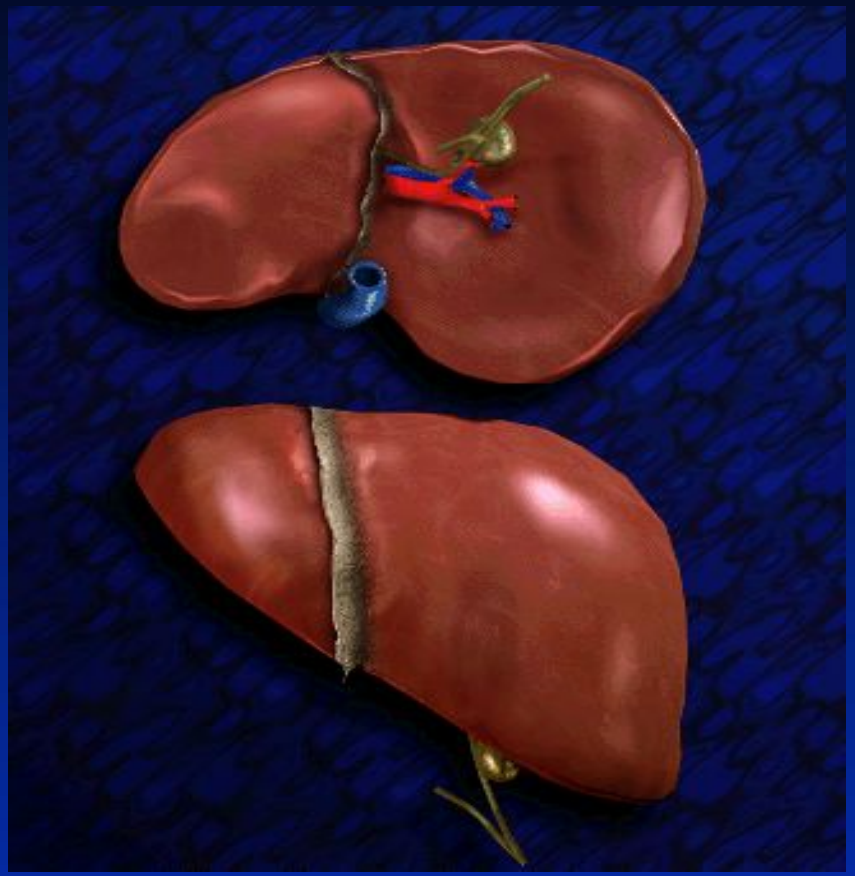
Viral Hepatitis



Summary

- Viral hepatitis is a common cause of acute and chronic liver disease
- Diagnosis relies on clinical suspicion and serology/virology for confirmation
- HBV is a treatable and preventable disease
- HCV can be cured with adequate therapy

Functions of the Liver

1. processing of dietary amino acids, carbohydrates, lipids and vitamins
2. removal of microbes and toxins in splanchnic blood
3. synthesis of plasma proteins
4. detoxification and excretion into bile of endogenous waste products & pollutant xenobiotics

PATHOGENESIS VIRAL HEPATITIS A

Ingestion / inoculation

Replication - Viremia

Liver – major site replication.

Cellular immune response.

Apoptosis, necrosis of hepatocytes.

Inflammation - Hepatitis

Bridging Hepatocyte necrosis (Central vein, portal triad)

Fibrosis – patchy/bridging

Cirrhosis – extensive fibrosis with loss of archetecture & regenerating nodules.

Liver Failure, Coma, Carcinoma..

PATHOGENESIS VIRAL HEPATITIS B, C

Ingestion / inoculation

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Liver Failure, Coma, Carcinoma..

Incubation period

Hepatitis A 15 – 49 days (average 25)

Hepatitis E 15 – 60 days

Hepatitis C 14 – 160 days

Hepatitis B 60-180 days

Hepatitis D 21 – 45 days

The next variants of prejaundice (prodromal) period:

- **1. Dyspeptic variant. – The patients complain loss of appetite, nausea, sometimes vomiting. The temperature is subfebrile. Duration of the period is 3 - 7 days.**
- **2. Astenovegetative variant. – The patients complain on weakness, headache, malaise, loss of appetite. Body temperature is subfebrile or 37 - 38˚C;**
- **3. Influenza-like variant. – The patients complain of headaches, weakness; muscular pains, loss of appetite. Body temperature is 37.5 - 39 C, and in some cases 39 - 40 C.**

Duration of 2nd and 3rd variant of prejudice period is 5 - 7 days;

- **4. Polyarthralgic variant. – It is principally observed in hepatitis B and C. The patients complain of pains in joints, sometimes muscular pains, weakness, loss of appetite. In this period subfebrile temperature is in the majority of the patients. Duration of this period is 7 - 14 days;**

- **5. Mixed type – all above mentioned signs of intoxication are in various degree of manifestation.**

Period of the clinical manifestation

The condition of the majority of the patients becomes better. The temperature is normalized, urine becomes dark, stool is decolorize. Scleras are icteric, jaundice grows gradually.

The further course of the disease depends on the degree of the liver damage with virus, who determines the severity of the disease.

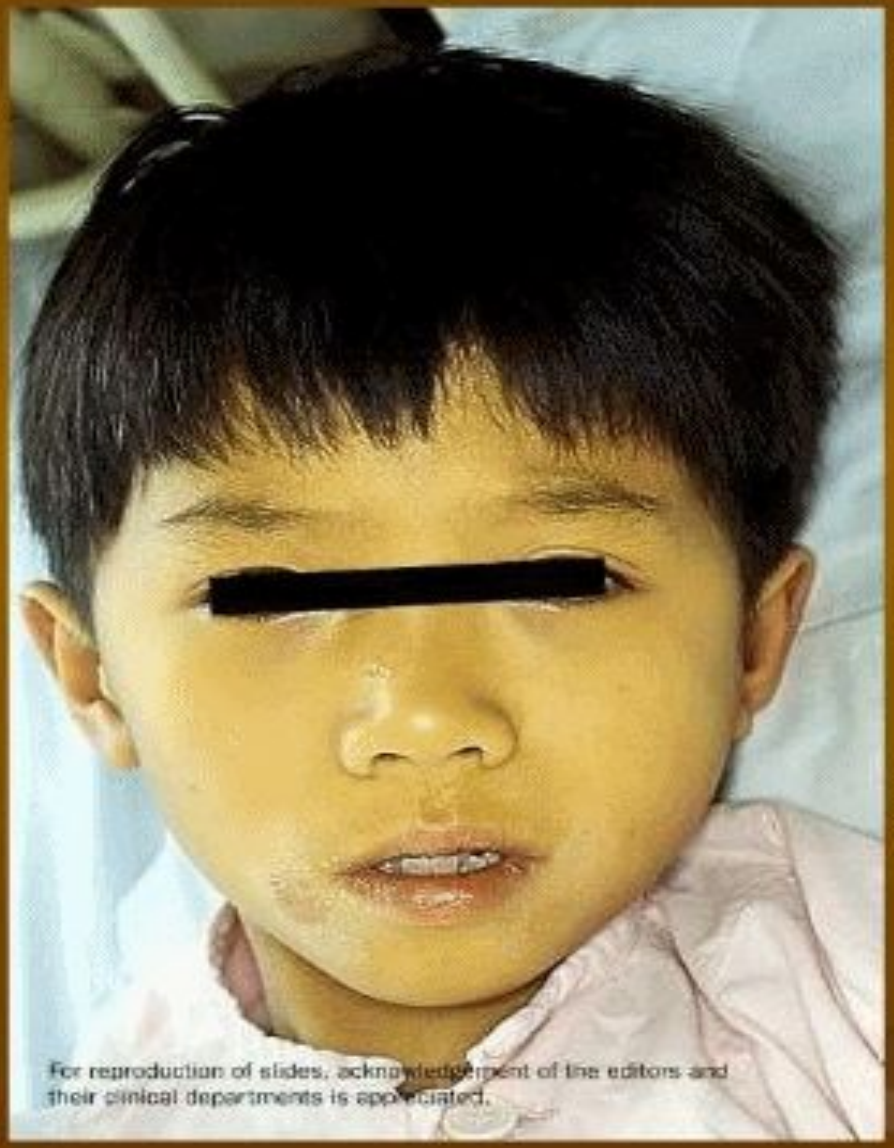
Signs and symptoms

Man with jaundice

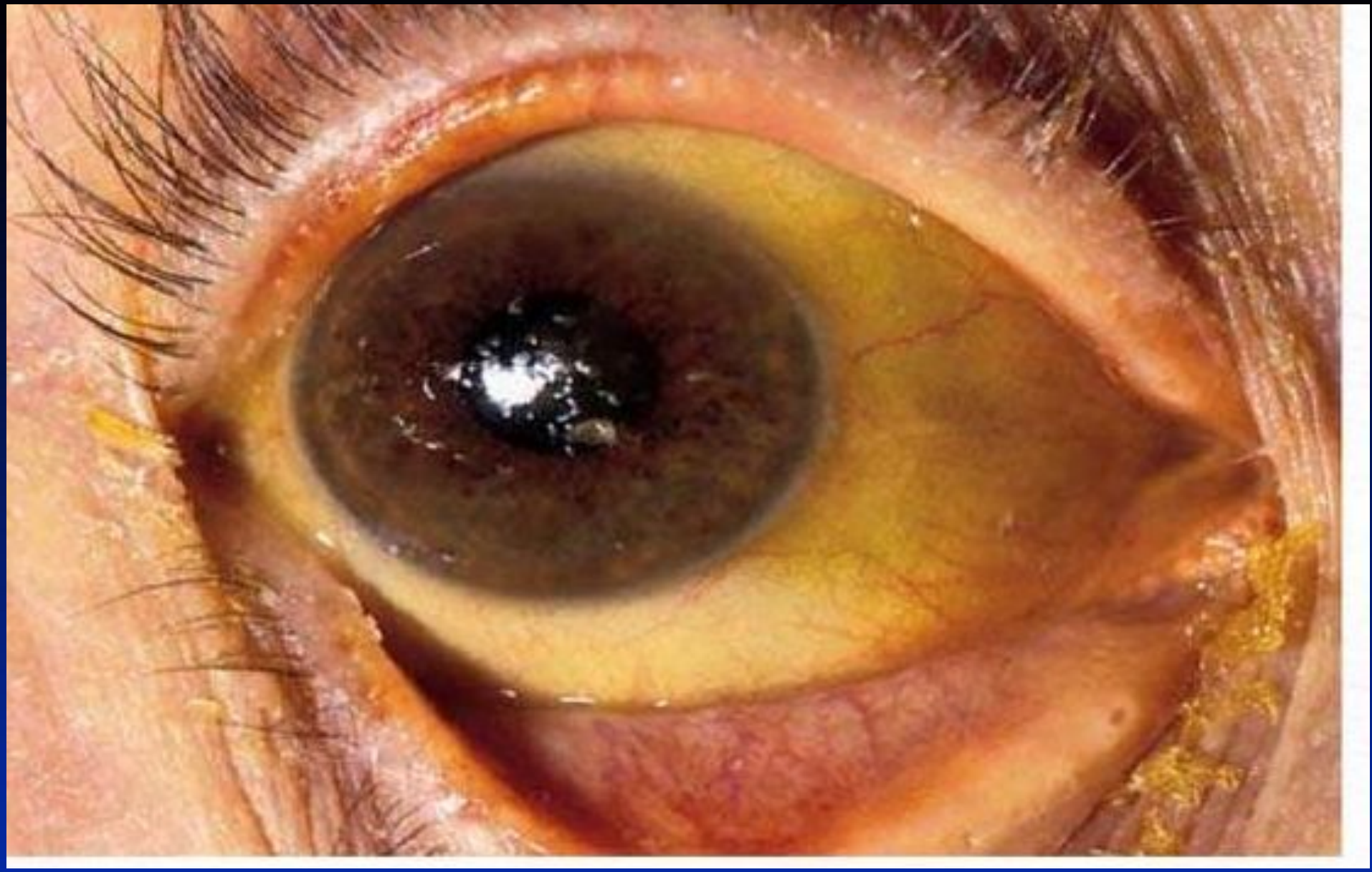
Icteric phase.

