بسم الله الرحمـٰن الرحيم

{وَإِذَا مَرِضْتُ فَهُوَ يَشْفِينِ}

GIS PBL Clinical | FINAL 2

Medicine :

Upper GIB , peptic ulcer disease & chronic liver disease

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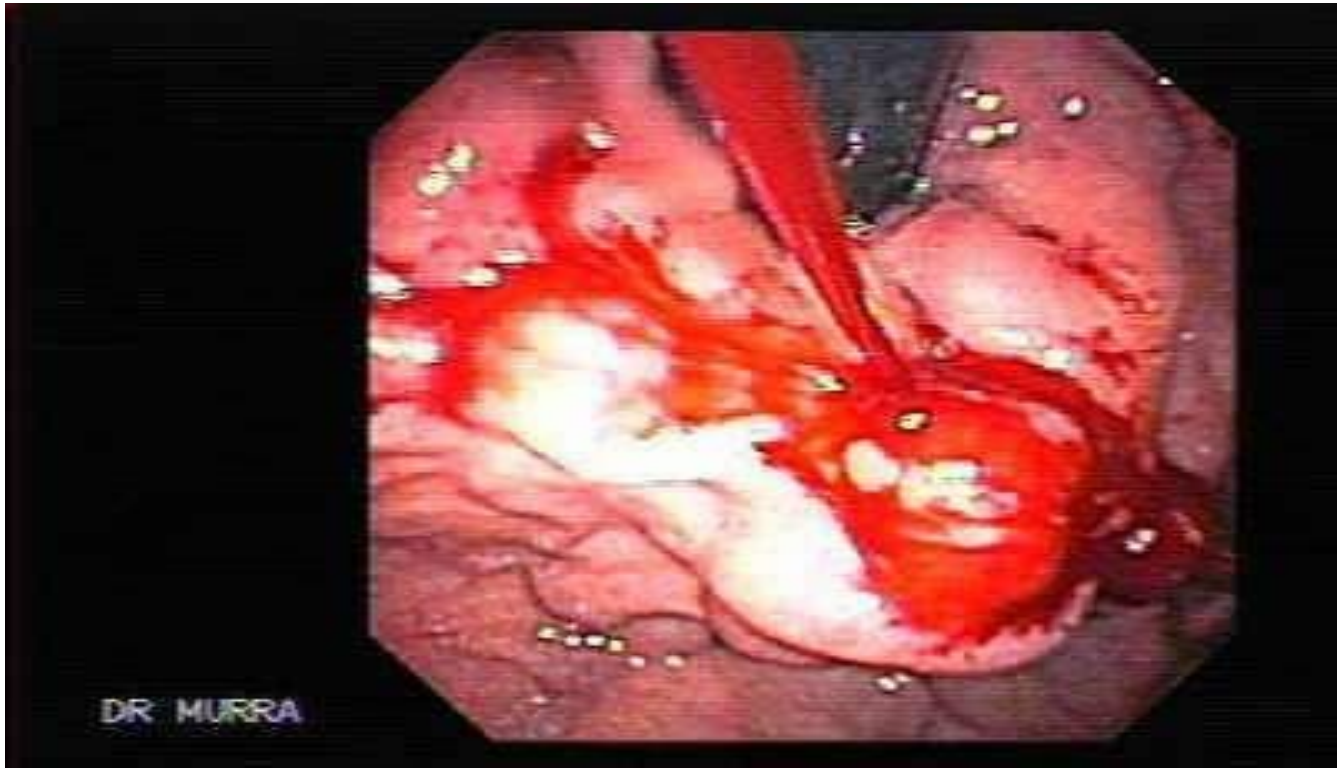
Reviewed by : NST

Upper GI bleeding



Don’t let the number of slides scare you, the lecture is easy and won’t take long insha’Allah. Good luck!

اللهم لا سهل إلا ما جعلته سهلاً

UPPER GI BLEEDING

Signs and Symptoms

- **Hematemesis**: vomiting fresh blood
- **Melena** : black , tarry , loose & shiny stool with an offensive odor.
- **Dizziness** (الدوخة / الدوار) : due to hypotension & volume loss.
- **Abd. Pain and symptoms of Peptic ulcer disease**
- **Hx of NSAID's use** (Hx means history)
- **Pallor** (شحوب) : due to anemia.
- **Hypotension**

UPPER GI BLEEDING

Signs and Symptoms

- **Orthostasis** : a condition characterized by a drop in blood pressure upon standing up from a sitting or lying position. It may progress to frank hypotension, which is a clear and clinically significant drop in blood pressure.
- **Clinical note:** In patients with upper GI bleeding, blood pressure should be checked while lying down (supine) and again after standing, because it may be normal when lying down but drop upon standing.
- **Jaundice and other stigmatas of chronic liver diseases**
- **Coffee ground vomiting**
- **Hematochezia:** may occur in massive upper GI bleeding when the blood passes through the gastrointestinal tract too quickly to be digested and converted into melena, resulting in **fresh rectal bleeding.**

UPPER GI BLEEDING

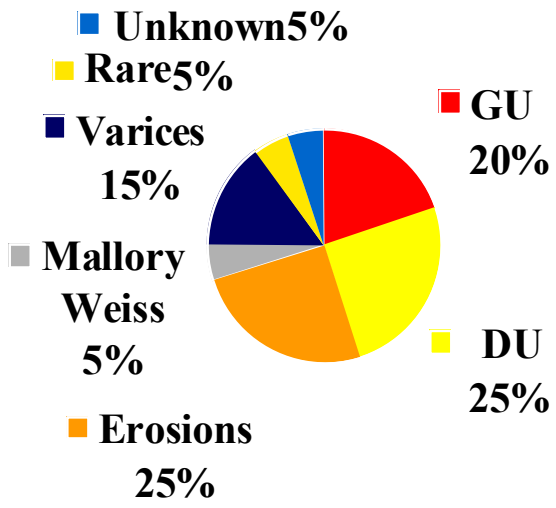
CAUSES

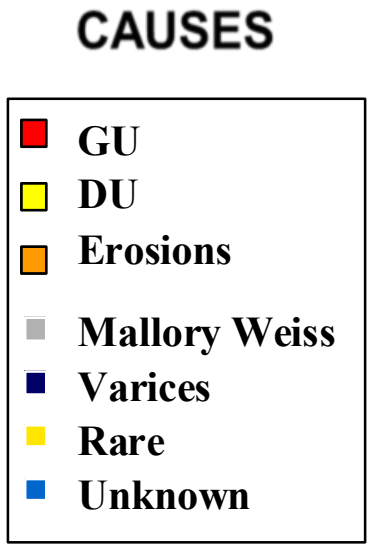
- Upper GI bleeding → bleeding above the Ligament of Treitz.

- **Causes of upper gastrointestinal (GI) bleeding:**

1. Peptic ulcer disease (PUD): The most common cause , including gastric and duodenal ulcers .
2. Esophageal varices
3. Mallory-Weiss tear:It is a longitudinal mucosal tear that occurs at the gastroesophageal junction. It usually happens after forceful or repeated vomiting
4. Erosions

- PUD and esophageal varices are the most common





Other rare causes of upper GI bleeding are listed here

RARE CAUSES

Neoplasms

AVM/Ectasia

Dieulafoy's

Stoma ulcers

Esophageal ulcers

Deodenitis

Hemobilia

Aorto-enteric fistulas

Aorto-enteric fistula: occurs in patients with aortic aneurysms, usually after surgical repair. A fistula forms between the aorta and the colon, leading to massive gastrointestinal bleeding.

UPPER GI BLEEDING

Peptic Ulcer Disease

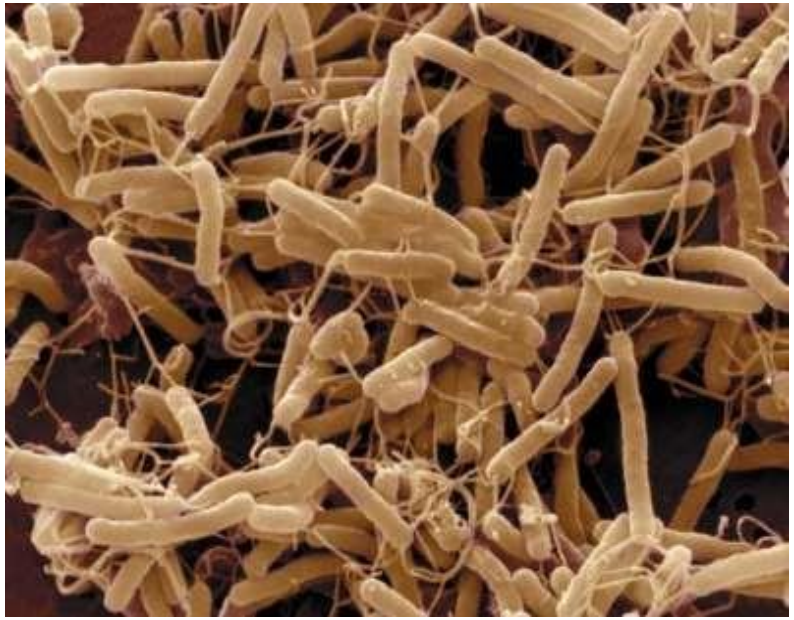
- Defect in the GI mucosa extending through the muscularis mucosa.
- Decreasing incidence.
- Caused by imbalance between the aggressive and defensive factors.
- There are two common causes of peptic ulcer disease (PUD):

1. Helicobacter pylori infection.
2. (NSAIDs) use.

UPPER GI BLEEDING

Peptic Ulcer Disease

- **Helicobacter Pylori**
- **NSAID's**
- **Acid Hypersecretory state.**
- **Antral G cell Hyperplasia**



UPPER GI BLEEDING

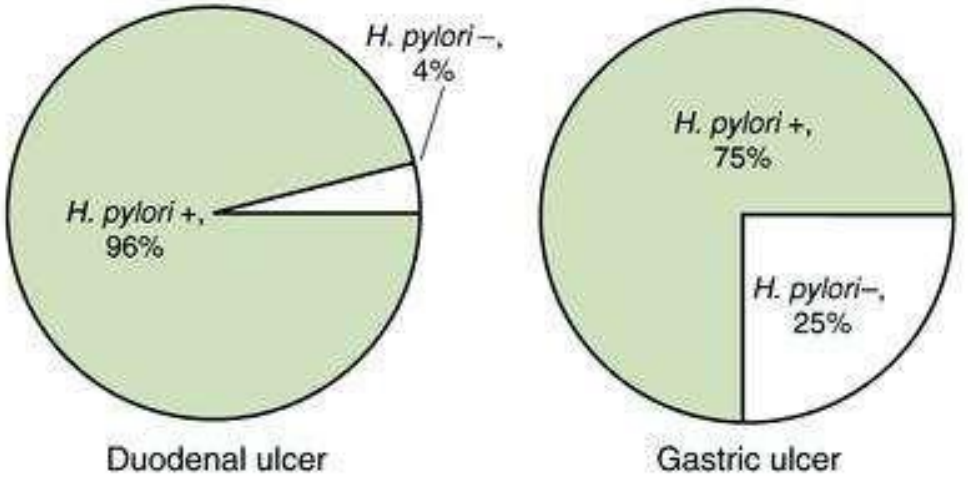
Peptic Ulcer Disease

Association between H. pylori and peptic ulcers (duodenal & gastric ulcers):

- About **96% of duodenal ulcers** are associated with *Helicobacter pylori* infection.
- In comparison, only about **75% of gastric ulcers** are associated with *H. pylori*.

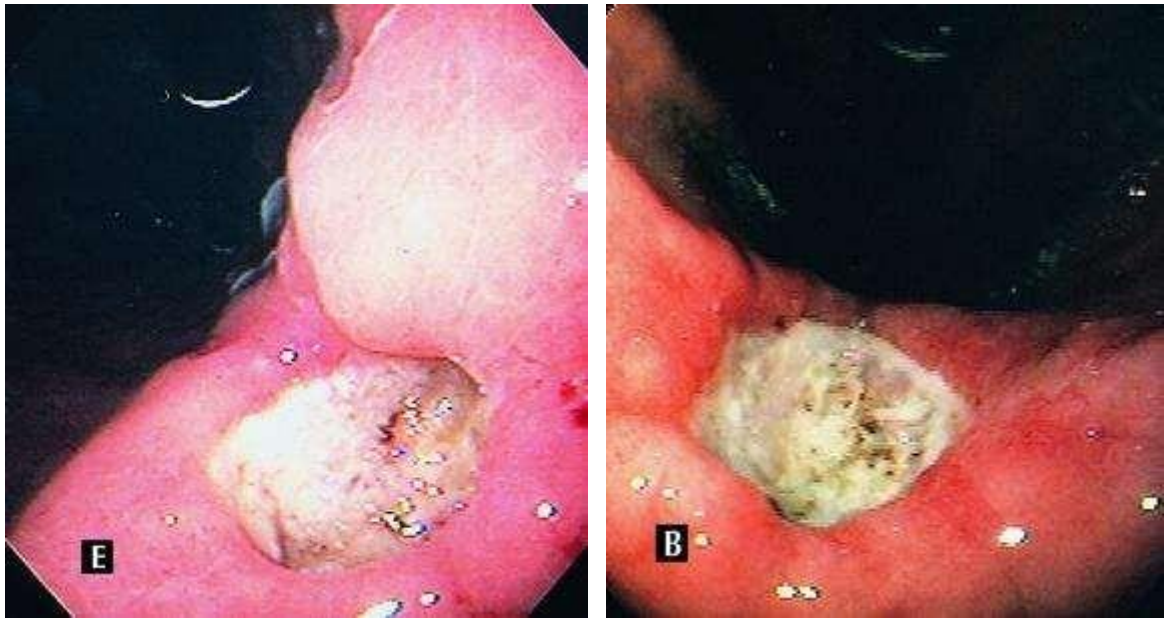
Because the association is very strong in duodenal ulcers, patients were sometimes treated for *H. pylori* infection **even without testing for infection first.**

However, in gastric ulcers, testing for *H. pylori* is important because around **25% of cases are not related to the infection**. Other causes may include: NSAID use , malignancy , etc.



