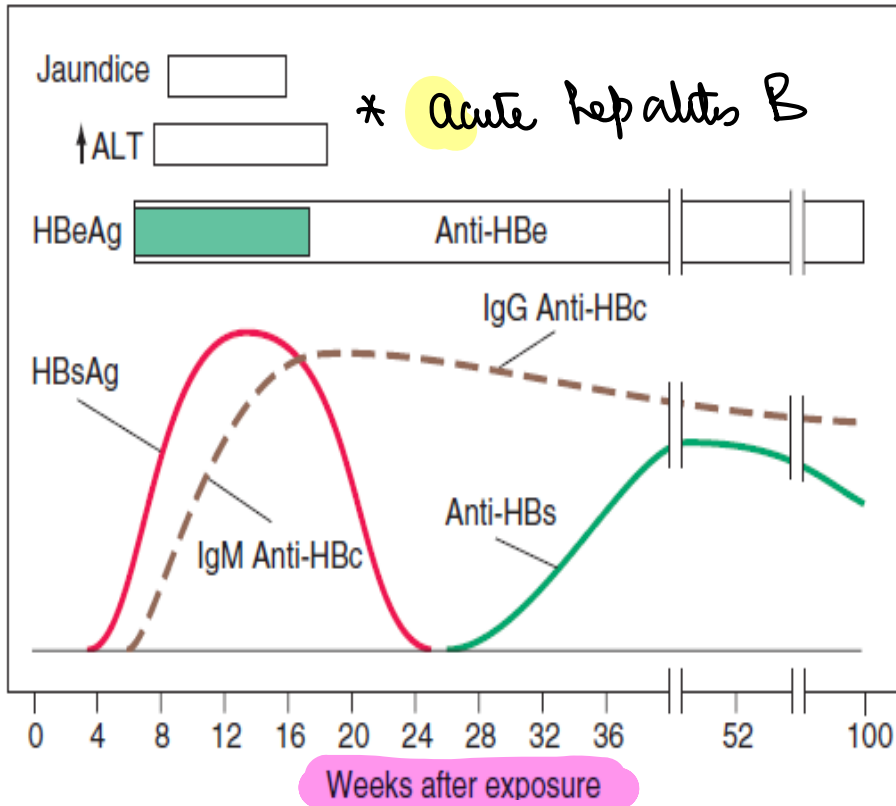
HBsAg	<u>earliest</u> SEROLOGICAL indicator
HBc Ag	NEVER TO APPEAR in blood
HB _e Ag	Replication/ INFECTIVITY
HB _x Ag	HCC : ⊖ CD 95 ⊖ apoptosis cytopathic effect
PCR → HBV DNA	≥ 2 × 10 ⁴ IU DNA/ml + (SGPT × 2) Rx: TENOFIVIR

* TRANSFUSION associated Hepatitis: HBV

Antibodies

1.	Anti HB _s	SERO CONVERSION
* 2.	Anti HB _c	IgM ⇒ acute hep B, GAP PERIOD IgG ⇒ ch. hep B, Remote infection
3.	Anti HB _e	INFECTIVITY ↓

Serological interpretation



HBsAg	Anti HBs	Anti HBc	HBe Ag	
<u>+</u>	-	<u>IgM</u>	+	acute hepatitis B highly infectious
<u>+</u>	-	<u>IgG</u>	+	chronic hepatitis B highly infectious
-	+	-	-	vaccinated
-	-	IgM	-	gap period
-	-	IgG	-	Remote infection

→ stellate cells: FIBROSIS ⊕

CIRRHOSIS

-MCC is NAFLD (>5% of hepatocytes showing steatosis)

-MCC of need for OLT IS NAFLD

-Non-invasive imaging for NAFLD is Fibro-scan ^{Transient} ELASTOGRAPHY

-Drugs approved for NAFLD

1. Resmetirom ✓

MOA: Thyroid hormone receptor-β (THR-β) agonist : ↑ Hepatic fat metabolism and reduces liver fat

2. Semaglutide (GLP-1 receptor agonist)

Lanifibranor: Pan-PPAR agonist in phase III trials

Scores for severity of cirrhosis **ALBUMIN**, **ASCITES**, **ASTERIXIS** : NH₃ ↑

*

*Child-Turcotte-Pugh Class obtained by adding score for each parameter (total points)

Class A = 5 to 6 points

Class B = 7 to 9 points

Class C = 10 to 15 points

value ≥ 8 : Register for O.L.T

(B) 8

MELD Score	MELD 3.0
<p>* C: S. CREATININE B: S. BILIRUBIN I: INR (Hepato-Renal syn) 6-40 = Range >17: Register for OLT</p>	<p>* CBI : ✓ + * Sodium * albumin INR: PT 😊 aPTT 😞</p>