- Dark urine appears first (bilirubinuria).
- Pale stool soon follows, although this is not universal.
- Jaundice occurs in most (70-85%) adults with acute hepatitis A virus infection.
- Abdominal pain occurs in approximately 40% of patients.
- Itch (pruritus), although less common than jaundice, is generally accompanied by jaundice.
- Arthralgias and skin rash, although associated, are less frequent than the above symptoms. Rash more often occurs on the lower limbs and may have a vasculitic appearance.

Physical examination



The physical examination focuses on detecting features to support a diagnosis of acute hepatitis and should include assessment for features of chronic liver disease or similarly assessment for evidence of decompensation. Hepatomegaly is common. Jaundice or scleral icterus may occur. Patients may have a fever with temperatures of up to 40°C.



Jaundice, a yellow discoloration of all tissues and organs, including the eye and the skin.

- **In hepatitis B, C and D moderately severe and severe course, prolonged and chronic forms of the disease and lethal outcome are not infrequently observed.**

The main clinical syndromes VG

- Intoxication
- Jaundice
- Hemorrhagic
- Cytolytic
- Cholestatic
- Neurological

Criteria for the severity of VG:

- The degree of intoxication

- The level of hyperbilirubinemia:

- Up to 100 mcmol / l mild course
- Up to 200 mcmol / l for moderately course
- More than 200 mcmol / l severe course

- **In the mild course of viral hepatitis jaundice grows for 3 - 5 days. It is at one level for one week. Disappearance of jaundice is observed on the 15-16th day. Urine becomes more light at the end of the first-second week of the jaundice period.**

- **During moderate and severe courses of the disease yellowish colouring of the sclera's, skin is more intensive, jaundice period is more prolonged (20 - 45 day).**

- **Present the signs of the disorder of the cardiovascular system: hypotension, bradycardia, dull hearts sounds.**

- **In most of patients the liver is enlarged, its surface is smooth, borders are curved, moderately painful.**

- **In 30 - 40% of the patients the spleen is palpated.**

- **Develops meteorism, caused by disorders of digestion (signs of the damage of pancreas, secretary glands of the stomach and disorders of biocenosis of the gastrointestinal tract) is observed in some patients.**

- **In some patients skin itching is marked.**

- **In severe cases course presence cerebral disorders caused by considerable dystrophic changes in the liver, endogenic intoxication.**

- **In the period of convalescence - reverse development of symptoms of the disease, normalization of biochemical indices is marked.**

Diagnostics

The preliminary diagnosis of viral hepatitis is based

- on epidemiological anamnesis
- findings of the disease development
- clinical picture with peculiarities of the ways of transmission
- duration of the incubation period
- presence of prejaundice period
- presence of typical subjective and objective signs
- taking to account of the patients age.

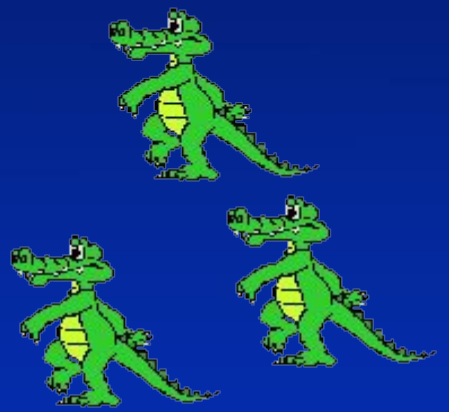
The diagnosis is confirmed by non-specific and specific laboratory tests:

In non-specific blood test of the patients with viral hepatitis:

- **leucopenia and lympho-monocytosis, ESR is slightly decreased**
- **Urobilin and bile pigments are observed in urine**
- **There is increased contents of general bilirubin in blood serum during all jaundice period, primarily on account of its direct fraction**
- **In all patients already in prejaundice period of the disease, during all jaundice period and in the period of early convalescence increased activity of ALT. It is evidence of cytolytic processes in the liver.**

specific laboratory tests

1. PCR
2. Elisa (Ig M, Ig G)



Interpretation of Serologic Markers

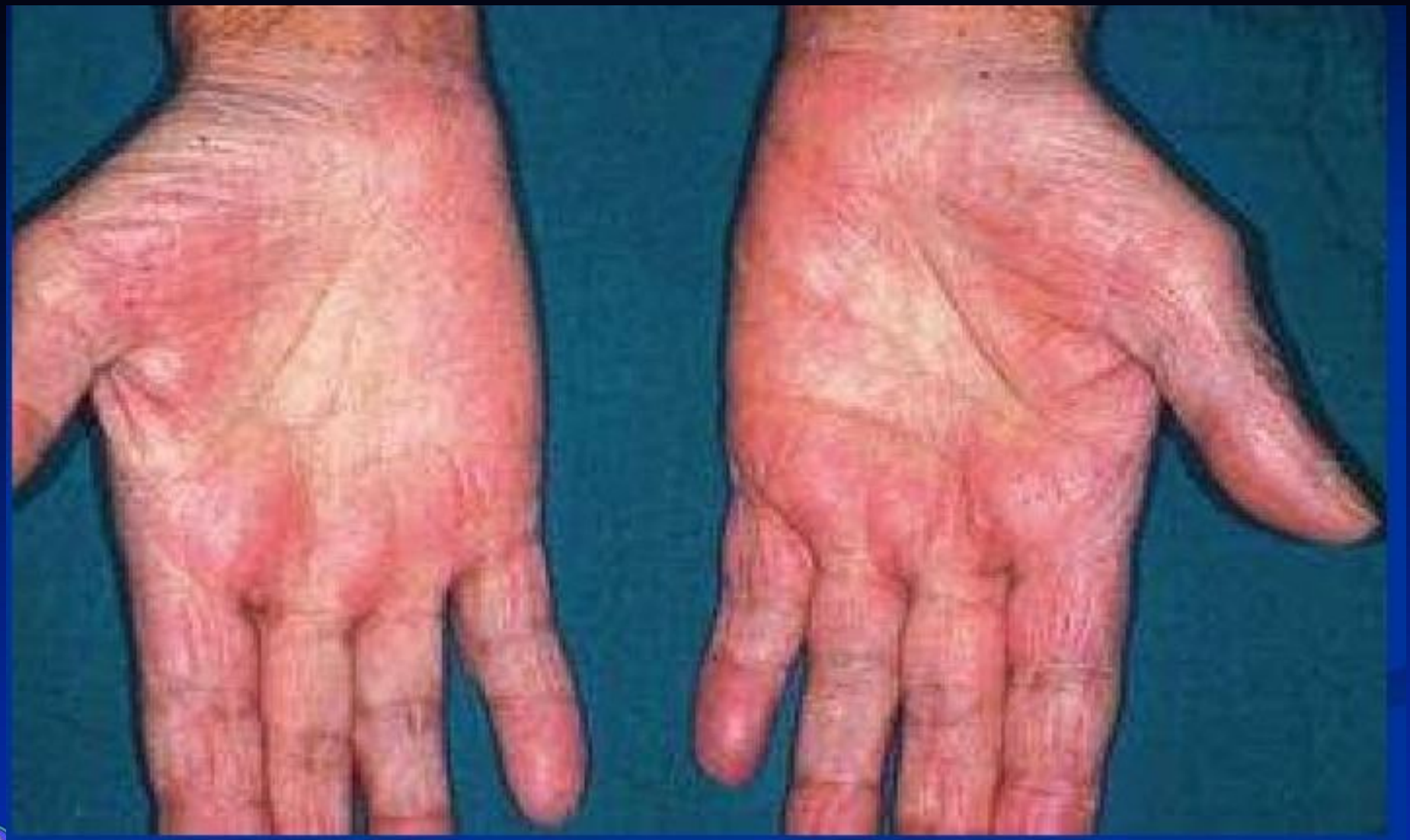
	Acute hepatitis B	Recovery from acute hepatitis B	Chronic HBeAg + disease	Chronic HBeAG – disease	Successful Vaccination	Resistance to antiviral agents
HBsAg	+		+	+		
Anti-HBs		+			+	
Anti-HBc IgM	+					
Anti-HBc	+	+	+	+		
HBeAg	+		+			
Anti-HBe		+ (in some Cases)		+		
HBV-DNA	+		+	+		+ (sequenc *pol* region)

CLINICAL AND LABORATORY SIGNS OF CHRONIC VIRAL HEPATITIS

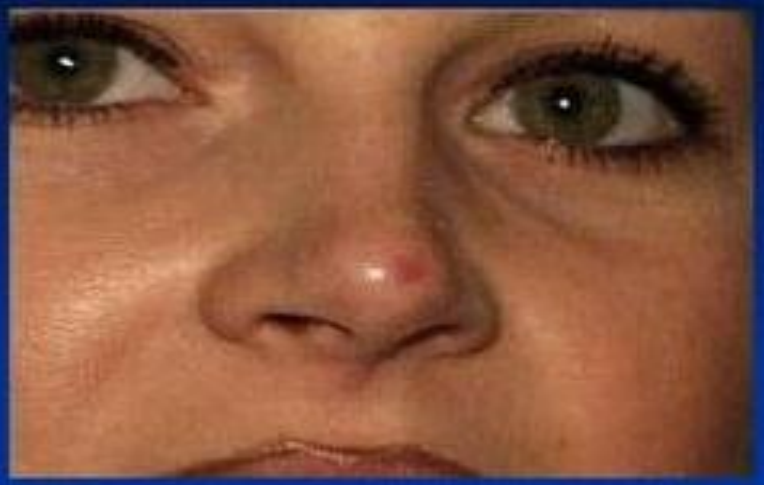
Clinical Features - Pathogenesis

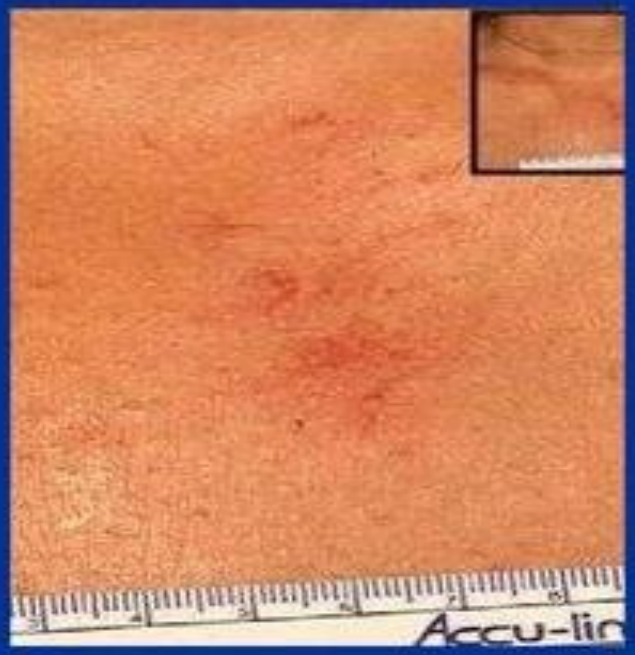
- Hypoalbuminemia
- Hyperammonemia
- Hypoglycemia
- Palmar erythema
- Spider angiomas
- Hypogonadism
- Gynecomastia
- Weight loss
- Muscle wasting
- Ascites
- Splenomegaly
- Esophageal varices
- Hemorrhoids
- Caput medusae-abdominal skin
- Complications of Hepatic Failure
- Coagulopathy
- Hepatic encephalopathy
- Hepatorenal syndrome