UPPER GI BLEEDING

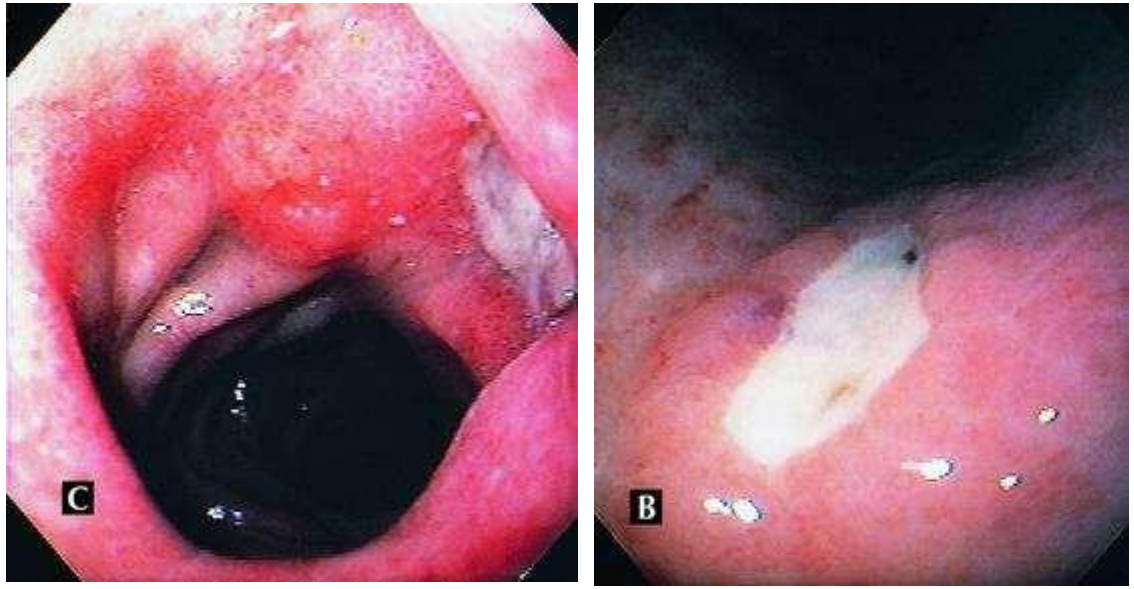
Gastric Ulcers



This is a gastric ulcer. It appears as a defect in the normal gastric mucosa with a clean ulcer base, therefore it is described as a **clean-based ulcer**.

UPPER GI BLEEDING

Duodenal Ulcers

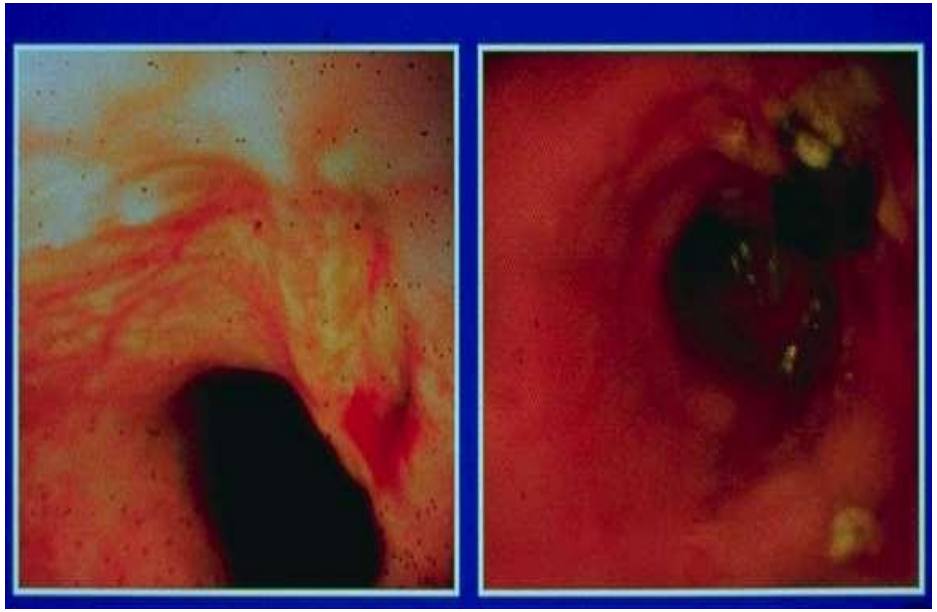


This is a duodenal ulcer. It appears as a mucosal defect surrounded by inflammatory changes and edema around the ulcer.

UPPER GI BLEEDING

Mallory - Weiss

- **Mallory-Weiss tear:** is a mucosal laceration that occurs at the gastroesophageal junction that occurs after **repeated or forceful vomiting**. It is commonly seen in alcoholic patients and pregnant women with hyperemesis gravidarum (severe and excessive vomiting during early pregnancy, especially in the first trimester). The bleeding **is usually mild and self-limiting** rather than massive.

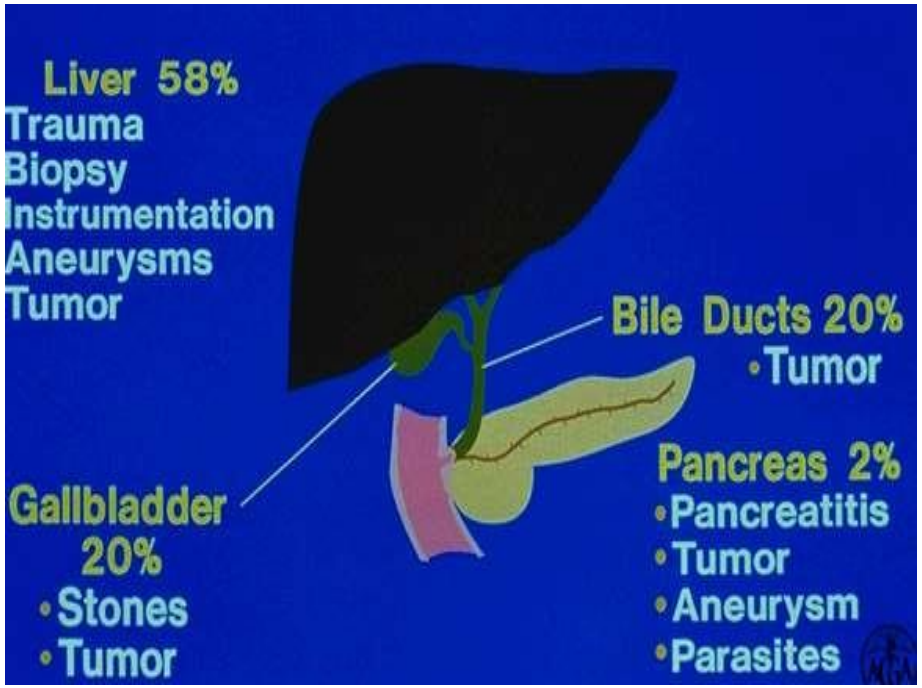


Laceration around the GE junction
Classical presentation as bleeding after episode of vomiting
Classical presentation found in 50% only
Self- limiting

UPPER GI BLEEDING

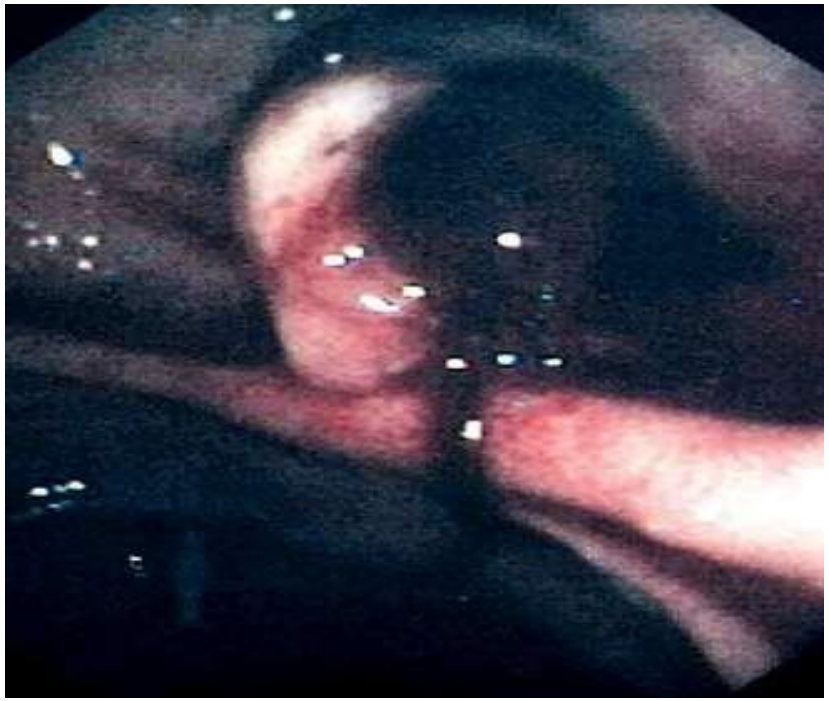
Hemobilia

- **Hemobilia:** is bleeding that occurs inside the bile ducts (biliary system), so blood mixes with bile. It may happen due to problems in the liver, bile ducts, pancreas, or nearby organs, such as tumors (like cholangiocarcinoma or biliary cancer) or gallstones.



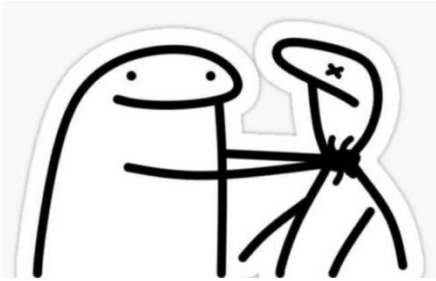
UPPER GI BLEEDING

Hemobilia



Recall: The Ampulla of Vater is the opening in the 2nd part duodenum where the bile duct and pancreatic duct join and release their secretions into the intestine.

and if you do forget it:



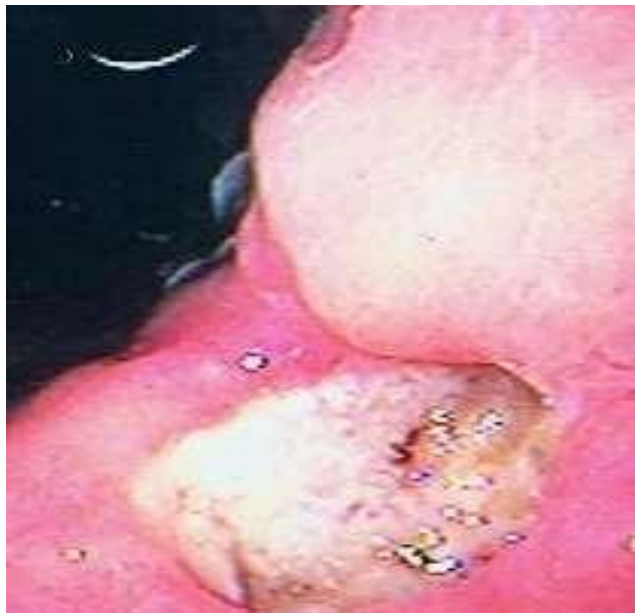
Dr. Al-Muhtaseb You

- This is the ampulla of Vater and the blood is coming out of it, which is called hemobilia.
- In hemobilia, blood may drain through the biliary system and appear at the ampulla of Vater, where bile enters the duodenum.

UPPER GI BLEEDING

stress ulcers

- Caused by Vagal hyperstimulation (such as head injury or burns) and vascular hypoperfusion.
- It's not related to H.pylori infection or NSAIDs use.
- Body and fundus more affected unlike H. pylori-related peptic ulcers, which most commonly occur in the antrum.
- Multiple
- Prophylaxis is indicated in critically ill ICU patients
- Stress ulcers are caused by severe physiological stress (e.g., critical illness, trauma, burns), not psychological stress.
- **Types of stress ulcers:**



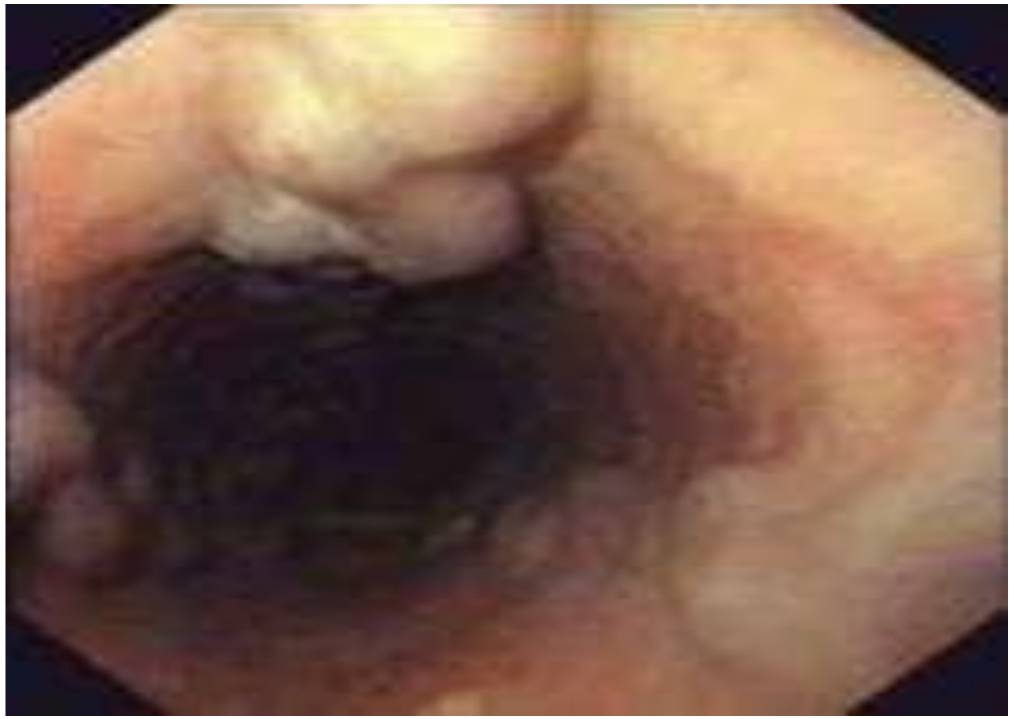
a. Curling Extensive burn :occur after severe burns.

b.Cushing Head Injury :associated with CNS injury (e.g., head trauma or brain tumors)

UPPER GI BLEEDING

BLEEDING ESOPHAGEAL VARICEAL

- Dilated tortuous veins of the lower and mid esophagus.
- Secondary to portal HTN (It occurs in patients with portal hypertension)
- 30% mortality after the first episode (It is considered a very high mortality rate)
- 60% Rebleeding rate

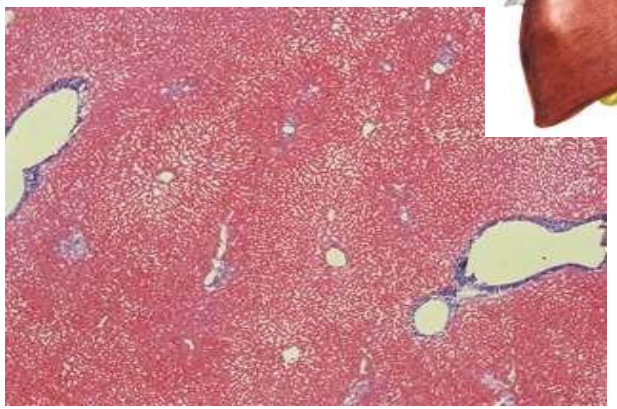


This is an upper endoscopy image showing dilated, tortuous veins in the distal esophagus, indicative of esophageal varices.