Ascites

Serum albumin ascites gradient

SAAG > 1.1 g/dl		SAAG < 1.1 g/dl
<p>Ascitic Fluid Protein < 2.5 g/dL</p>	<p>Ascitic Fluid Protein > 2.5 g/dL</p>	<p>↓ T.P.N 1. TB 2. PERITONEAL carcinomatosis - Ca ovary - Ca stomach 3. PANCREATITIS 4. Nephrotic syndrome</p>
<p>Alcoholic liver disease Portal vein thrombosis * Late Budd-Chiari syndrome</p>	<p>CHF Early Budd-Chiari syndrome Sinusoidal obstruction syndrome (veno-occlusive disease) → Endemic Ascites</p>	

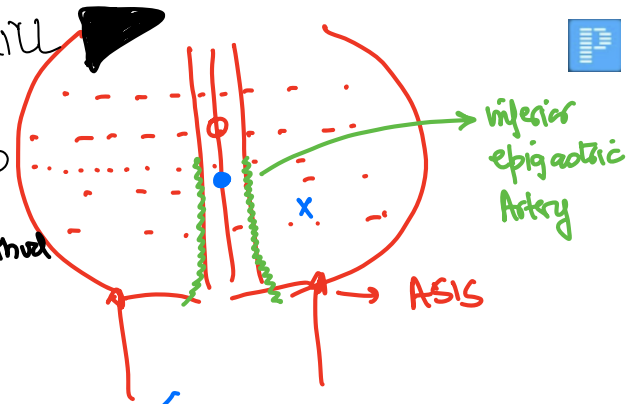
* SAAG > 1.1, Ascitic protein: < 2.5g/dl: Late budd Chiari syn

Abd. PARACENTESIS

* FLUID THRILL
* SHIFTING dullness

Site of ascitic tap in refractory ascites

1. 2cm below umbilicus
2. 2-3 cm above medial to ASIS **Z-method**



Spontaneous bacterial peritonitis

Q. A 54-year-old male with CLD and ascites presents with massive ascites. While in hospital he develops fever, abdominal pain and worsening ascites associated with shortness of breath. Which of the following is correct about clinical diagnosis of SBP?

- a. PMN cells > 50 cells/cu.mm in ascitic fluid
- b. PMN cells > 100 cells/cu.mm in ascitic fluid
- c. PMN cells > 150 cells/cu.mm in ascitic fluid
- d. PMN cells > 250 cells/cu.mm in ascitic fluid**

diagnostic paracentesis

E. coli
Trans-migration of bacteria via bowel wall

DOC for management of SBP = cefotaxime

Prevention of SBP = norfloxacin **FO**

Hepatorenal syndrome	Hepatopulmonary syndrome
Occurs due to Severe splanchnic vasodilation causing effective arterial volume causing reflex renal vasoconstriction Presents as 1. Oliguria 2. AKI	Nitric oxide-mediated intrapulmonary vascular dilatation [A-MYXOMA, HPS] SOB: ↑ in sitting position Presents as: 1. Platypnea 2. Orthodeoxia $spO_2 \downarrow$: on position change
1. Terlipressin + albumin 2. Midodrine + octreotide + Albumin 3. Definitive is OLT	Rx: O.L.T + I.P.P.V Coagulopathy* any I* encephalopathy*