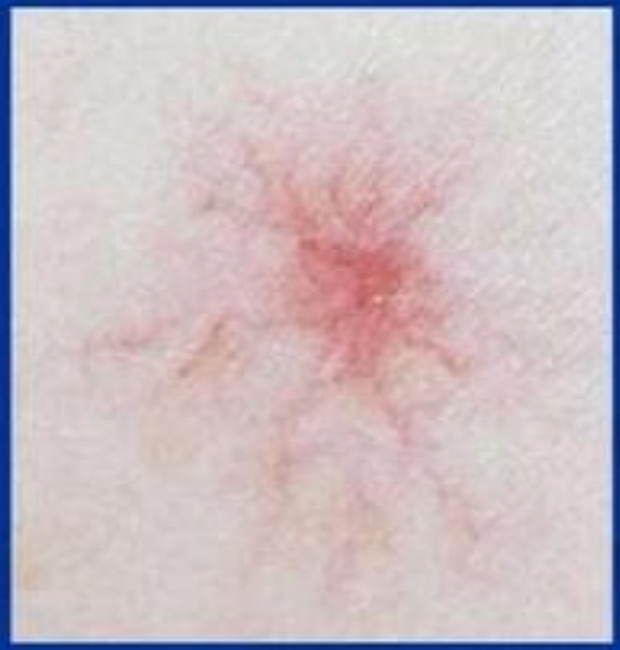
Palmar erythema

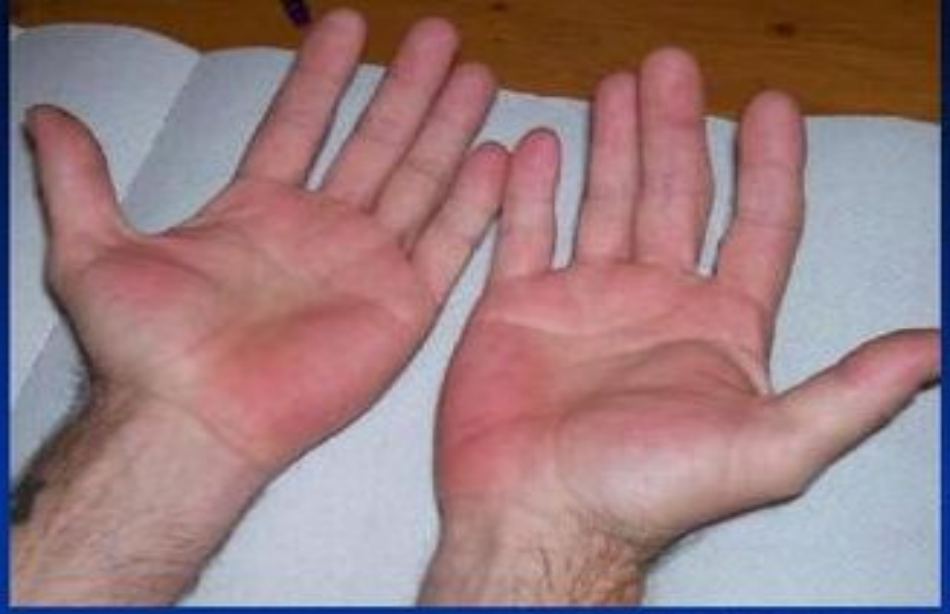


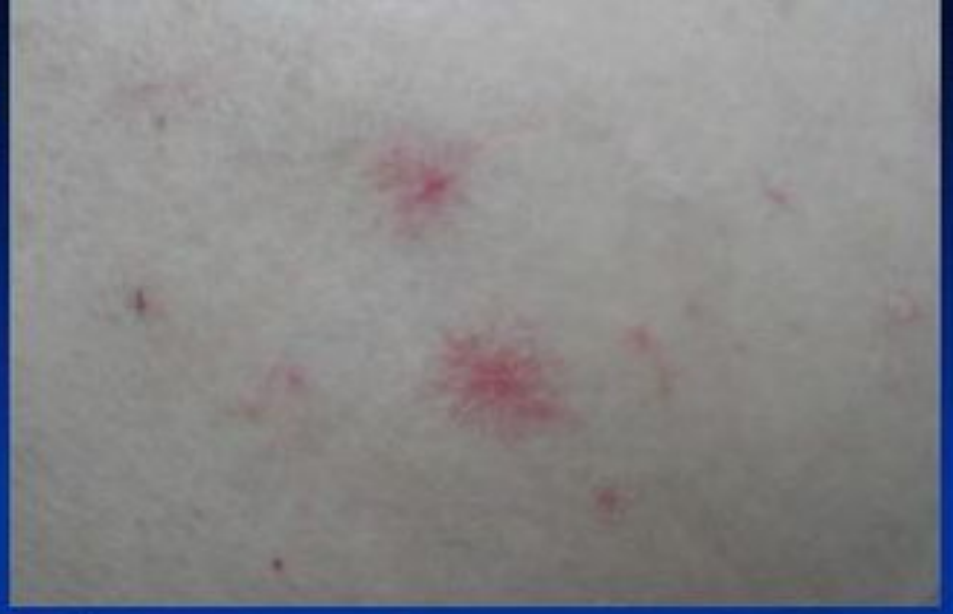
Spider nevi

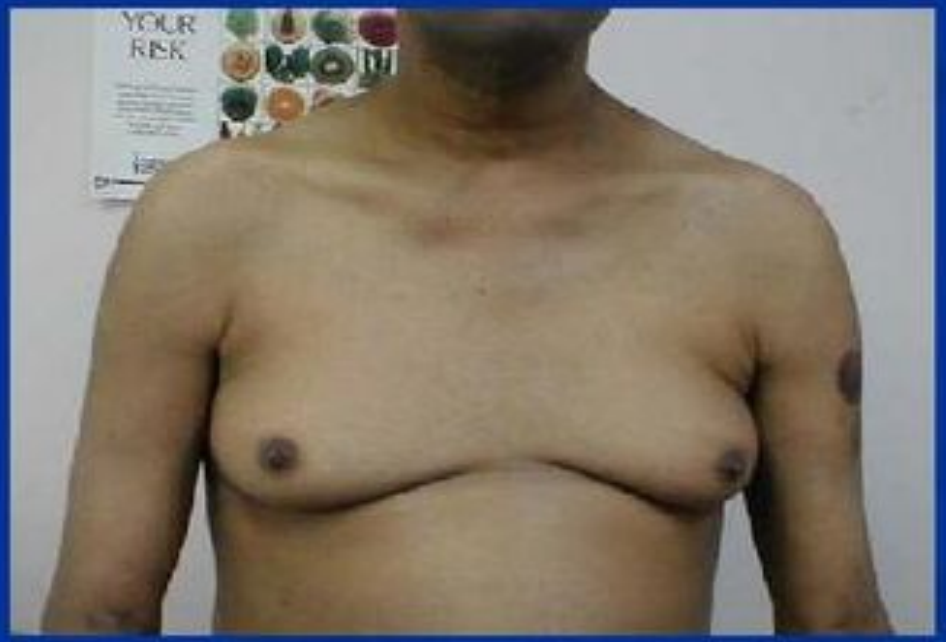








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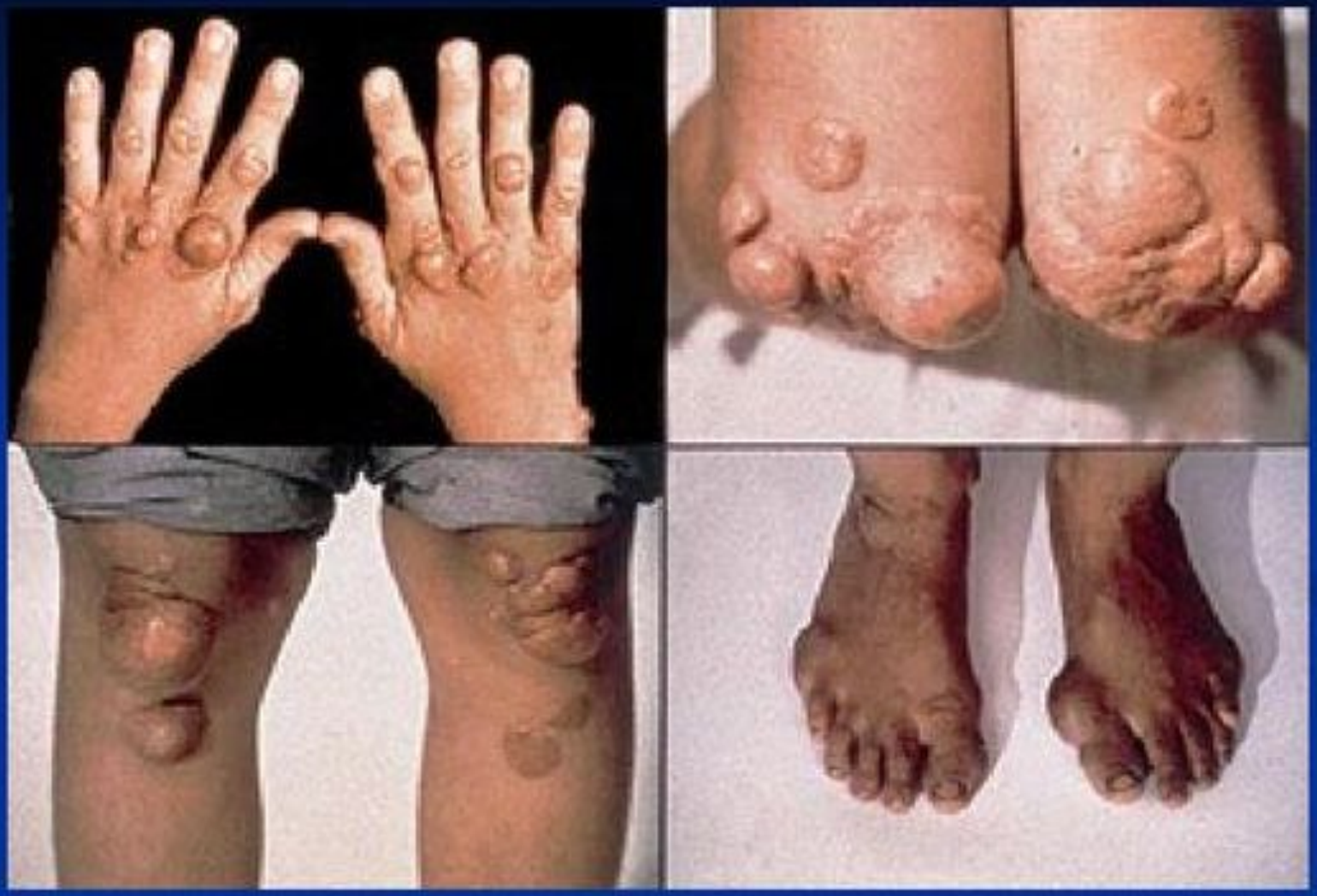
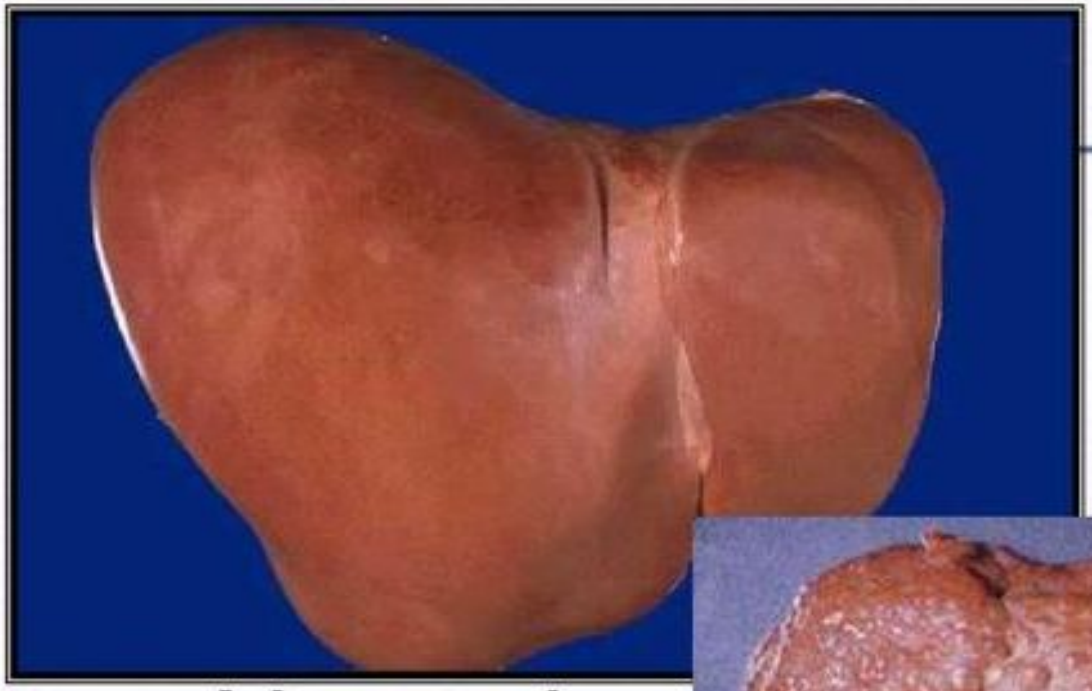


Skin xanthomas

Hepatic cirrhosis is a chronic, degenerative disease in which normal liver cells are damaged and are then replaced by scar tissue.