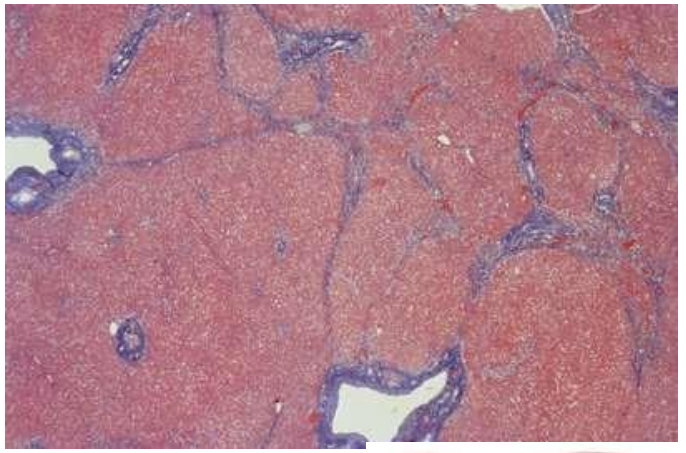
Cirrhosis and Portal hypertension

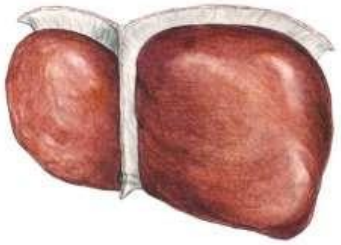
اللهم صلِّ وسلم وبارك على سيدنا محمد

Healthy Liver



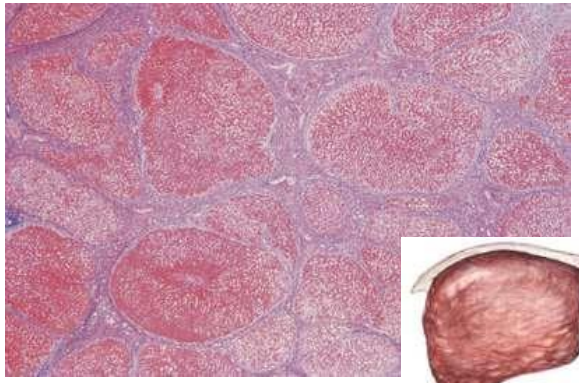
Liver Fibrosis

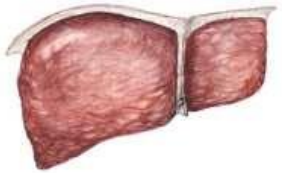




Cirrhosis

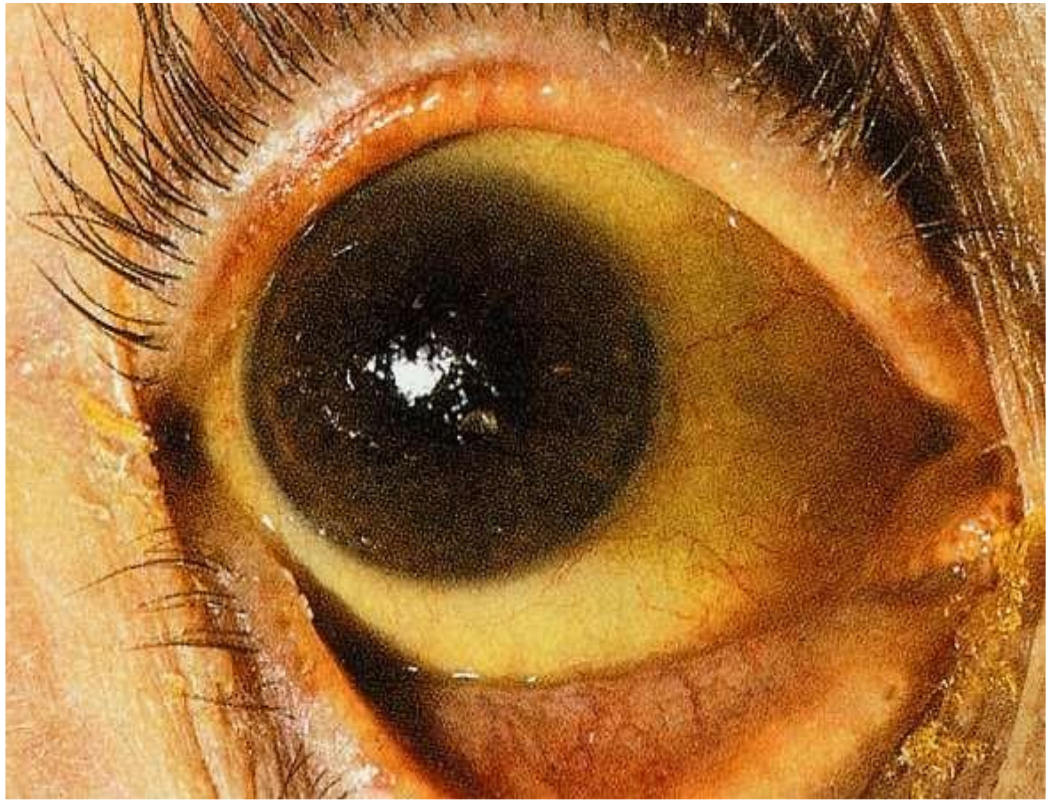
Nodular liver

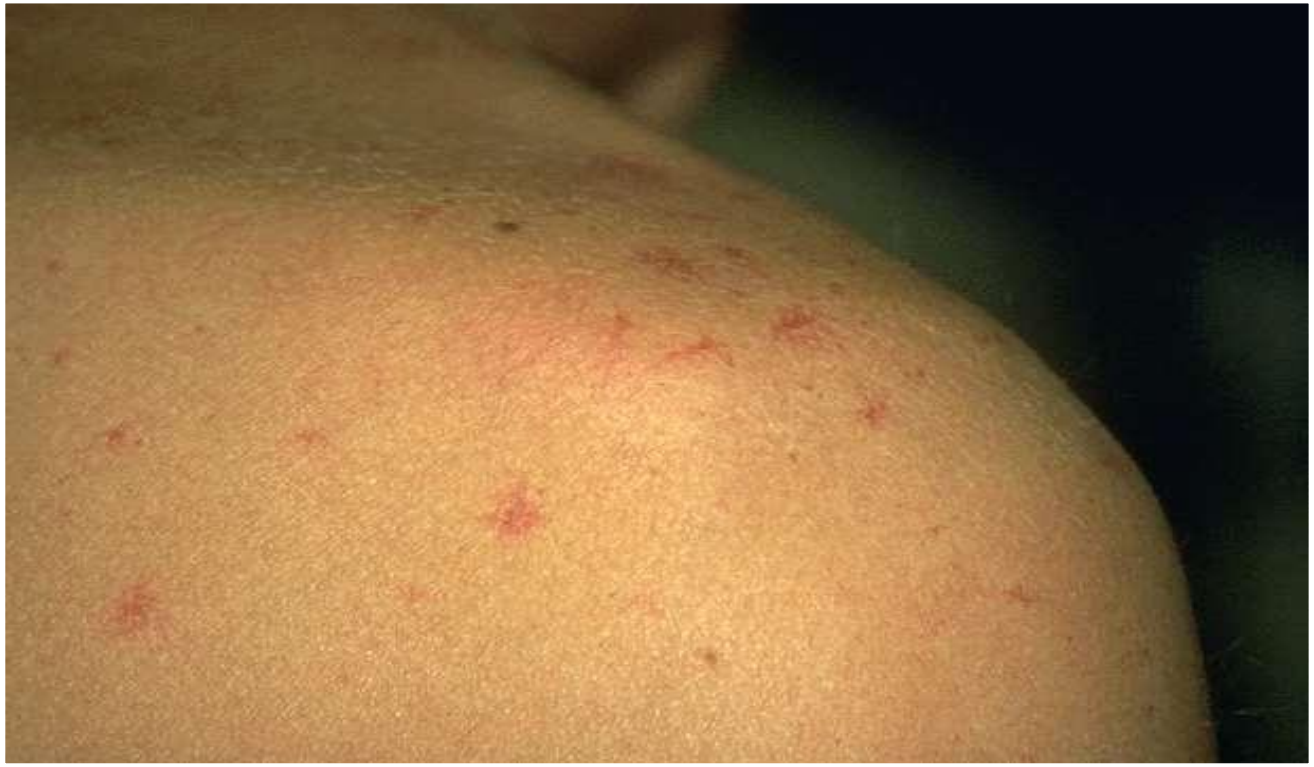




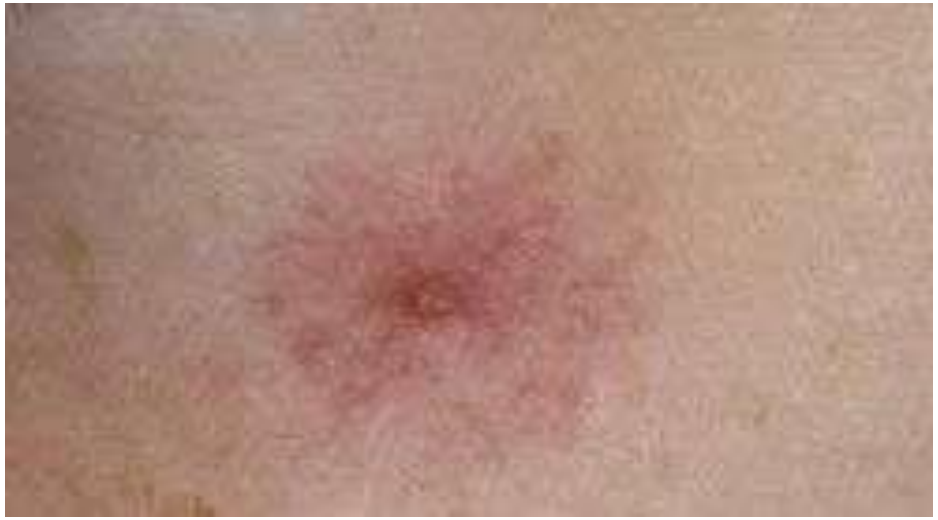
Jaundice

- One of the common manifestations of liver cirrhosis.
- Accumulation of bilirubin in the blood stream causing yellowish discoloration of plasma and heavily perfused tissues
- Jaundice can be clinically detected when bilirubin levels rise **above about 2.5–3 mg/dL.**
- It appears as yellow discoloration of the sclera and mucous membranes.





These are spider angiomas. They commonly appear on the anterior & posterior parts of the chest as small spider-like dilated blood vessels. They occur due to hyperestrogenemia.
Hyperestrogenemia = elevated estrogen levels in the blood.



Spider Angiomas

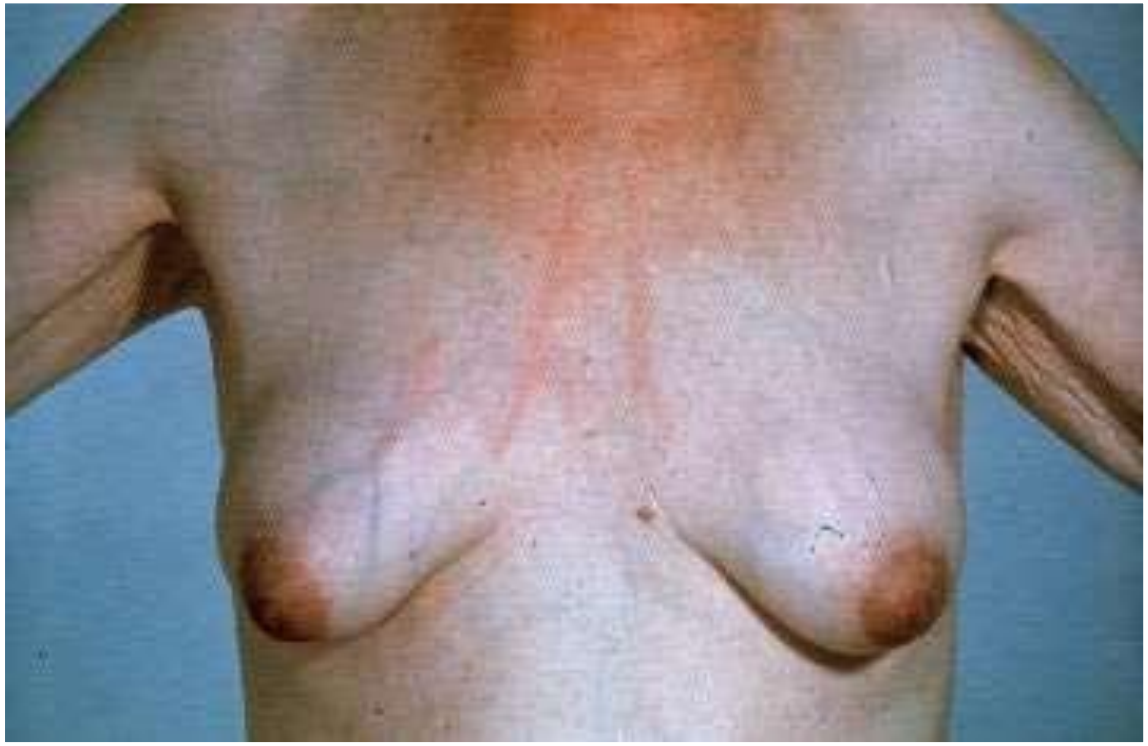
Small, centrally raised bumps (papules) caused by a dilated arteriole (small artery). A network of dilated capillaries (tiny blood vessels) radiate from the arteriole. Pressing on the lesion causes the redness to disappear briefly, and there is a rapid return of redness once the pressure is lifted.

Finger Clubbing

- Common manifestation of liver cirrhosis, but it can also occur in other conditions such as inflammatory bowel disease and cyanotic congenital heart disease.
- a condition where there is enlargement of the terminal end of the digit over the distal phalanx.
- It is usually symmetrical and affects the fingers
- Early change: loss of the normal angle between the nail and nail bed.
- Progressive change: increased curvature of the nail over time.
- In some cases, it may be familial.