OLT

1. fulm. hepatitis
2. HRS
3. HPS
4. WILSON

* HVPg > 5mm Hg

PORTAL HYPERTENSION



Portal vein > 10 mm Hg



Caput Medusae
veins



A

Spider Naevi
SVC dis
Estrogen +



B



TERRY NAIL

C



D

↓
palmar Erythema

signs of liver cell failure

MC complication of CLD

↳ PORTAL HTN

Earliest and most reliable examination finding of portal hypertension

↳ splenomegaly

Portal hypertension definition is HVPg > 5 mm Hg

HVPg > 12 mm Hg increases risk of hematemesis due to esophageal varices

↳ BRIGHT Red vomitus

Source of bleeding in esophageal varices

Portal side = left gastric vein

COFFEE-GROUND
VOMITUS

* Systemic vein = azygos vein

Best management for prevention of bleeding from esophageal varices

↳ E.V.L or Nadolol

DOC for prevention of bleeding from esophageal varices

↳ Nadolol or Propranolol

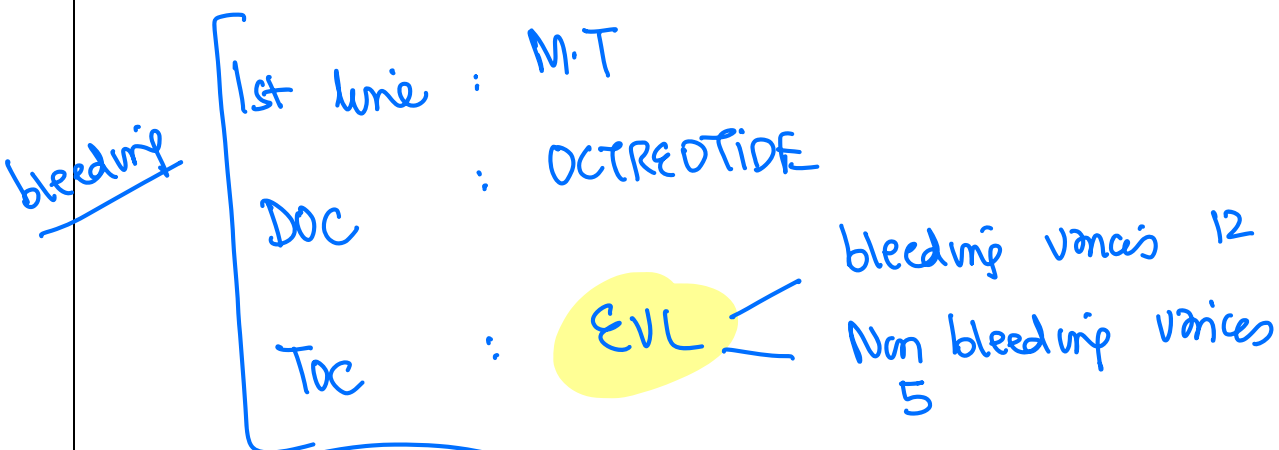
DOC of bleeding O. Varices ⇒ OCTREOTIDE
TERLIPRESSIN

Cold clammy Extremities

Management of acute variceal bleeding with BP < 90/60 mm Hg

- 1** Wide bore cannula **GRAY**
- 1:1:1 ⇒ 6 units PRBC: 6 units FFP: 6 units P.R.P
 1:1:0.25 ⇒ " " : 1 unit S.D.P
- 2** Massive transfusion **> SL of whole blood | 24 HOURS**
 (via visine warmer) **> 2.5L " " | 4 HOURS**
- 3** Permissive hypotension **~ TARGET BP = 90/60**

- 4** Octreotide / Somatostatin **DOC**
- 5** Endoscopic variceal ligation **TOC**

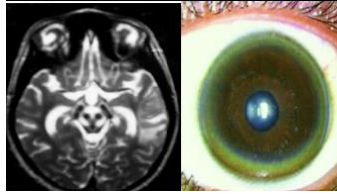


- TRIAD of death
1. HYPOTHERMIA
 2. COAGULOPATHY
 3. M. ACIDOSIS

XLR MENUE: ATP 7A gene, ch X, Boy: MR + kinky hair



Wilson disease



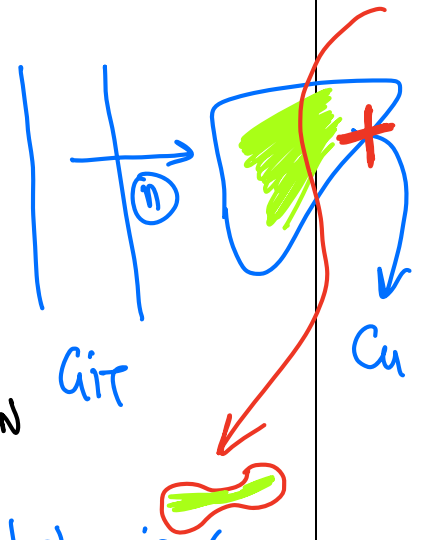
Gene ATP 7B

Chromosome 13

Inheritance AR

Defect

* DEFECTIVE Hepatobiliary xcretion of COPPER



C/F: Mnemonic- **COPPER**

- 1. **Chronic hepatitis** SPT ↑
- 2. **Ocular features** KF ring
Slit lamp: descemet memb^N
- 3. **Psychiatric features** bizzare behaviour
- 4. **PD like features** lenticular N #
dystonia, Rigidity, Bradykinesia
EPS TREMORS
- 5. **Renal tubular disorder** PCT #
amino aciduria, glucosuria renal
RTA 2

Screening [S. CERULOPLASMIN ↓
+ 24 hr urinary copper

* ^{10C} LIVER Bx c̄ estimation of
Hepatic copper content / g of dry
weight of liver

* DOC ↓
1st line drug

TRIENTENE

* CNS ⊕ = TETRATHIOMOLYBDATE

↳ MRI: GIANT face of Panda

* DOC for **maintainene** in wilson ⇒ zinc acetate
* OLT based NAZER INDEX

HBsAg ⊕ PATIENT

Additional Notes

↓
Accidental needle stick injury :

Recapping of needle
HCW

↓
chances = 30%.