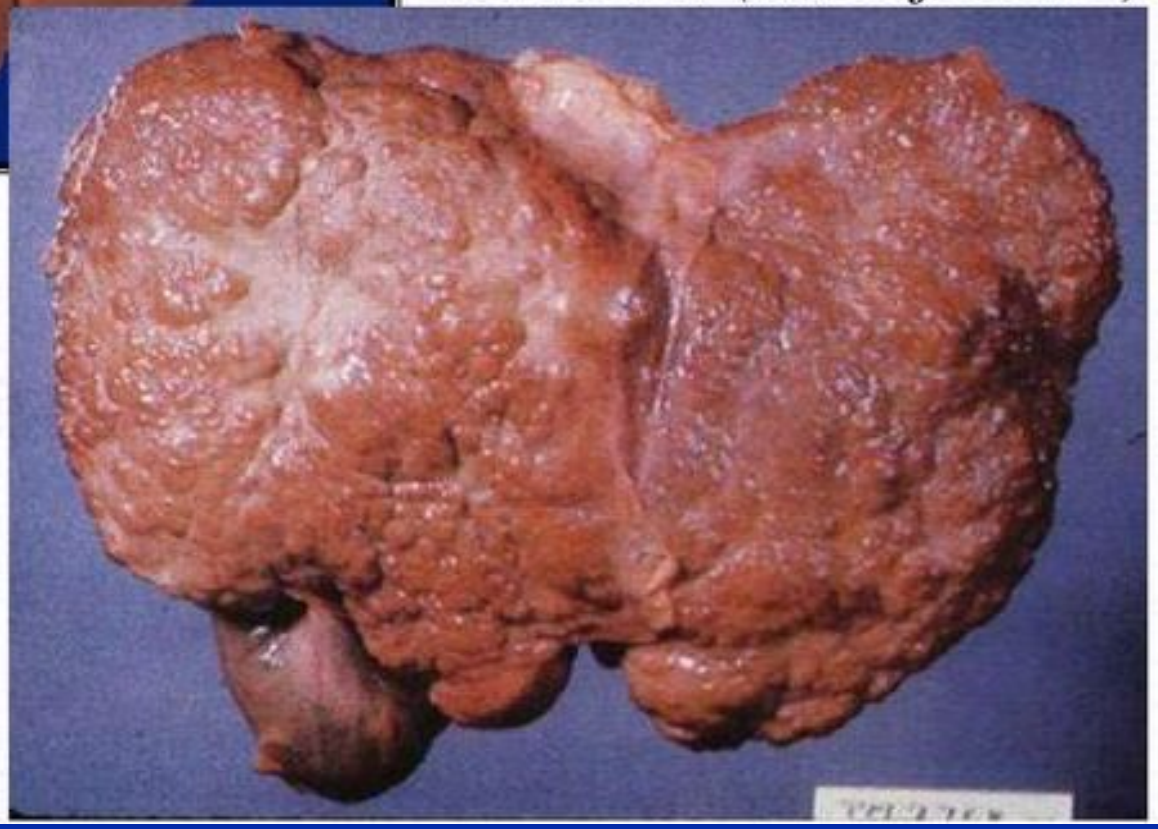


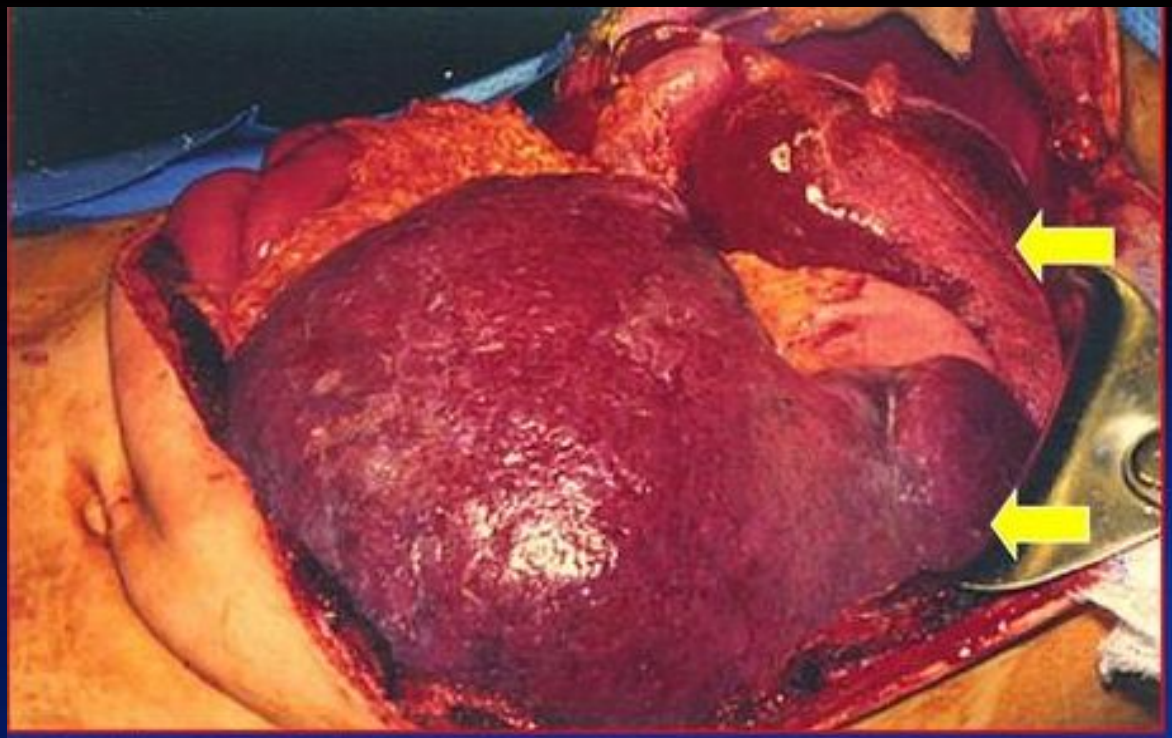
Normal

Cirrhosis

End stage of diffuse liver disease. scaring with regenerating nodules. (liver failure)