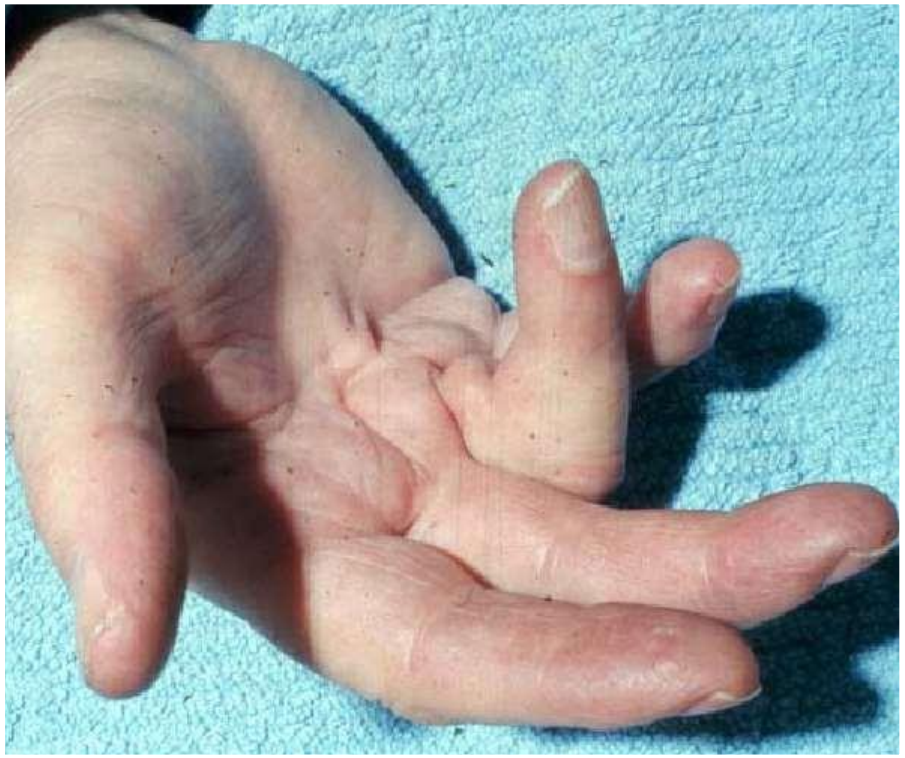
Gynecomastia

- It occurs due to liver cirrhosis
- Breast development in men
- In female patients with liver cirrhosis, breast atrophy may occur due to hormonal imbalance, especially reduced estrogen metabolism and altered sex hormone levels.



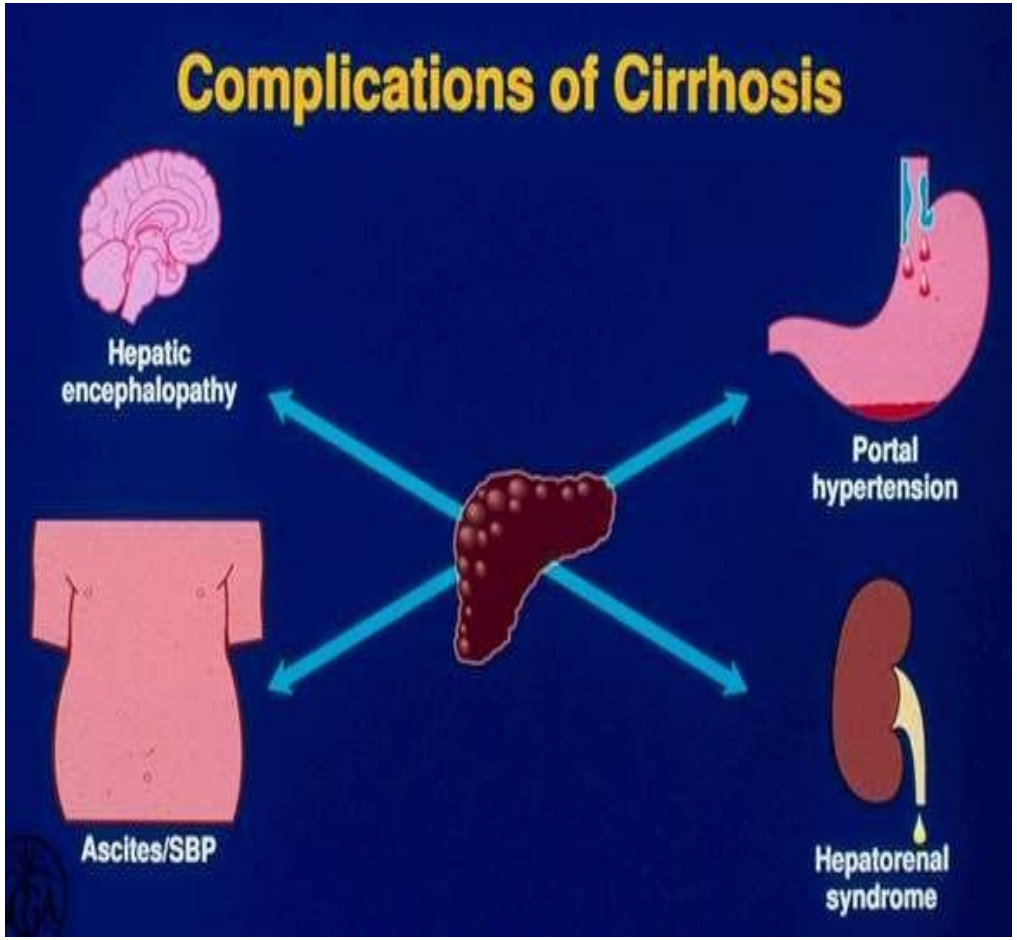
Dupuytren's Contractures

- Joint contractures
- It also occurs in patients with liver cirrhosis



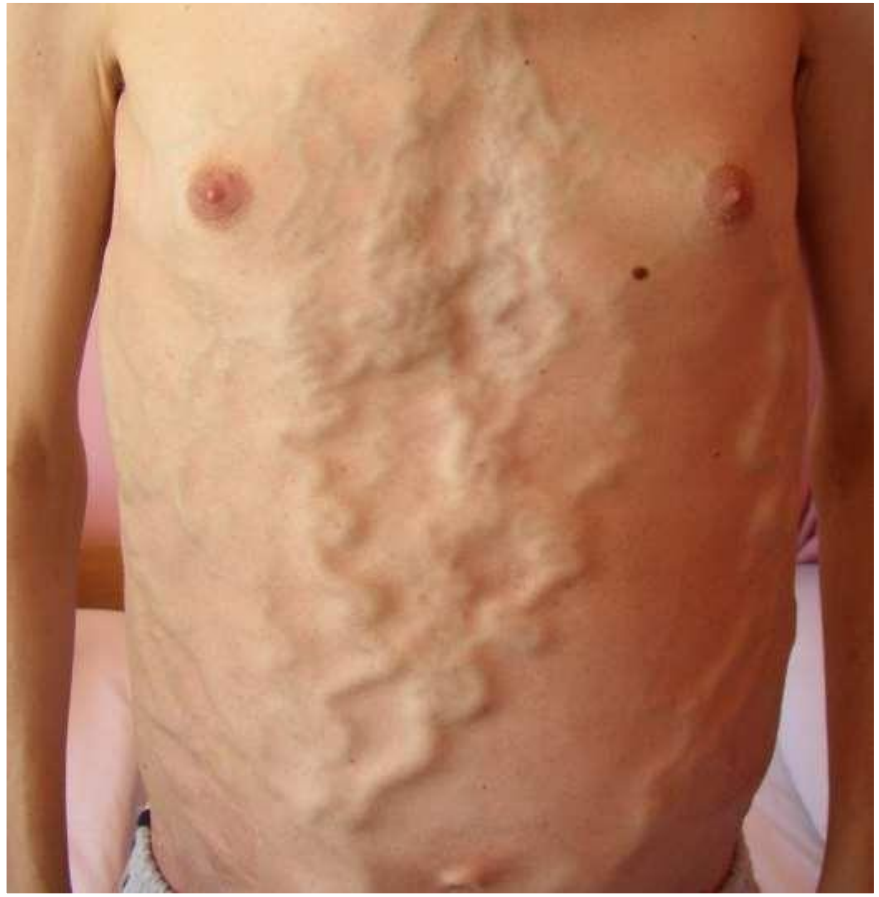
Common complications of liver cirrhosis and portal hypertension:

1. Variceal bleeding, which is a life-threatening complication with high mortality.
2. Hepatorenal syndrome, which is acute kidney injury in patients with advanced liver disease.
3. Ascites with spontaneous bacterial peritonitis.
4. Hepatic encephalopathy, caused by increased ammonia levels, which leads to decreased level of consciousness in severe cases.

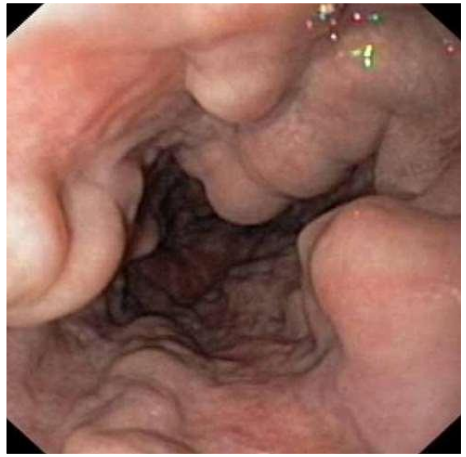


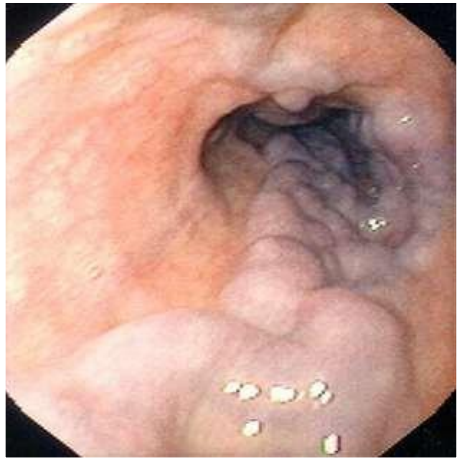
Caput Medusae

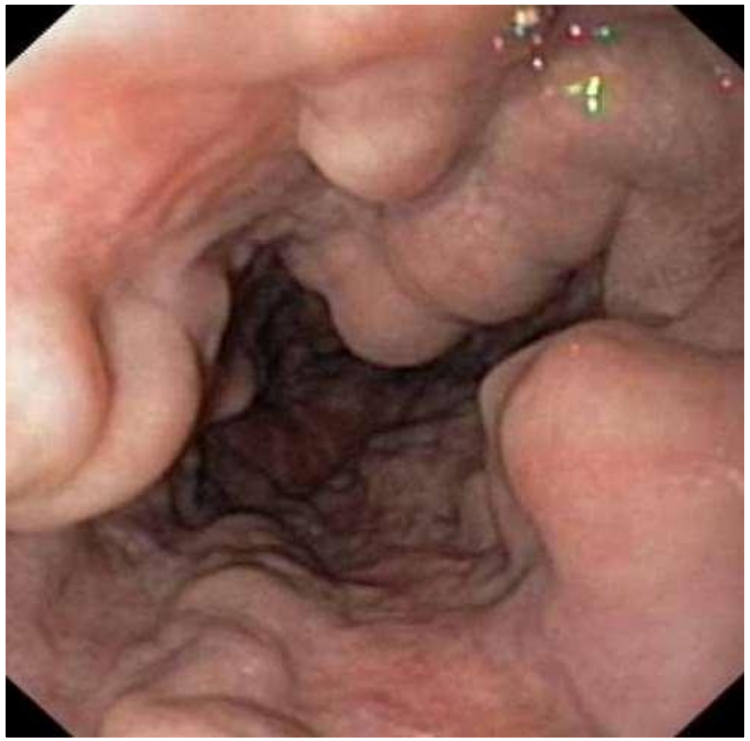
- Another manifestation of liver cirrhosis
- Distended and engorged umbilical veins which are seen radiating from the umbilicus across the abdomen to join systemic veins.
- They are dilated, engorged umbilical veins seen on the anterior abdominal wall.