Unvaccinated

Rx: HB Ig 1.m. < 24 HOURS

HCW partially vaccinated

↳ next best step:

Anti HBs TITER

< 10 IU/ml

* Single dose of
Hepatitis B
Vaccine

≥ 10 IU/ml

No need of
further
intervention

Subunit vaccine : 20µg: only one dose

primary course : 0, 1, 6 mths

Anti HBs < 10 IU/ml

Repeat course : 0, 1, 6 months

Anti HBs < 10 IU/ml

TRUE NON RESPONDER :

Two courses given

HB Ig iv x 2
dosages, 1mth
apart

if occupational exposure: then give

0.

Additional Notes

if HBV + HDV Coinfection vs superinfection

⊕ Igm Anti HBC Ag

IVDU
HBsAg +
* Igm anti HBC +
acute hep B
April 2026

IVDU
Igm anti HDV + = Coinfection
acute hep D
May 2026

accidental needle stick injury

HIV ⊕ = chances = 0.3%

Rx = TLD Tenofovir + lamivudine +
DOLUTEGAVIR

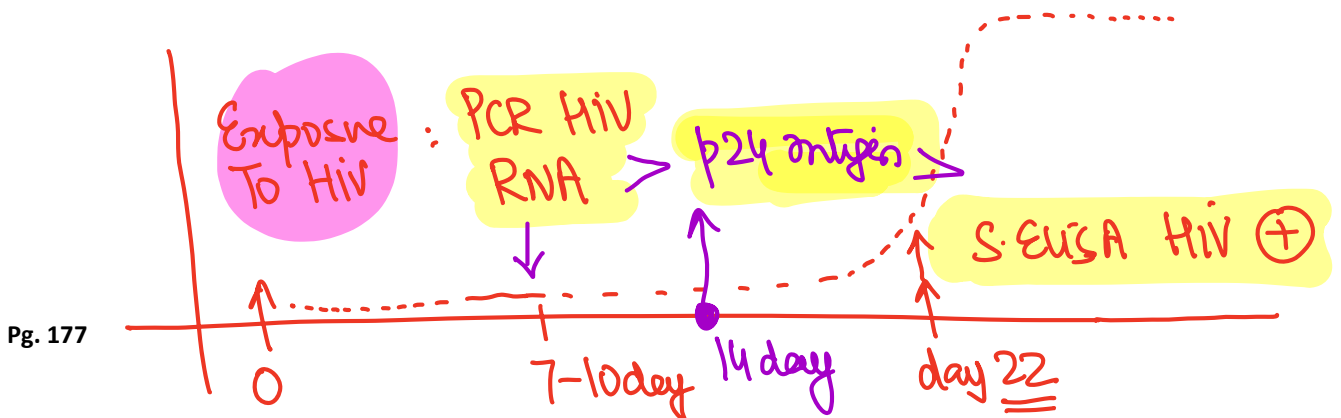
* START: < 4 HOURS

* Not effective: > 72 HOURS

* duration ⇒ 28 days

HIV = 0.3%
HCV = 3%
HBV = 30%

accidental needle
stick injury





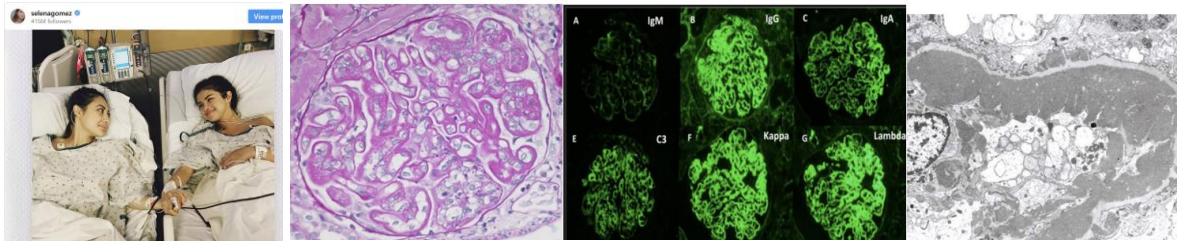
Additional Notes



Additional Notes

RHEUMATOLOGY

Systemic lupus erythematosus SLE



- Type 3 HSR
- Must have criteria for diagnosis is ANA
- MC body system involved is MUSCULO-SKELETAL invol^N
- Type of arthritis seen is non erosive polyarticular involvement
- MC Type of anemia is NCNC ANAEMIA : ACD
- MC type of anemia taken as diagnostic criteria is A-I-H-A
- Leading cause of death after decade Accelerated atherosclerosis : CV mortality ↑
- Leading cause of death in first decade of diagnosis is LUPUS NEPHRITIS

Drug induced lupus erythematosus

$$SLE = \text{♀} : \text{♂} = 6 : 1$$

- ✓ 1:1 male ratio DUE
- ✓ Renal involvement and CNS involvement is rare
- ✓ Causative drugs are

H Hydralazine
 I INH
 P Procainamide

Methyldopa, minocycline and Quinidine