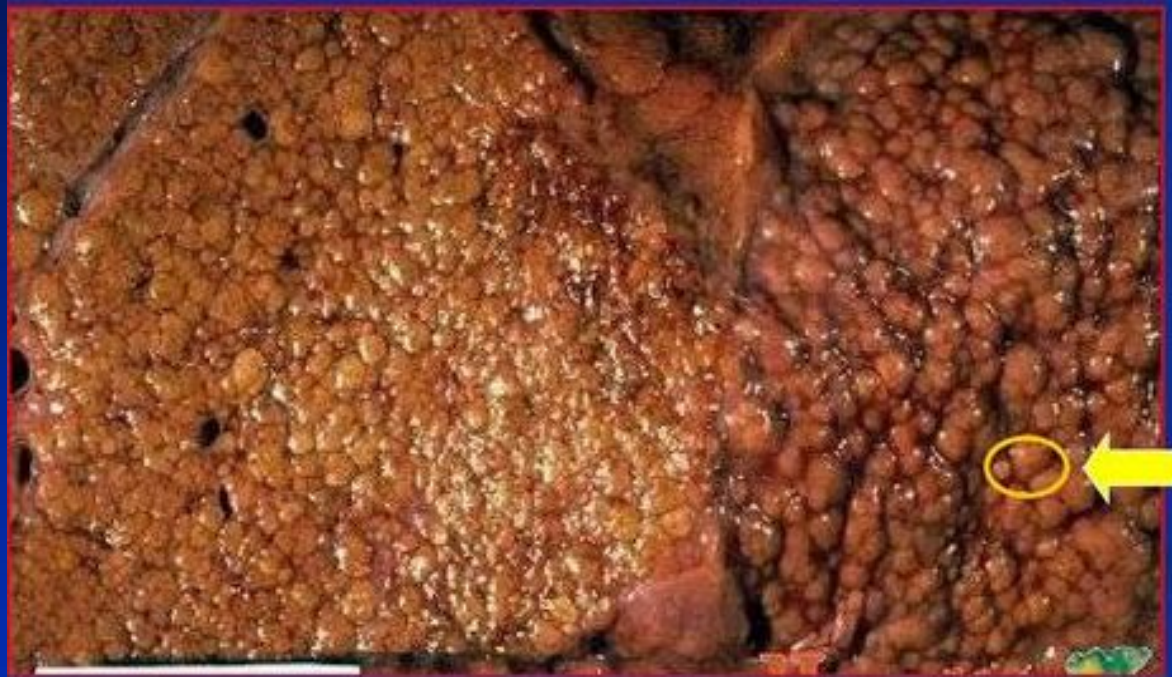
Cirrhosis →





Cirrhosis of Liver

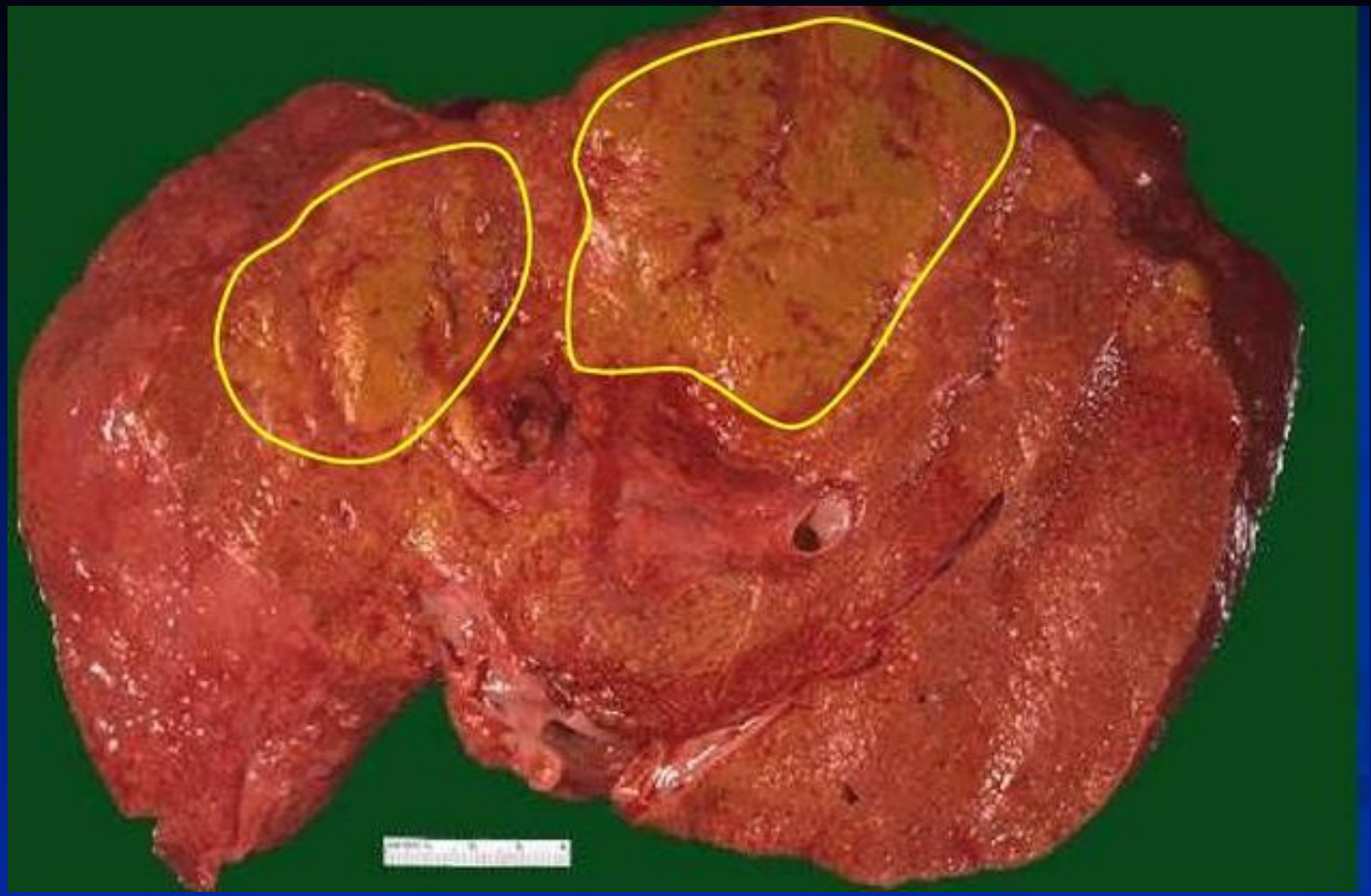
Spleen



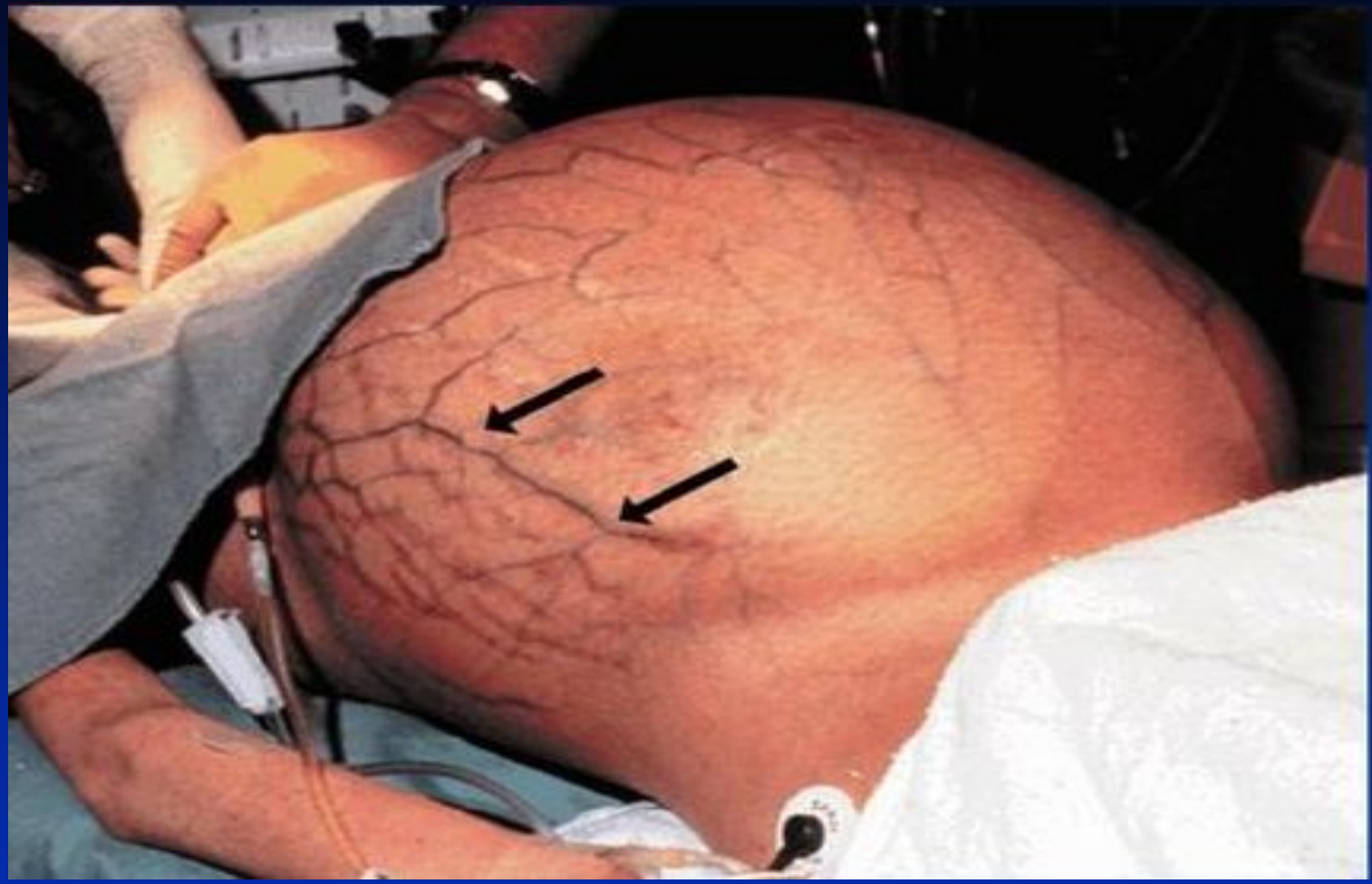
Cirrhosis of Liver

Nodular

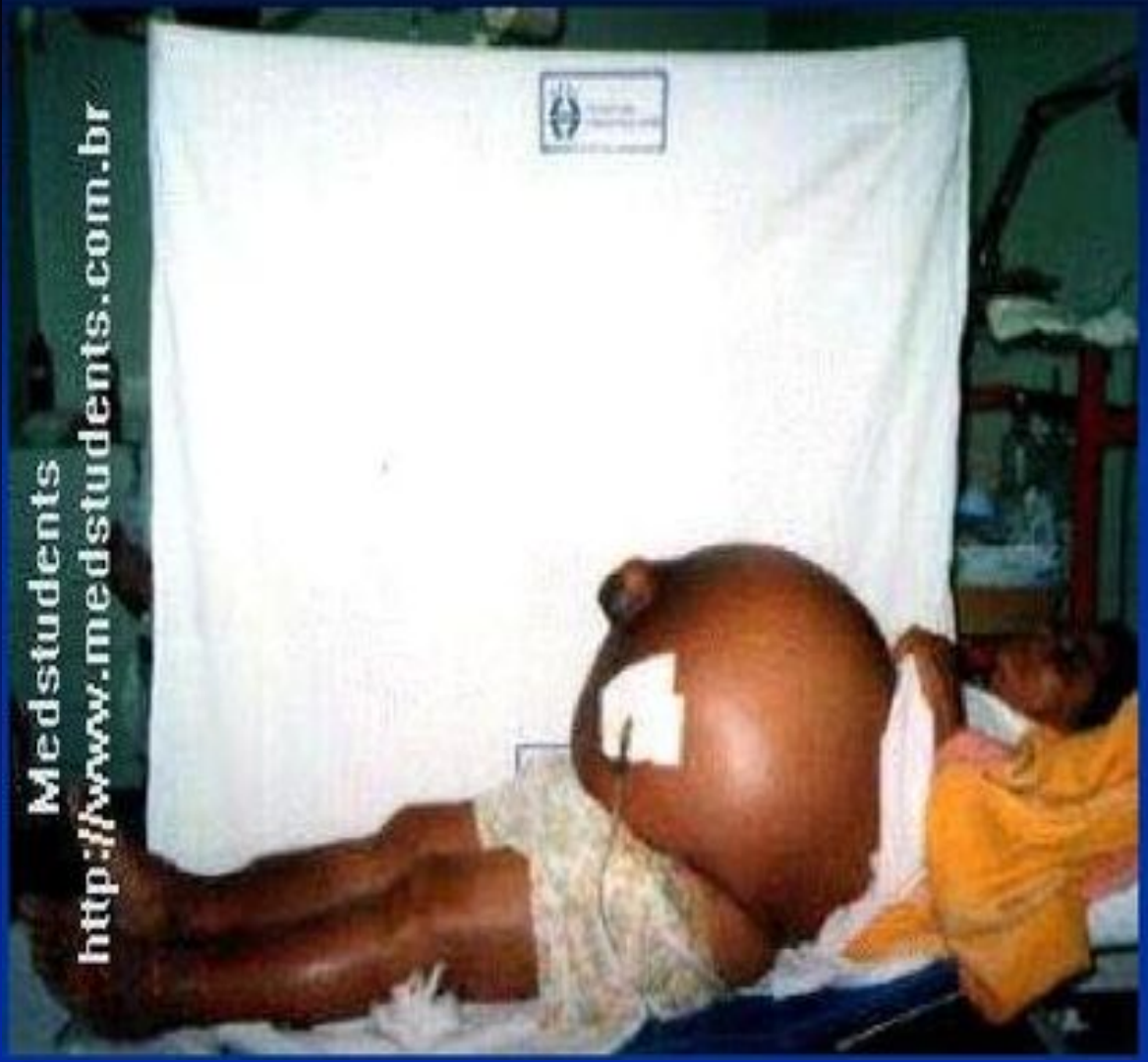
Hepatocellular carcinoma

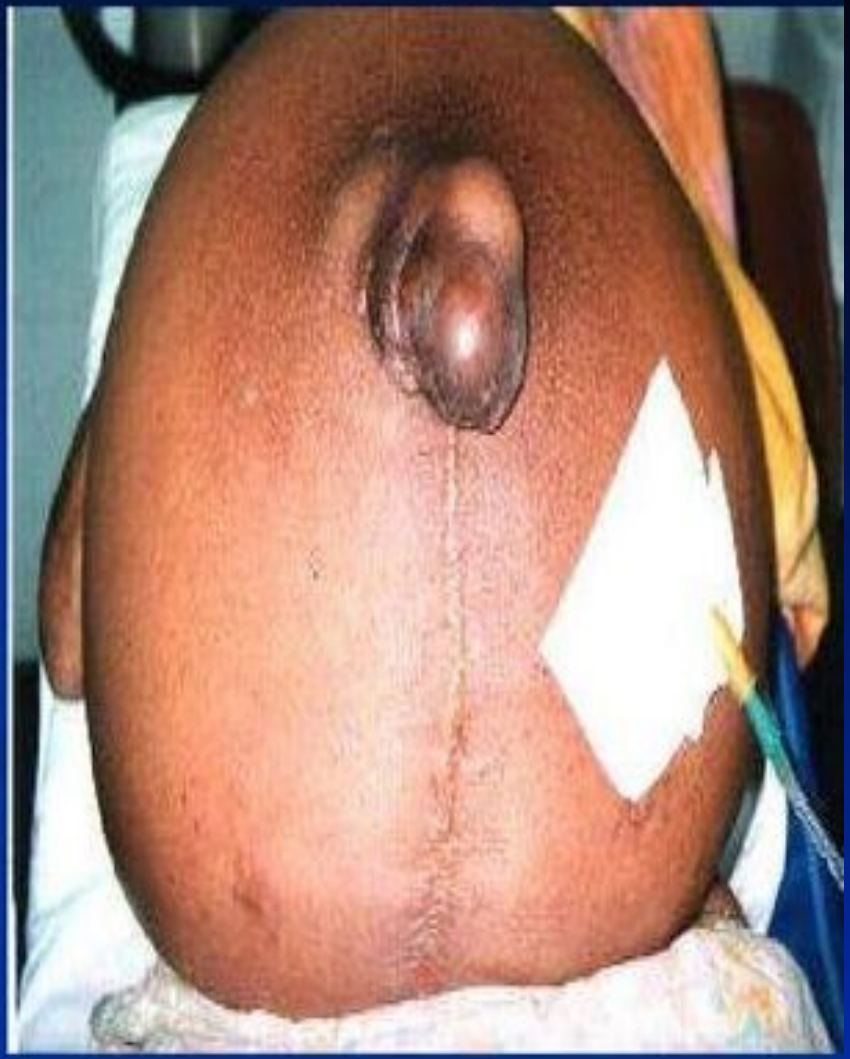


Photograph shows a caput medusae accentuated by a large amount of ascites in a patient being prepared for liver transplantation. An extensive plexus of veins is seen emanating from the umbilical region and radiating across the anterior abdominal wall.