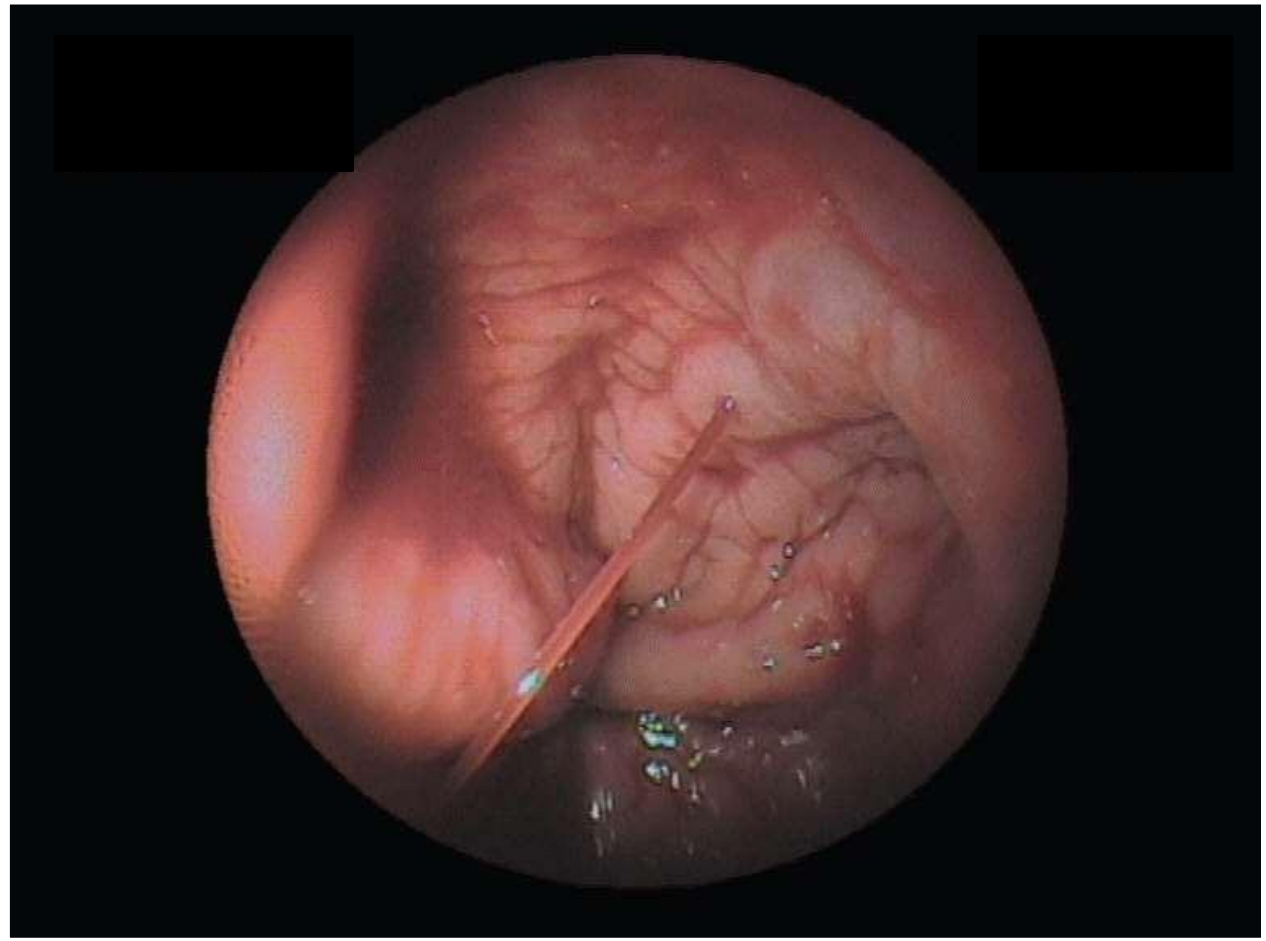


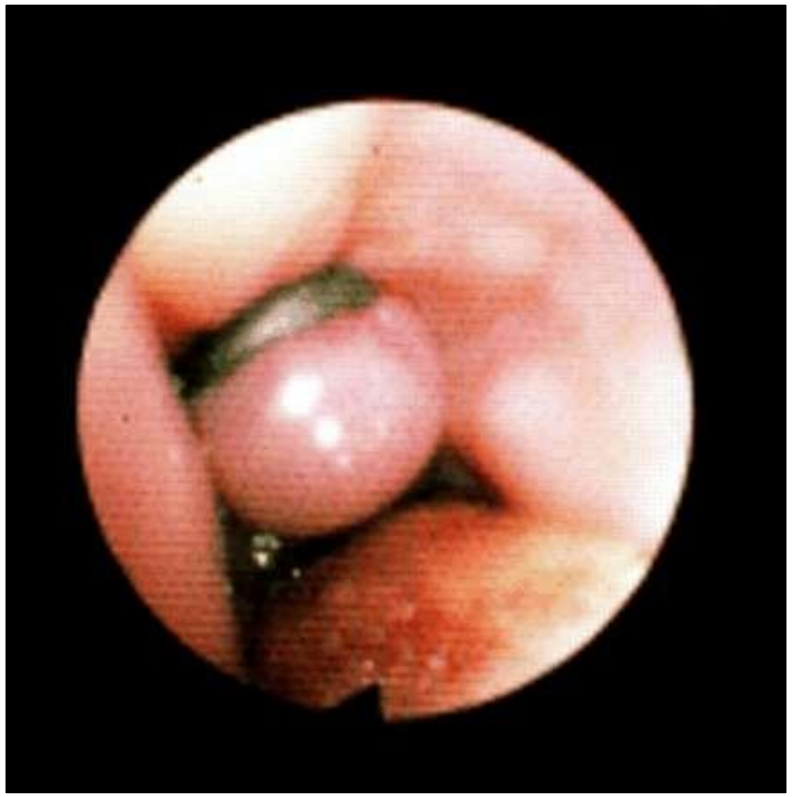
Esophageal Varices



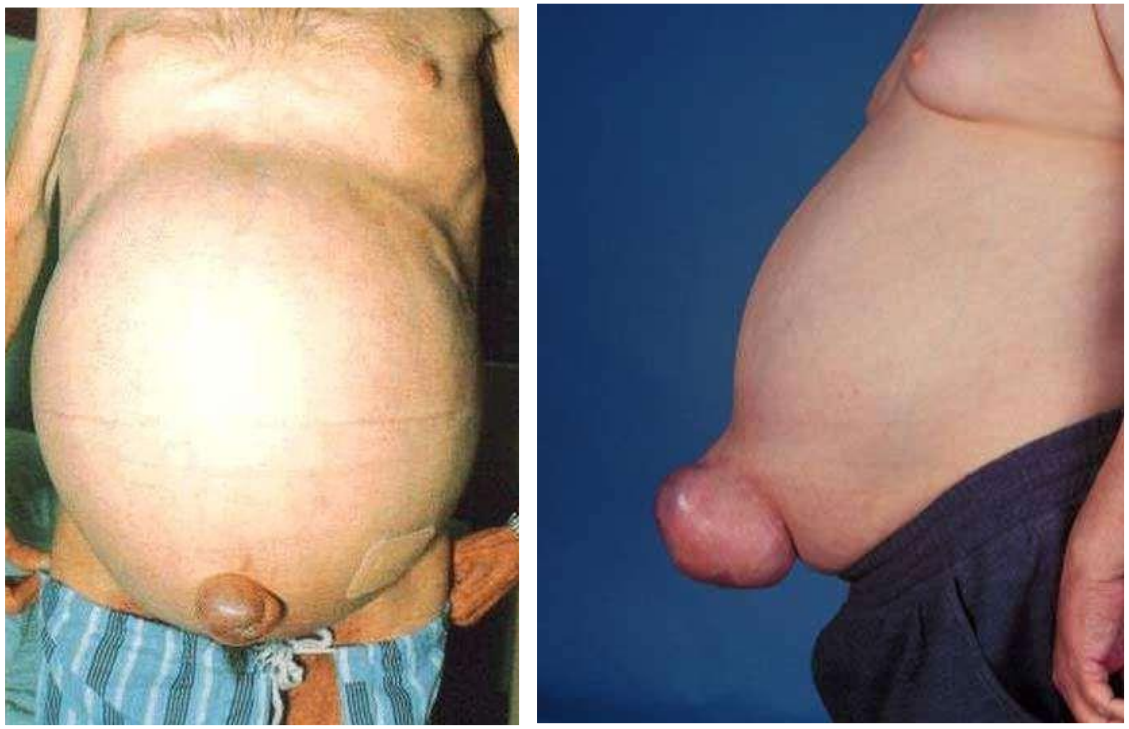








This is **esophageal band ligation**, a procedure performed during endoscopy as a treatment option for patients with bleeding esophageal varices.

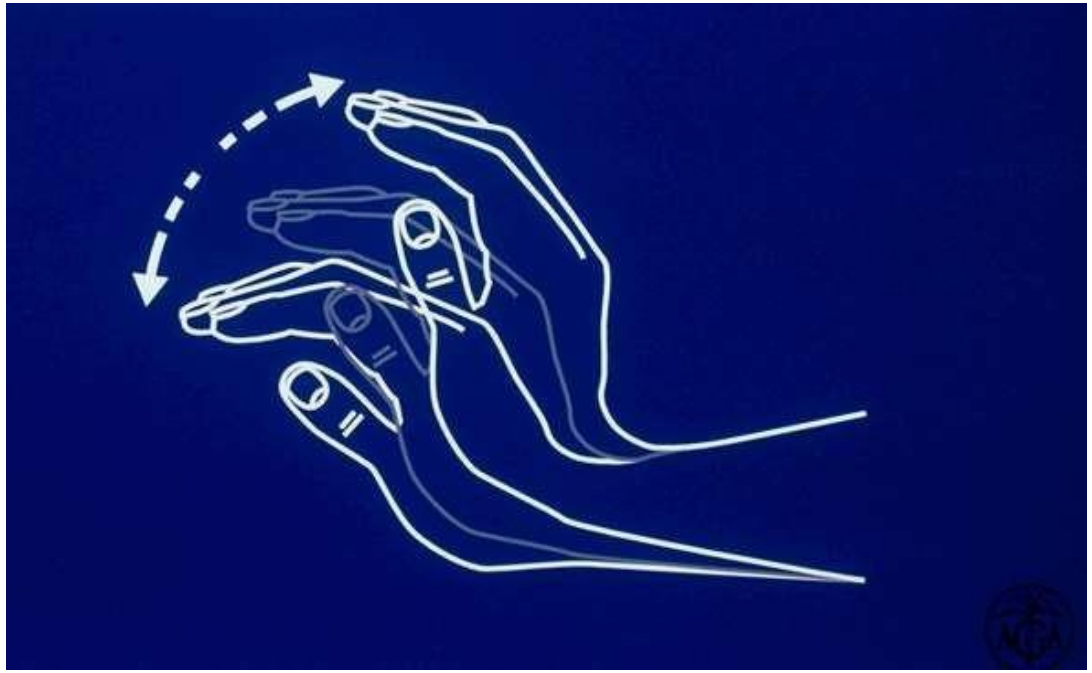


This patient has massive ascites and also an umbilical hernia.

Ascites is the accumulation of excess fluid in the peritoneal (abdominal) cavity, most commonly due to liver cirrhosis and portal hypertension.

Astraxia

- It occurs in patients with hepatic encephalopathy , heart failure & respiratory failure.
- Flapping tremors, quick arrythmic movement in back ground tonic muscle contracion



Click on **asterixis** to see how it appears clinically.

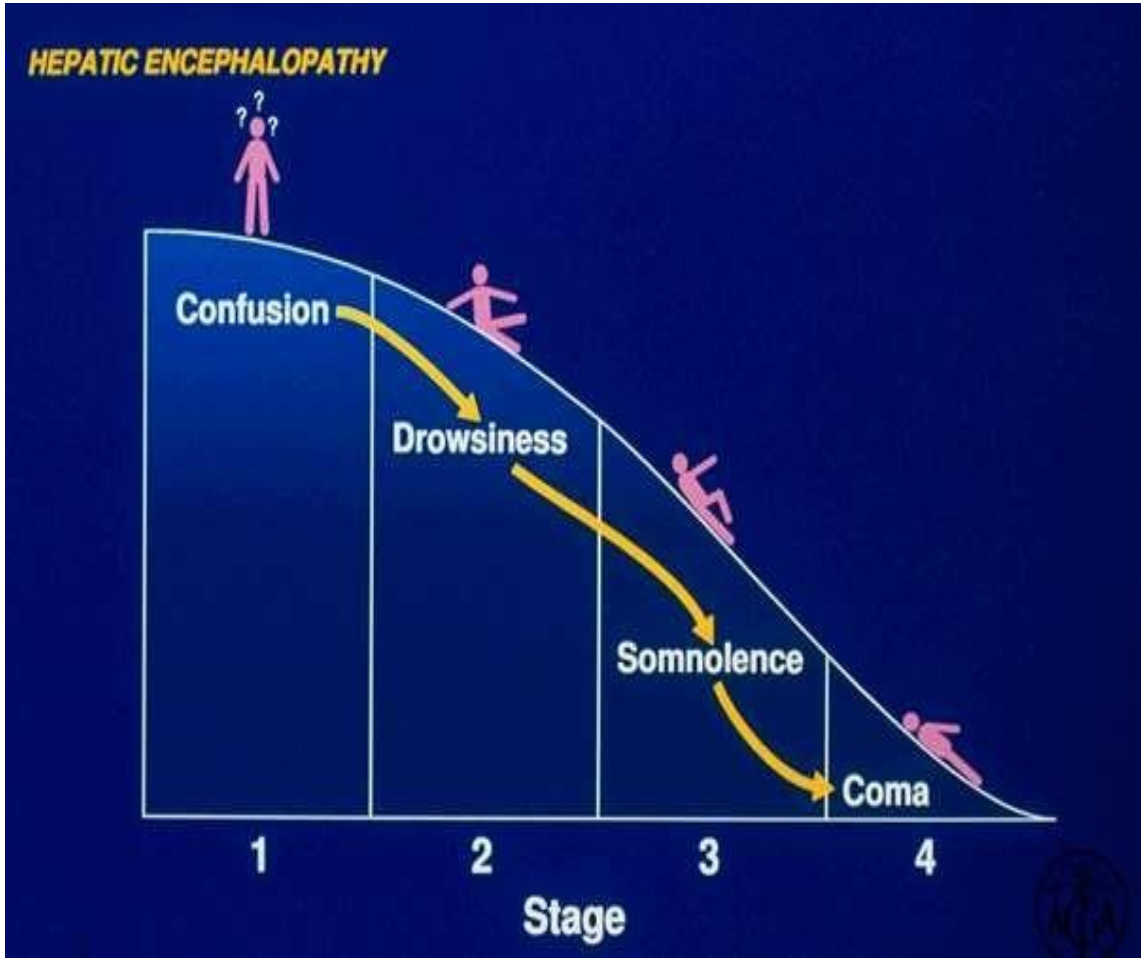
Stages of hepatic encephalopathy (West Haven staging system) :

Stage 1: Mild confusion with reversal of sleep-wake cycle.

Stage 2: Drowsiness and disorientation.

Stage 3: Marked somnolence; patient is sleepy most of the time but still arousable.

Stage 4: Coma.



Hepatitis A-E Viruses

- An Overview

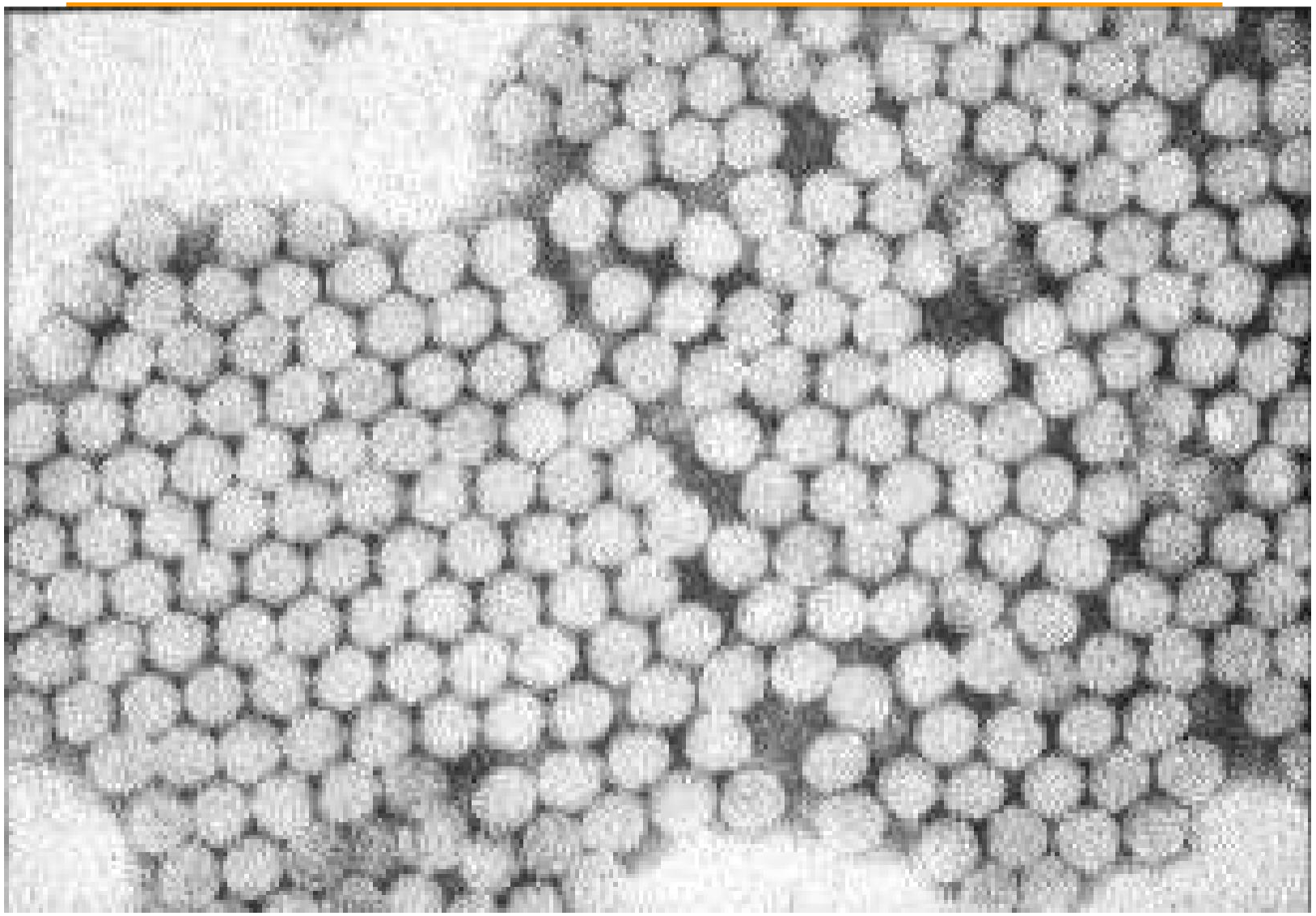
Take a tea break, then continue;)

Summary table

Type of Hepatitis

	A	B	C	D	E
Source of virus	feces	blood/ blood-derived body fluids	blood/ blood-derived body fluids	blood/ blood-derived body fluids	feces
Route of transmission	fecal-oral	percutaneous permucosal	percutaneous permucosal	percutaneous permucosal	fecal-oral
Chronic infection	no	yes	yes	yes	no
Prevention	pre/post-exposure immunization	pre/post-exposure immunization	blood donor screening; risk behavior modification	pre/post-exposure immunization; risk behavior modification	ensure safe drinking water

Hepatitis A Virus



Hepatitis A - Clinical Features

- Incubation period: Average 30 days
  Range 15-50 days

- Jaundice by age group:

<6 yrs, <10%
6-14 yrs, 40%-50%
>14 yrs, 70%-80%

In the pediatric age group (<6 year olds), the infection is usually mild and may resemble a nonspecific viral illness with fever and slight jaundice that often goes unnoticed. Adult patients (>14 year olds), however, commonly present with jaundice, abdominal pain, and vomiting.

- Complications:

Fulminant hepatitis
Cholestatic hepatitis
Relapsing hepatitis

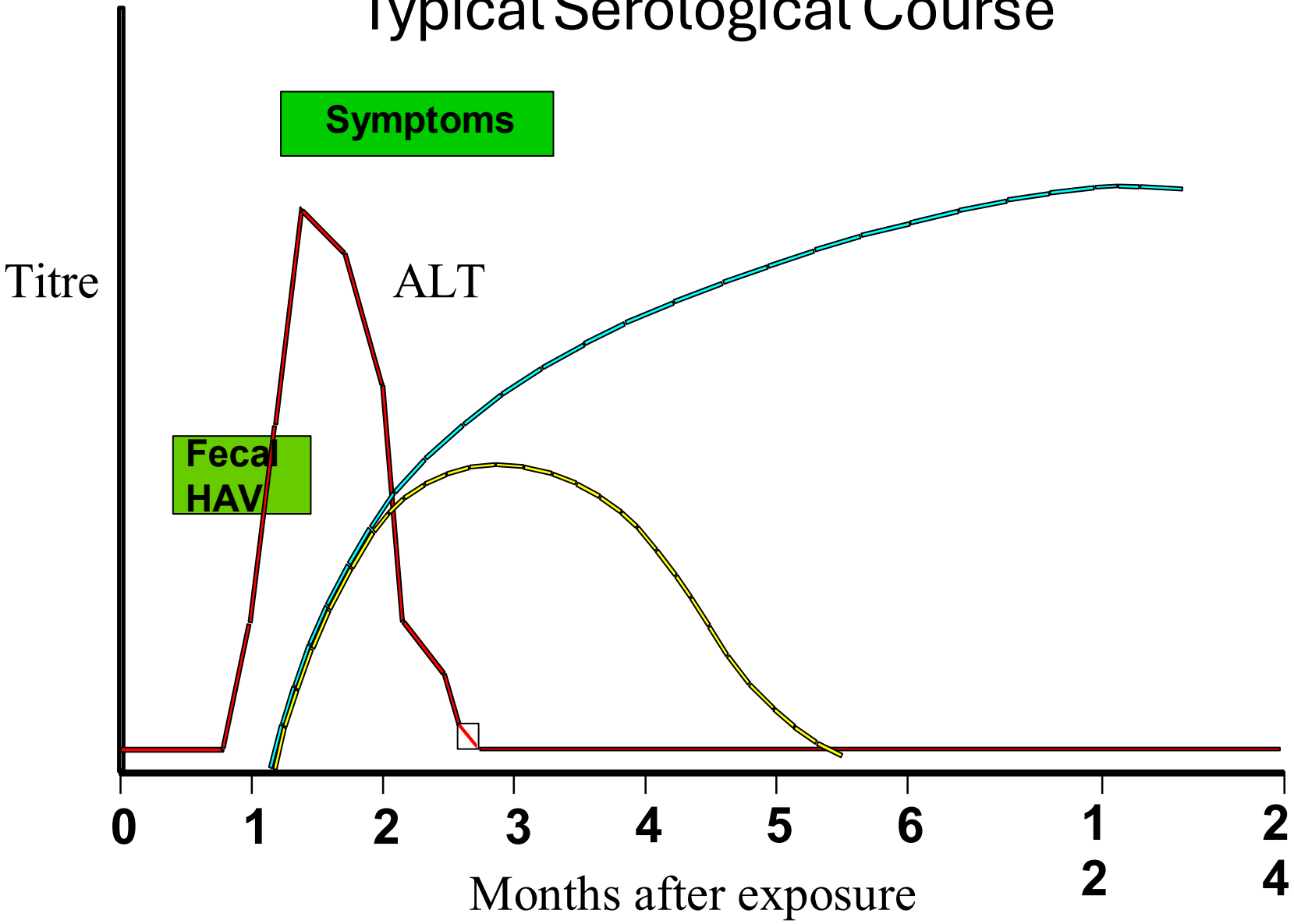
Rare, fulminant hepatitis occurs in fewer than 1% of cases, patients may develop acute liver failure.

- Chronic sequelae: None

In more than 99% of patients, the infection resolves completely with full recovery.
In a very small proportion of cases, fulminant hepatitis may occur, potentially leading to death or the need for liver transplantation

Hepatitis A Infection

Typical Serological Course



Hepatitis A Virus Transmission

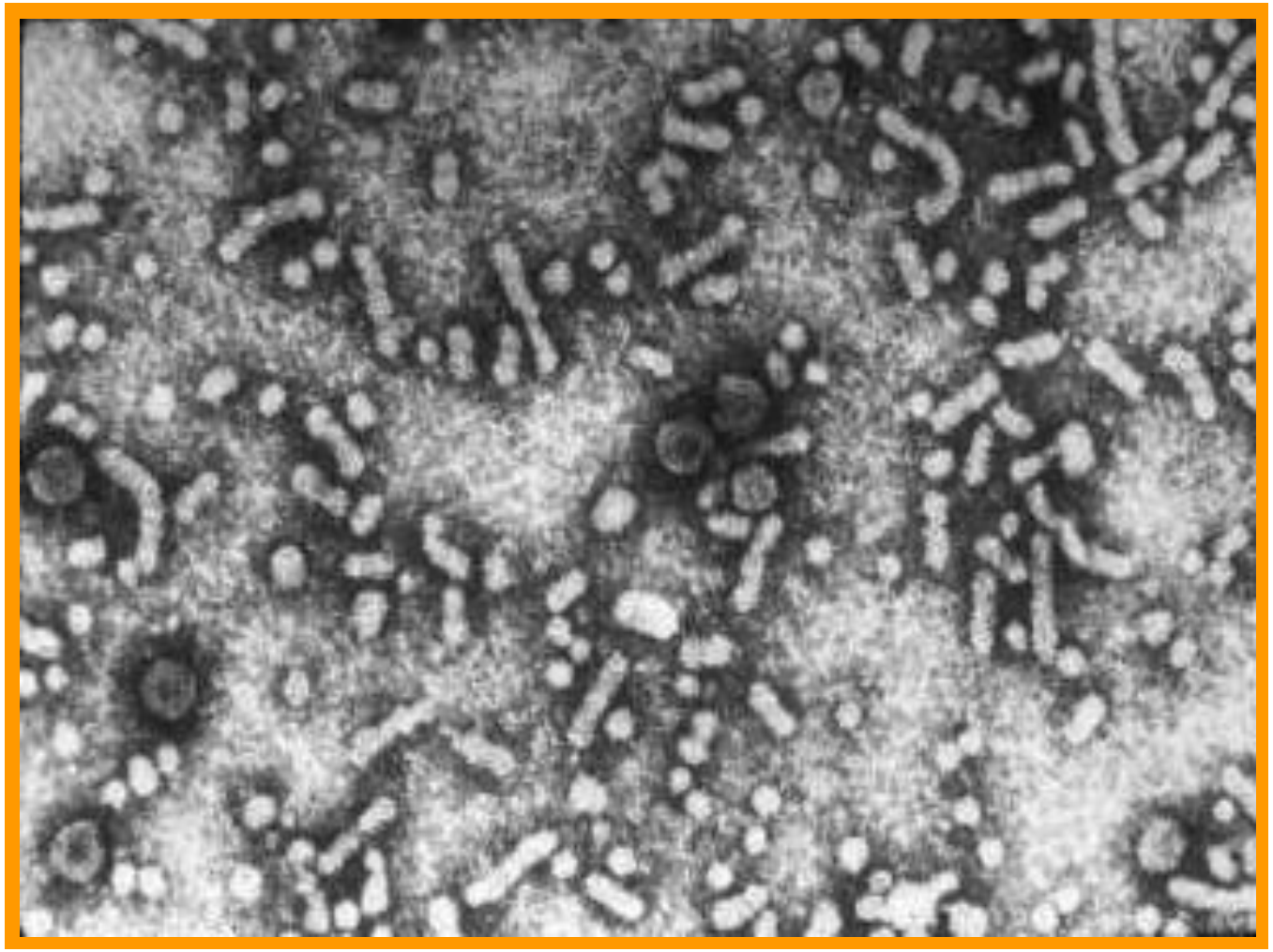
Hepatitis A virus infection is transmitted through the fecal-oral route, where the virus is shed in the feces of infected patients.

- **Close personal contact**
  (e.g., household contact, sex contact, child day care centers)
- **Contaminated food, water**
  (e.g., infected food handlers, raw shellfish)
- **Blood exposure (rare)**
  (e.g., injecting drug use, transfusion)

Laboratory Diagnosis

- **Acute or recent infection is diagnosed by the detection of HAV-IgM in serum by EIA.**
- **Past Infection i.e. immunity is determined by the detection of HAV-IgG by EIA.**

Hepatitis B Virus



Hepatitis B - Clinical Features

Incubation period:	Average 60-90 days Range 45-180 days
Clinical illness (jaundice):	<5 yrs, <10% show clinical illness >5 yrs, 30%-50%
Acute case-fatality rate:	0.5%-1%
Chronic infection:	<5 yrs, 30%-90% >5 yrs, 2%-10%
Premature mortality from chronic liver disease:	15%-25%

Clinical Manifestations and Chronicity

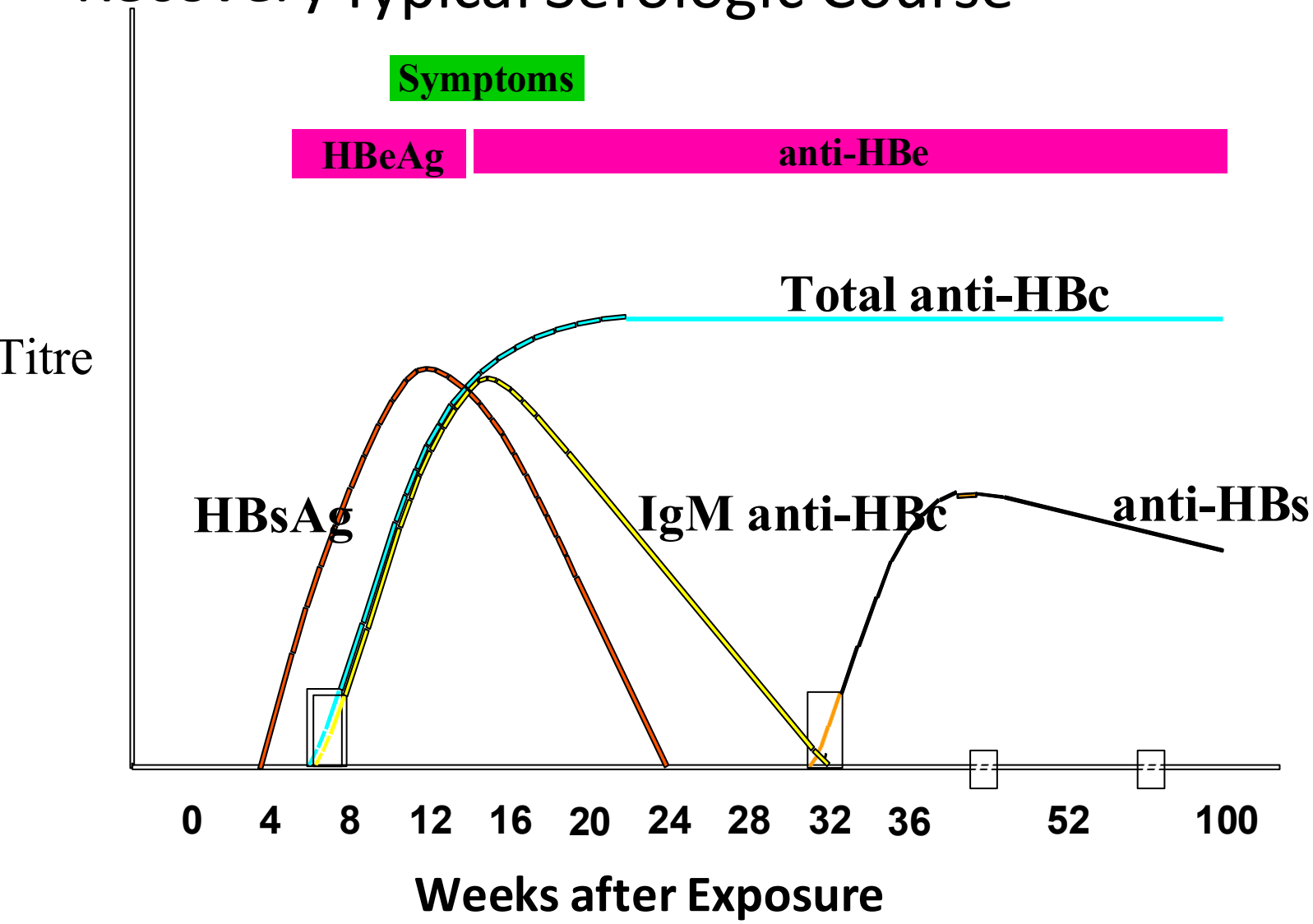
Clinical illness is much more common in adults once they acquire the virus. However, the major concern in the pediatric age group is the high risk of chronic infection. Most adults develop acute clinical manifestations such as jaundice, abdominal pain, and other signs of acute hepatitis B infection, but they are usually able to clear the virus and recover. In contrast, more than 90% of infected young children develop chronic infection.