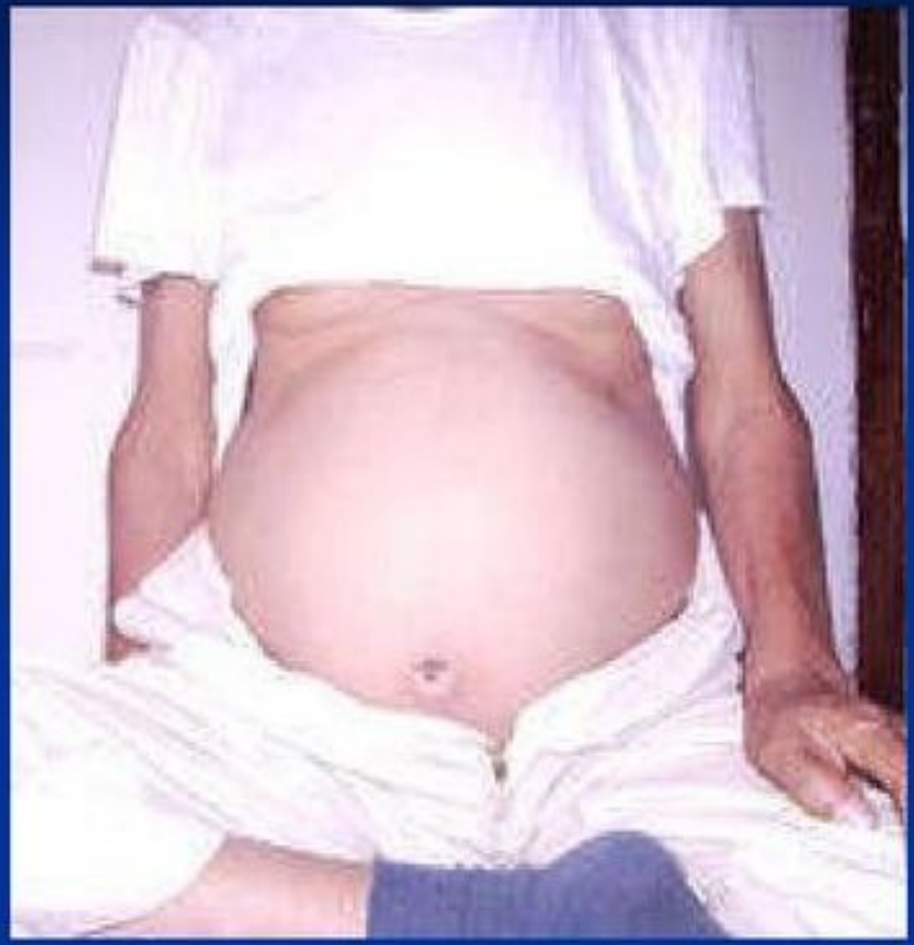


Ascites

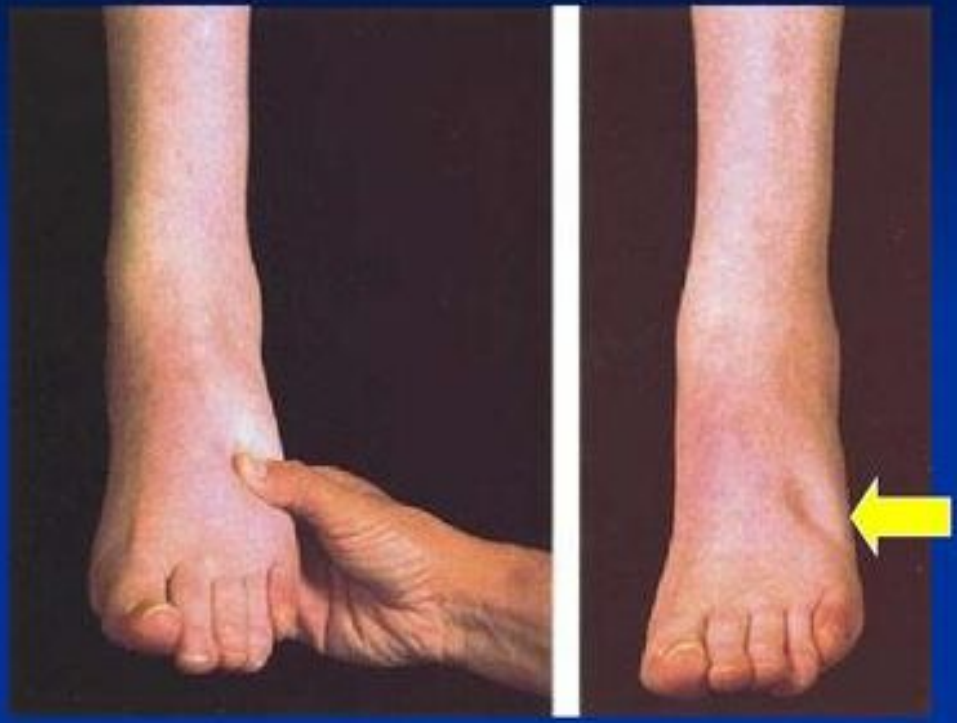


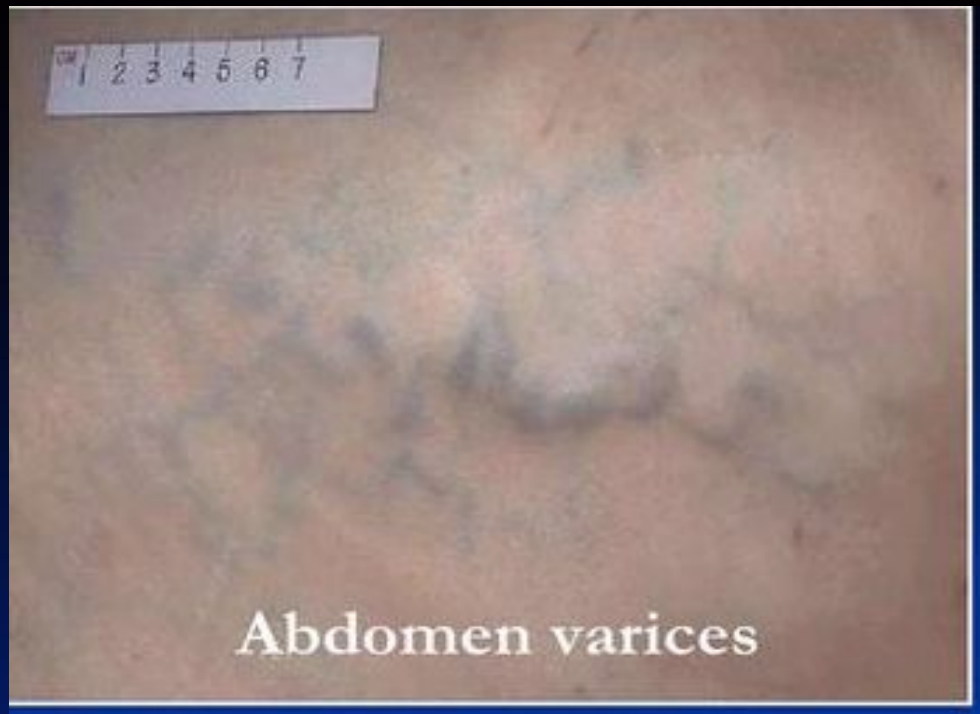


Ascites

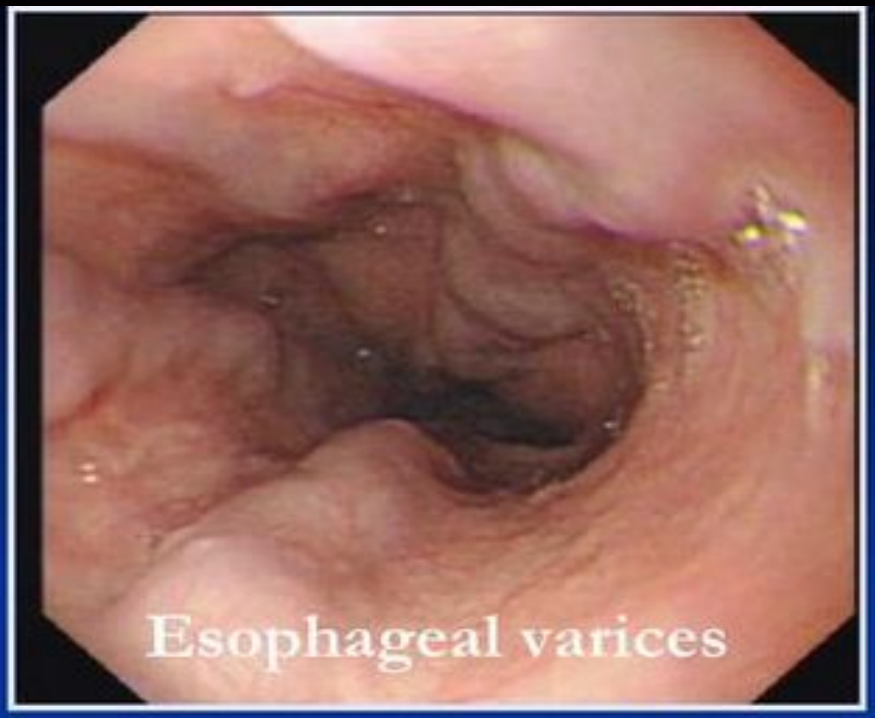


Legs edema



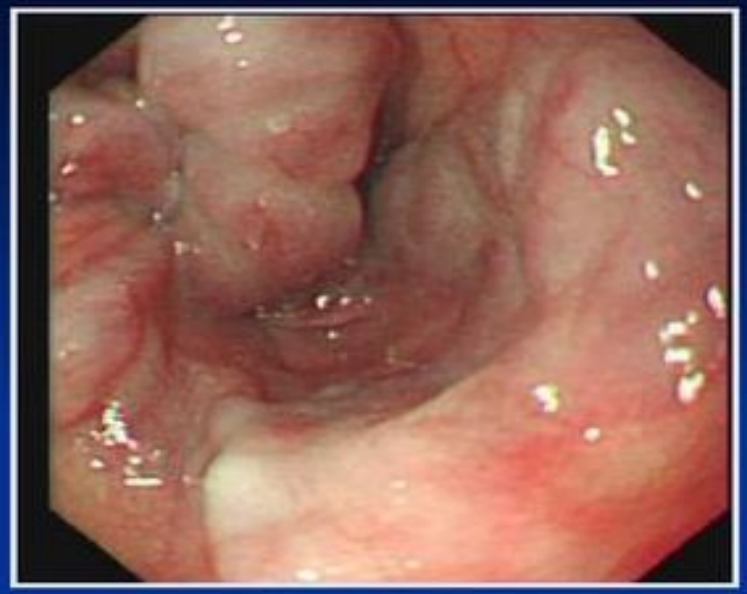


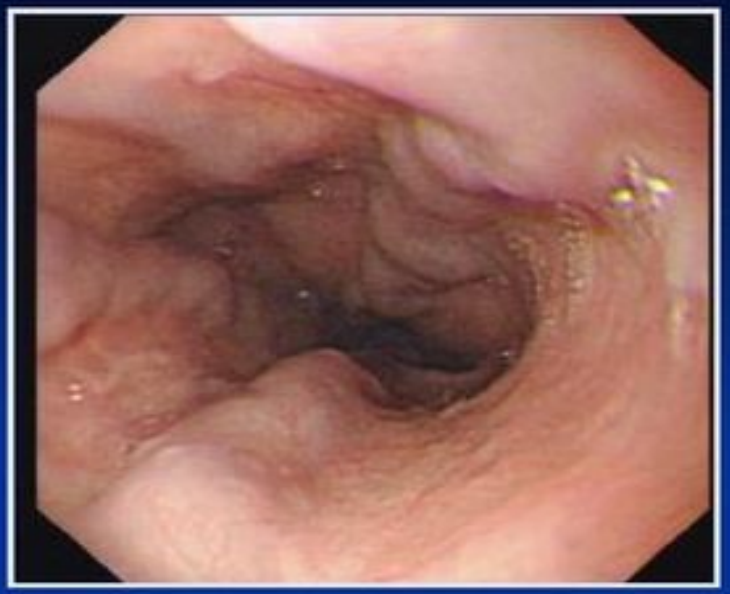
Portal hypertension results from the abnormal blood flow pattern in liver created by cirrhosis. The increased pressure is transmitted to collateral venous channels. Sometimes these venous collaterals are dilated. Seen here is "caput medusae" which consists of dilated veins seen on the abdomen of a patient with cirrhosis of the liver.