Hepatitis B virus can be transmitted vertically from mother to baby during delivery in infected pregnant females. If no preventive measures were taken, a baby born to a highly infected mother will most likely acquire the infection and later develop chronic hepatitis B infection. This is thought to occur because the infant's immune system is not mature enough to recognize the virus effectively. As a result, the virus coexists silently within the hepatocytes for many years without causing an immune response. During childhood, the virus replicates extensively in the liver without significant hepatic inflammation. Later in life, often during the patient's 20s or 30s, the immune system may eventually recognize the virus as foreign, leading to the acute inflammatory phases of hepatitis.

Spectrum of Chronic Hepatitis B Diseases

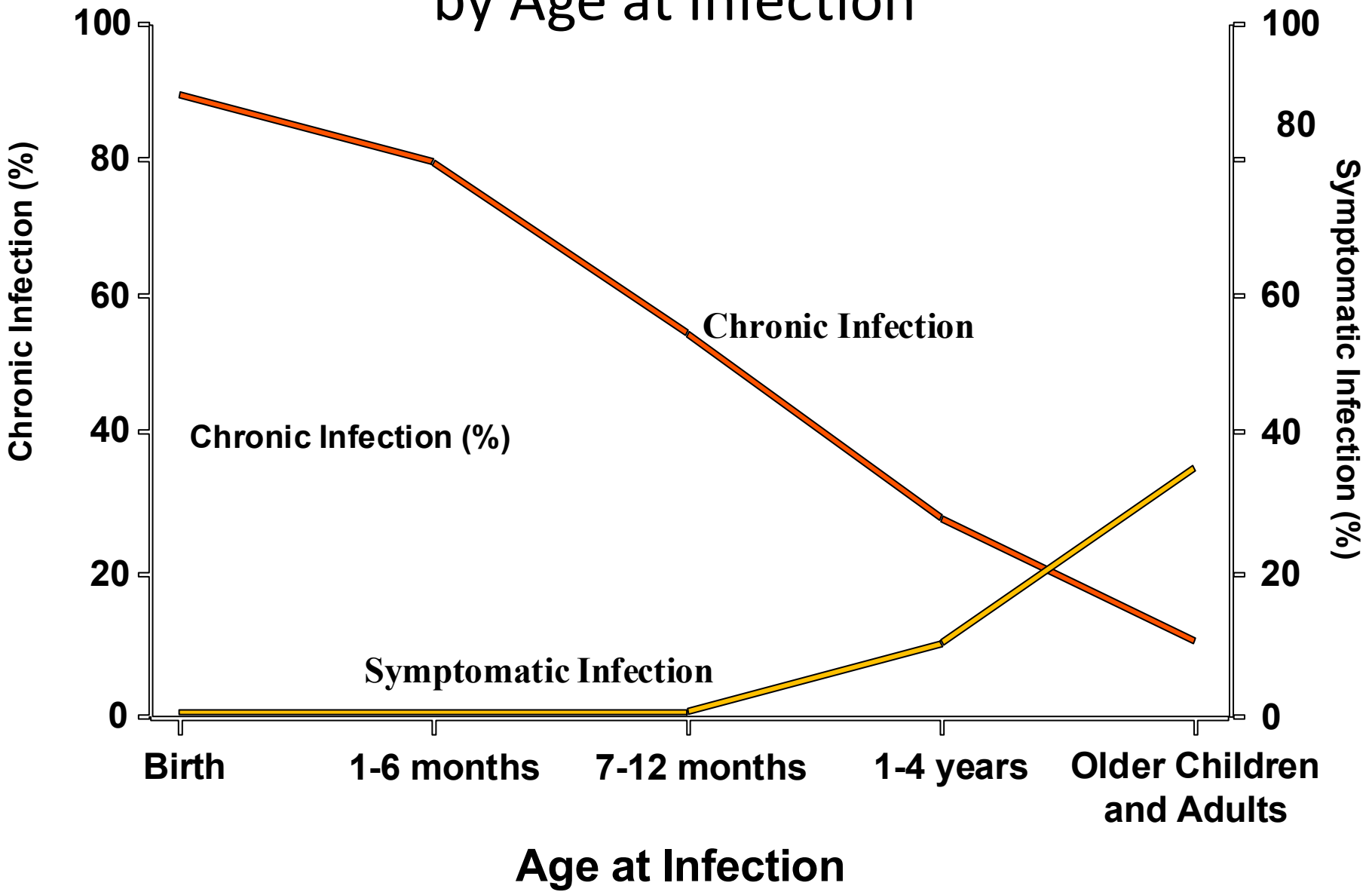
1. Chronic Persistent Hepatitis - asymptomatic
2. Chronic Active Hepatitis - symptomatic exacerbations of hepatitis
3. Cirrhosis of Liver
4. Hepatocellular Carcinoma

Acute Hepatitis B Virus Infection with Recovery Typical Serologic Course



- Serologic markers in hepatitis B virus infection are numerous and are important for identifying the stage of infection.
- This slide demonstrates acute hepatitis B virus infection followed by recovery, not chronic infection.
- During the acute phase of illness, patients have positive hepatitis B surface antigen (HbsAg). Later, after recovery, they develop anti-hepatitis B surface antibodies. During the period of active viral replication, hepatitis B e antigen levels are high. After recovery, patients also develop anti-hepatitis B e antibodies.
- Hepatitis B e antigen (HBeAg) as well as Hepatitis B surface antigen indicate active viral replication.
- There is a window period between the disappearance of hepatitis B surface antigen (red) and the appearance of anti-hepatitis B surface antibodies (black). During this period, the only detectable marker may be anti-hepatitis B **core** IgM antibody. Therefore, when acute hepatitis B virus infection is suspected, it is important to measure hepatitis B surface antigen, anti-hepatitis B surface antibody, and anti-core antibody, which is particularly useful during the window period.

Outcome of Hepatitis B Virus Infection by Age at Infection



Concentration of Hepatitis B Virus in Various Body Fluids

High	Moderate	Low/Not Detectable
blood	semen	urine
serum	vaginal fluid	feces
wound exudates	saliva	sweat
		tears
		breastmilk

The modes of transmission of hepatitis B virus include exposure to infected blood, serum, and wound exudates.

Hepatitis B Virus
Modes of Transmission

- Sexual - sex workers and homosexuals are particular at risk.
- Parenteral - IVDA, Health Workers are at increased risk.
- Perinatal (i.e. Vertical transmition) - Mothers who are HBeAg positive are much more likely to transmit to their offspring than those who are not. Perinatal transmission is the main means of transmission in high prevalence populations.

Diagnosis

Already discussed this topic, and you are not required to know the serologic markers of hepatitis B virus for the exam

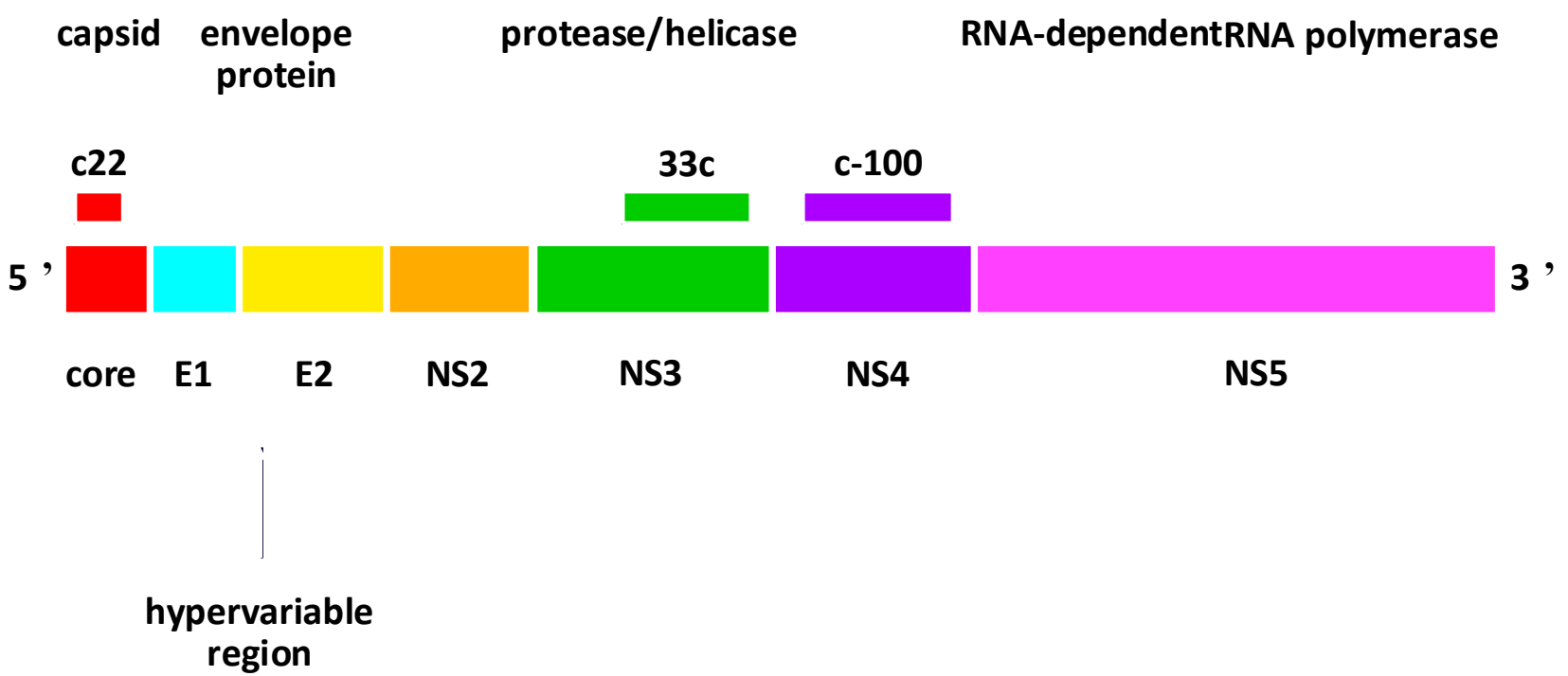
- **A battery of serological tests are used for the diagnosis of acute and chronic hepatitis B infection.**
- **HBsAg - used as a general marker of infection.**
- **HBsAb - used to document recovery and/or immunity to HBV infection.**
- **anti-HBc IgM - marker of acute infection.**
- **anti-HBcIgG - past or chronic infection.**
- **HBeAg - indicates active replication of virus and therefore infectiveness.**
- **Anti-Hbe - virus no longer replicating. However, the patient can still be positive for HBsAg which is made by integrated HBV.**
- **HBV-DNA - indicates active replication of virus, more accurate than HBeAg especially in cases of escape mutants. Used mainly for monitoring response to therapy.**

Prevention

- **Vaccination** -all of you have already been vaccinated against hepatitis B virus. **highly effective recombinant vaccines are now available. Vaccine can be given to those who are at increased risk of HBV infection such as health care workers. It is also given routinely to neonates as universal vaccination in many countries.**
- **Hepatitis B Immunoglobulin - HBIG may be used to protect persons who are exposed to hepatitis B. It is particular efficacious within 48 hours of the incident. It may also be given to neonates who are at increased risk of contracting hepatitis B i.e. whose mothers are HBsAg and HBeAg positive.**
- **Other measures - screening of blood donors, blood and body fluid precautions.**

Pregnant patients with a high hepatitis B viral load should receive treatment during the third trimester. After delivery, the newborn should immediately receive hepatitis B immunoglobulins together with the hepatitis B vaccine. These preventive measures are implemented to reduce and prevent vertical transmission from the mother to the baby.

Hepatitis C Virus RNA virus



Hepatitis C - Clinical Features

Incubation period:	Average 6-7 wks Range 2-26 wks
Clinical illness (jaundice):	30-40% (20-30%)
Chronic hepatitis:	70%
Persistent infection:	85-100%
Immunity:	No protective antibody response identified

Unlike hepatitis B virus, for which an effective vaccine is available, there is currently no proven vaccine for hepatitis C virus. There is also a vaccine for hepatitis A virus, although it is not included in the national vaccination program in Jordan. However, hepatitis A vaccination should be offered to high-risk patients, such as individuals with liver cirrhosis or chronic hepatitis B virus infection.

Chronic Hepatitis C Infection

- **The spectrum of chronic hepatitis C infection is essentially the same as chronic hepatitis B infection.**
- **All the manifestations of chronic hepatitis B infection may be seen, albeit with a lower**

Risk Factors Associated with Transmission of HCV

- **Blood Transfusion or transplant from infected donor**
- **Injecting drug use**
- **Hemodialysis (yrs on treatment)**
- **Accidental injuries with needles/sharps**
- **Sexual/household exposure to anti-HCV-positive contact**
- **Multiple sex partners**
- **Homosexual partners**
- **Birth to HCV-infected mother**

Laboratory Diagnosis

- **HCV antibody - generally used to diagnose hepatitis C infection. Not useful in the acute phase as it takes at least 4 weeks after infection before antibody appears.**
- **HCV-RNA - various techniques are available e.g. PCR and branched DNA. May be used to diagnose HCV infection in the acute phase. However, its main use is in monitoring the response to antiviral therapy.**
- **HCV-antigen - an EIA for HCV antigen is available. It is used in the same capacity as HCV-RNA tests but is much easier to carry out.**

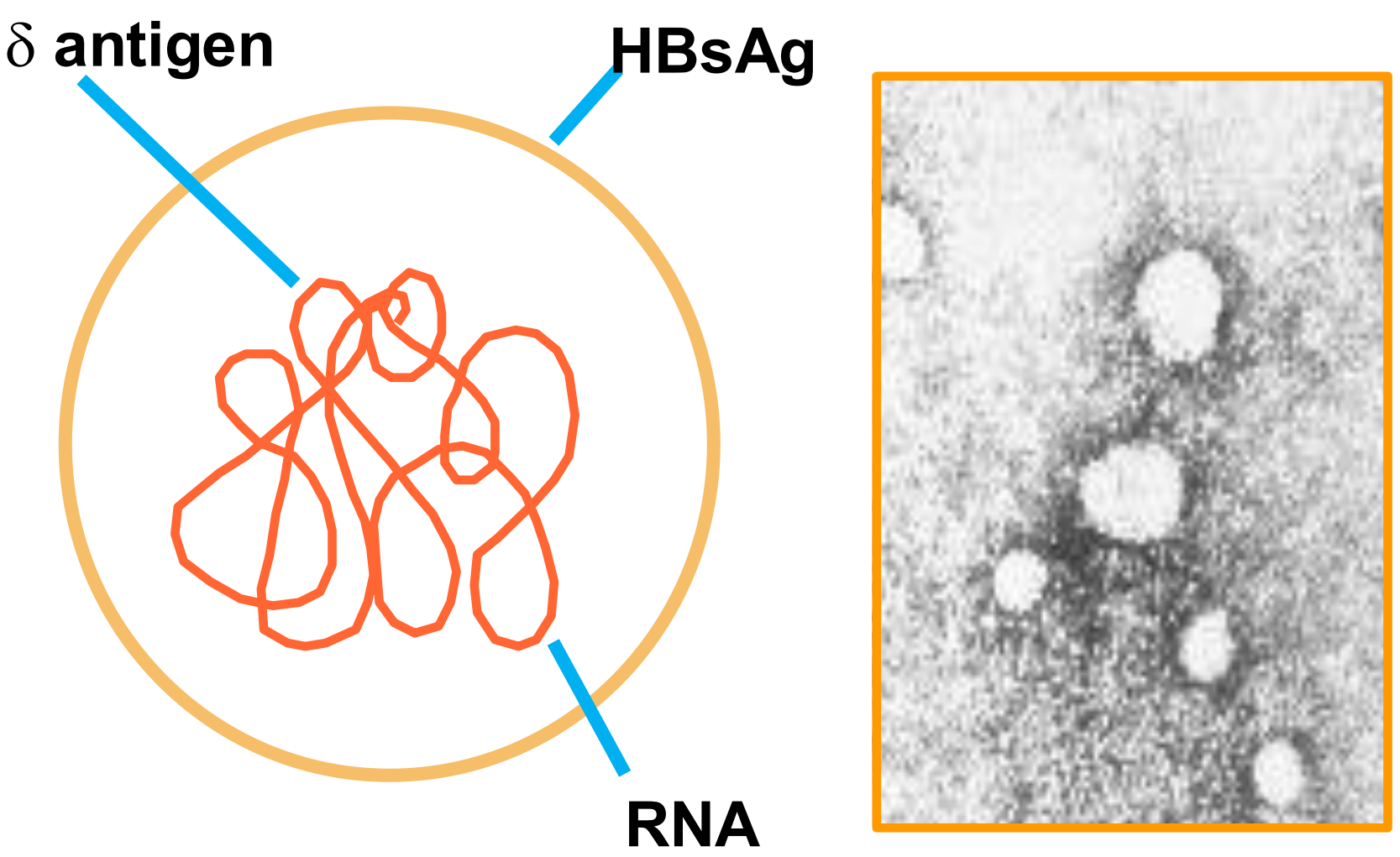
Laboratory Diagnosis

To diagnose hepatitis C virus infection, we first test for hepatitis C virus antibodies. If the antibody test is negative, chronic hepatitis C infection is excluded. If the antibody test is positive, there are two explanations: either the patient had a previous infection and recovered from the virus, or the patient currently has chronic hepatitis C infection.
To differentiate between these two possibilities, hepatitis C virus RNA is tested using PCR. If the PCR result is positive, this indicates that the patient has chronic hepatitis C virus infection. If the PCR result is negative, it means that the patient had a previous infection but successfully cleared the virus and recovered completely, with no need for further follow-up.

Prevention of Hepatitis C (remember that no vaccine is available)

- Screening of blood, organ, tissue donors
- High-risk behavior modification
- Blood and body fluid precautions

Hepatitis D (Delta) Virus



Hepatitis D virus cannot infect a patient on its own. It requires the presence of hepatitis B virus in order to replicate and cause infection, since hepatitis D depends on hepatitis B surface antigen to complete its life cycle.

Hepatitis D - Clinical Features

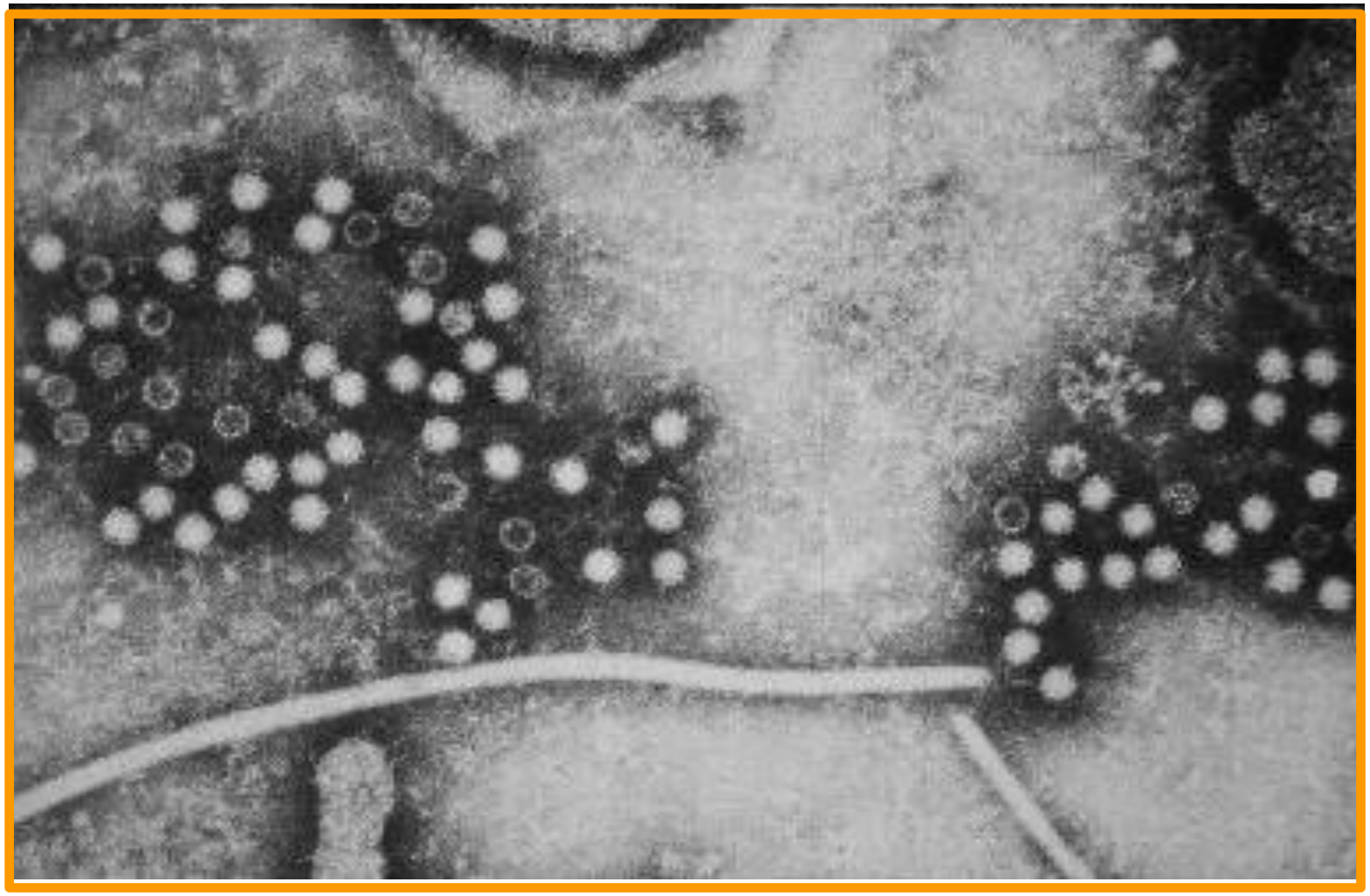
- Coinfection; the patient acquires both hepatitis B virus and hepatitis D virus simultaneously from the same source
  - severe acute disease.
  - low risk of chronic infection.
- Superinfection; the patient already has chronic hepatitis B virus infection and later acquires hepatitis D virus from another source
  - usually develop chronic HDV infection.
  - high risk of severe chronic liver disease.
  - may present as an acute hepatitis.

Hepatitis D Virus Modes of Transmission

It is transmitted in the same manner as hepatitis B virus, mainly through bloodborne transmission.

- Percutanous exposures
  - injecting drug use
- Permucosal exposures
  - sex contact

Hepatitis E Virus



Hepatitis E virus is similar to hepatitis A virus in its modes of transmission, primarily through the fecal-oral route, usually by ingestion of contaminated food or water.

Hepatitis E - Clinical Features

- Incubation period:
  - Average 40 days

  Range 15-60 days
- Case-fatality rate:
  - Overall, 1%-3%

  Pregnant women, 15%-25%
- Illness severity:
  - Increased with age
- Chronic sequelae:
  - None identified

In most patients, it causes a self-limiting acute illness and does not lead to chronic hepatitis, unlike hepatitis B and hepatitis C viruses. However, hepatitis E infection is associated with a high mortality rate in pregnant females when infection occurs during pregnancy.

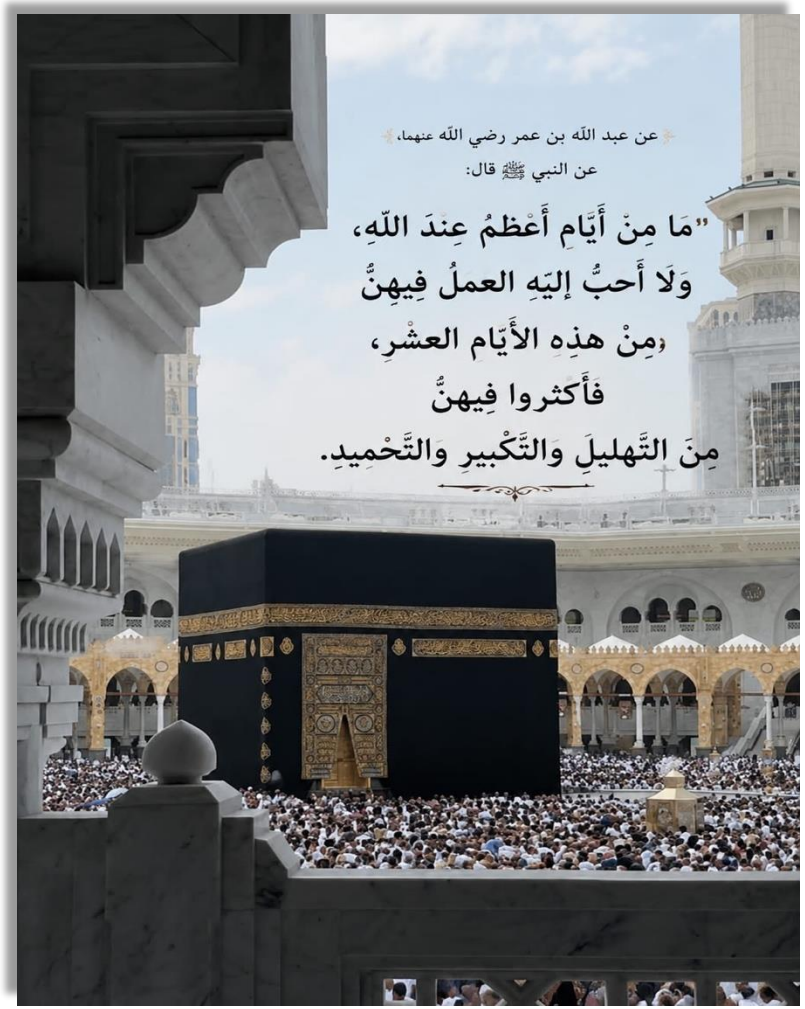
رسالة من الفريق العلمي:

﴿وَلَيَالٍ عَشْرٍ﴾

ما أقسمَ اللهُ بشيءٍ عبثًا، وكيف وهي أيامٌ تتنزّل فيها الرحمات، وتُفتح فيها أبواب القُرب، وتُكتب فيها البدايات الجديدة للقلوب.**عشرُ ذي الحجة** ليست أيامًا عابرة في التقويم، بل مواسمُ عمرٍ قد لا تتكرر، **يُضاعف فيها الأجر**، وتُغسل فيها الأرواح بالتكبير والذكر والطاعة.اغتنموها وكأنها رسالةُ لطفٍ من الله بعد تعبٍ طويل؛ أكثروا فيها من التكبير، وأحيوا أيامكم بالقرآن ولو بوردٍ يسير، وبالصدقة ولو بالقليل ، وبصيام ما استطعتم ، وخاصة **يوم عرفة** ؛ يومٌ تُرجى فيه المغفرة وتُعتق فيه الرقاب.

وفي هذه العشر، لا تبحثوا فقط عن الأعمال العظيمة، بل عن الصدق فيها؛ ركعتان بخشوع، دعوة من قلبٍ مُنهك ، إصلاحُ ما بينك وبين الله ، برُّ والدين، جبرُ خاطر، أو حتى توبةٌ صادقة... قد تكون سببًا لتبدّل العمر كله.

هي أيامٌ قصيرة، لكنها ثقيلةٌ في الميزان، فاملؤوها بما تحبّون أن تلقوا الله به.



دعواتكم لنا في الأيام الفضيلة🌷

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Corrections from previous versions:

Versions	Slide # and Place of Error	Before Correction	After Correction
V0 → V1			
V1 → V2			