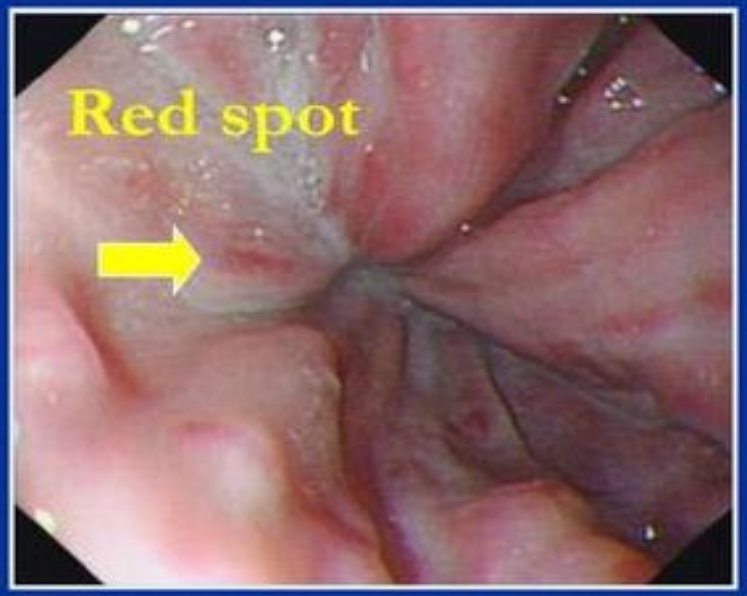


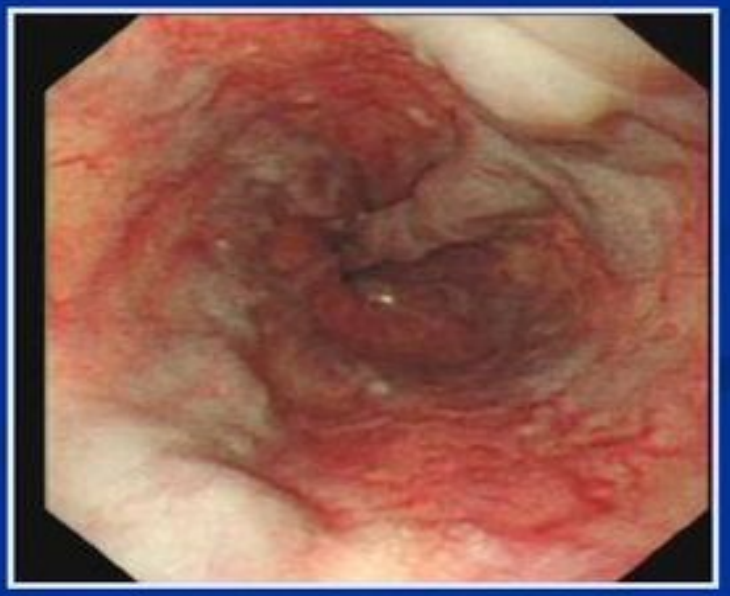
A much more serious problem produced by portal hypertension results when submucosal veins in the esophagus become dilated. These are known as esophageal varices. Varices are seen here in the lower esophagus as linear blue dilated veins. There is hemorrhage around one of them. Such varices are easily eroded, leading to massive gastrointestinal hemorrhage

Esophageal varices

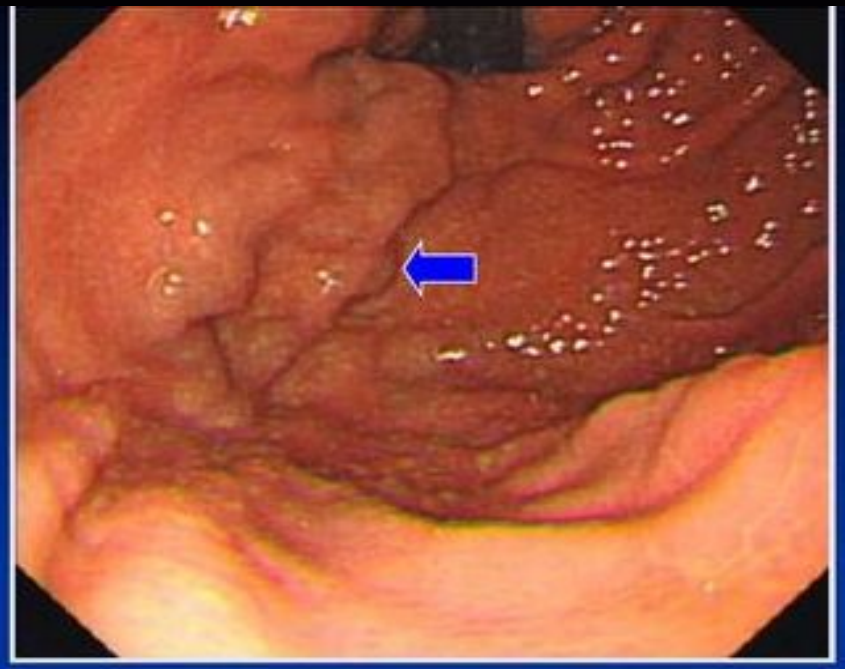


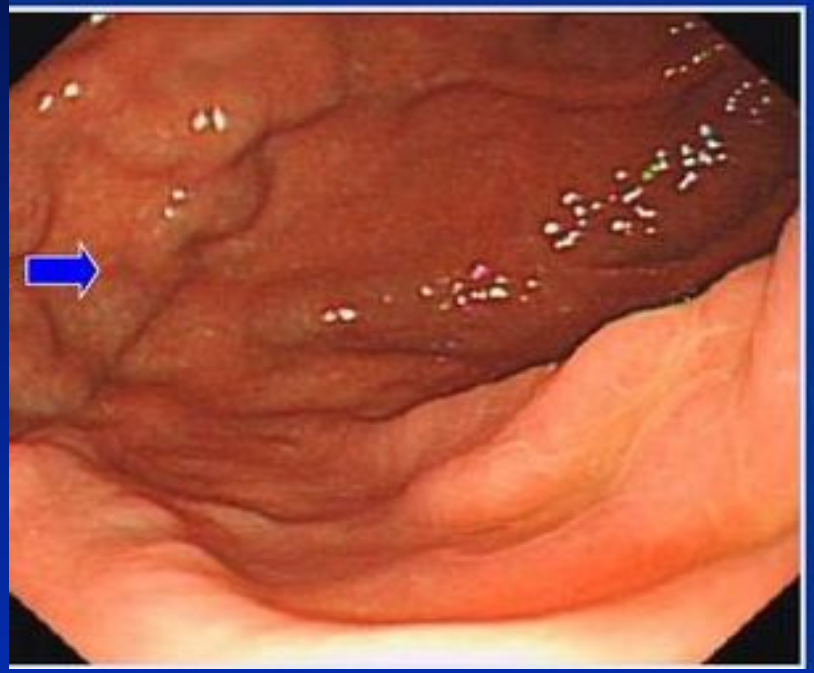


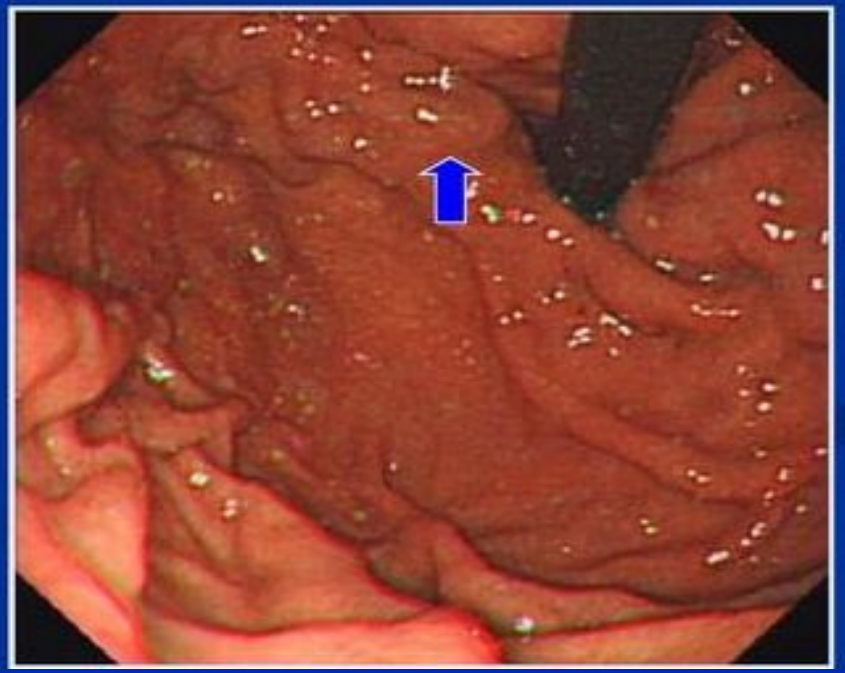




Gastric varices







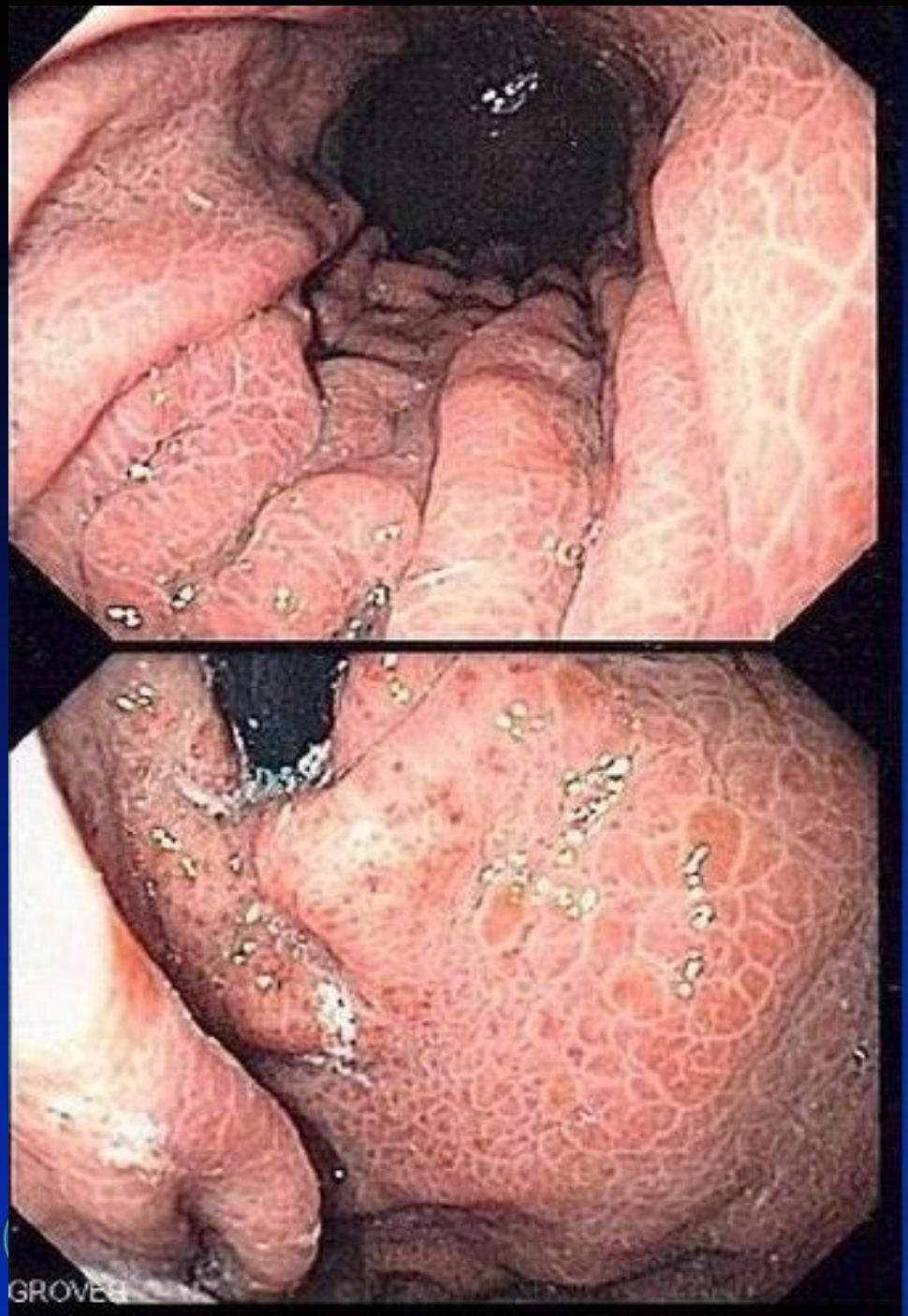


Image of portal hypertensive gastropathy seen on endoscopy of the stomach. The normally smooth mucosa of the stomach has developed a mosaic like appearance, that resembles snake-skin

Autoimmune Hepatitis

-Characterized by hepatic inflammation with plasma cells & fibrosis
-Generally a disease of young women but can occur in either gender at any age
-25% od cases present as an acute attack of hepatitis or follow a viral illness i.e. Hep A, Epstein-Barr infection,or measles or exposure to a drug or toxin
-CM: multiple spider nevi, acne, hirsutism, hepatomegaly
-Extrahepatic manifestations: arthritis, thyroiditis, nephritis, ulcerative colitis, & Coombs' positive Hemolytic anemia

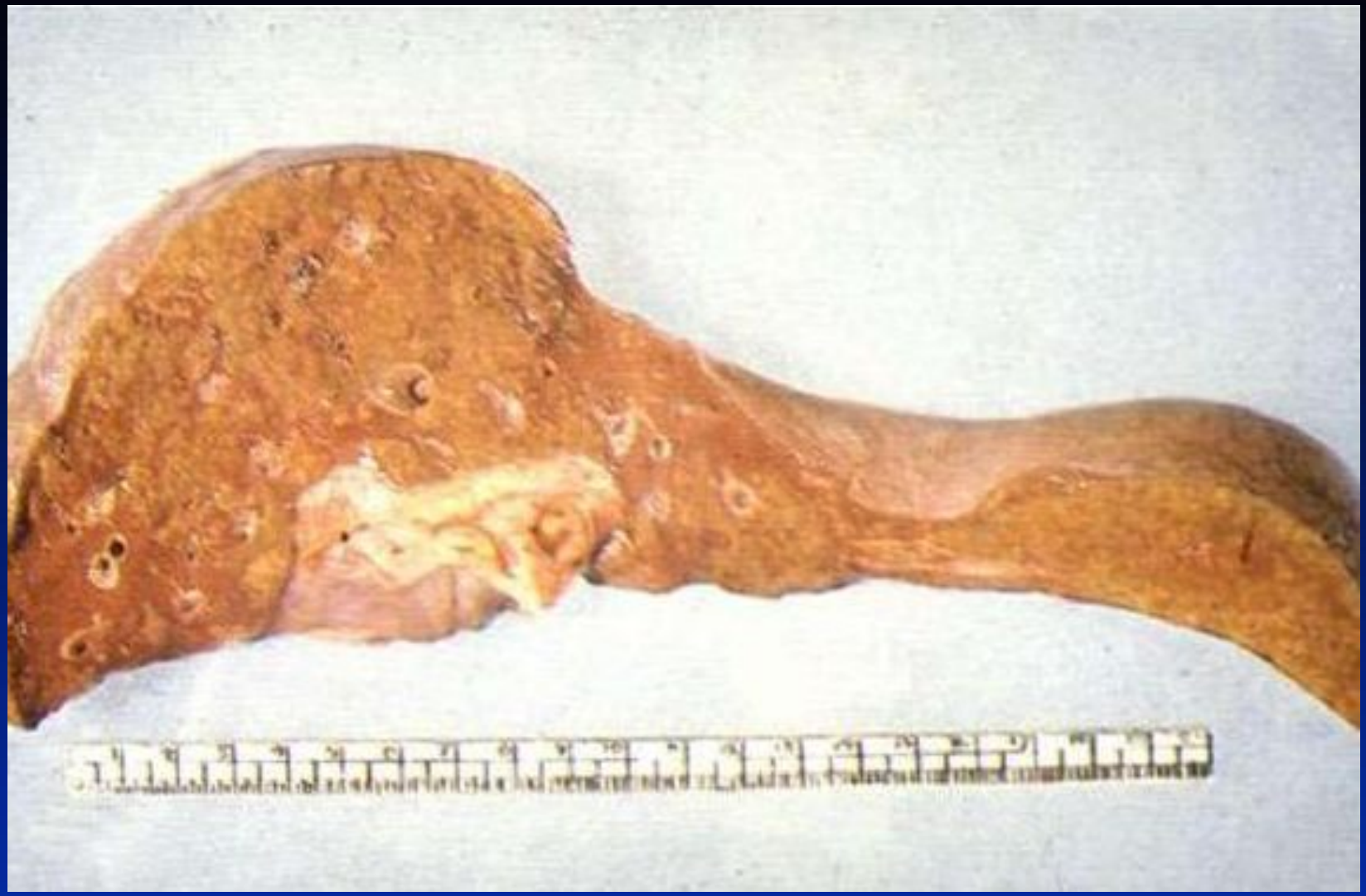
Hepatic Encephalopathy

-Occurs with profound liver failure and may result from the accumulation of ammonia and other toxic metabolites in the blood

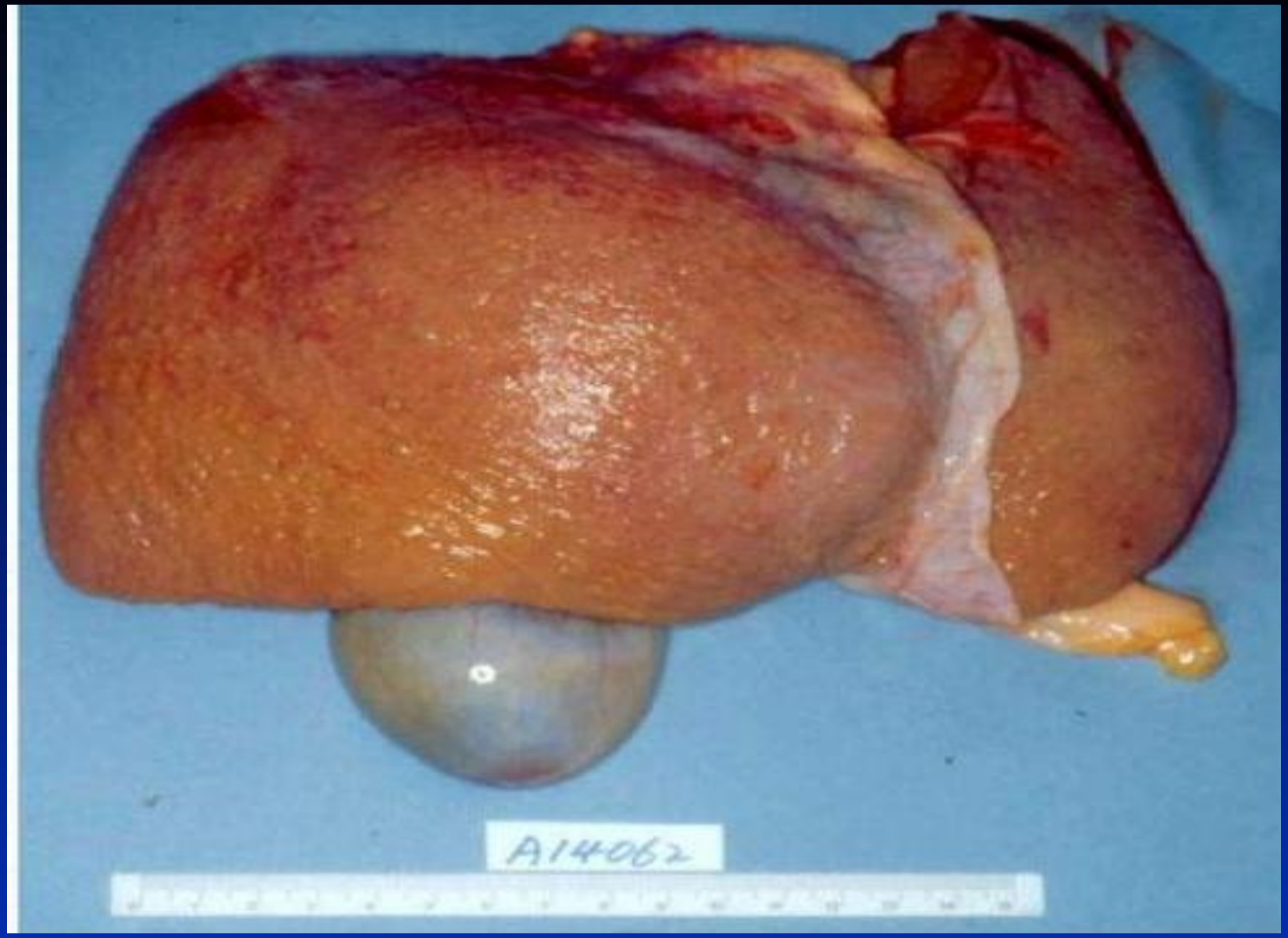
Hepatic coma

-most advanced stage of heaptic encephalopathy
- cause: false neurotransmitter but the exact mechanism is not fully understood

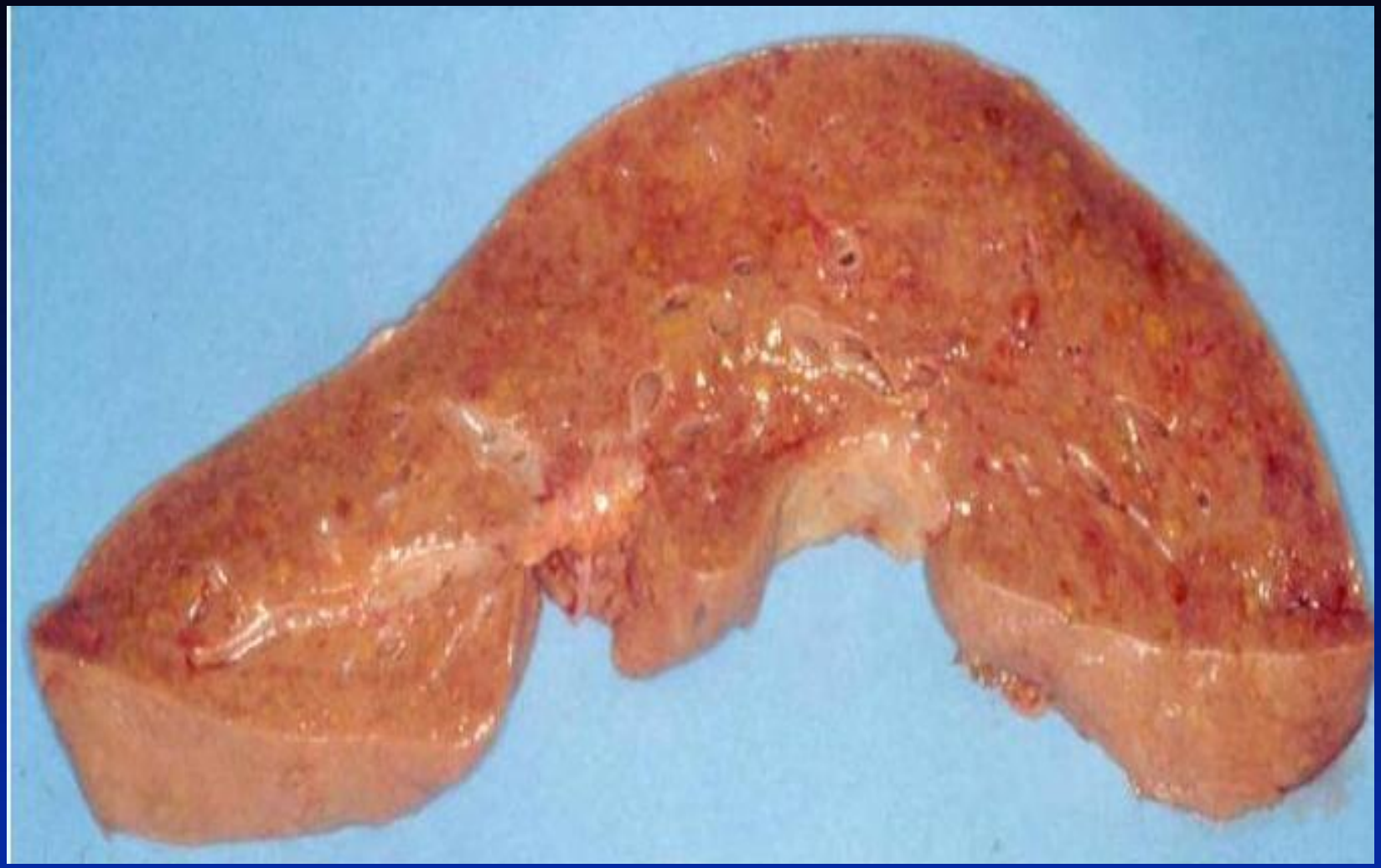
Fulminant Hepatitis:



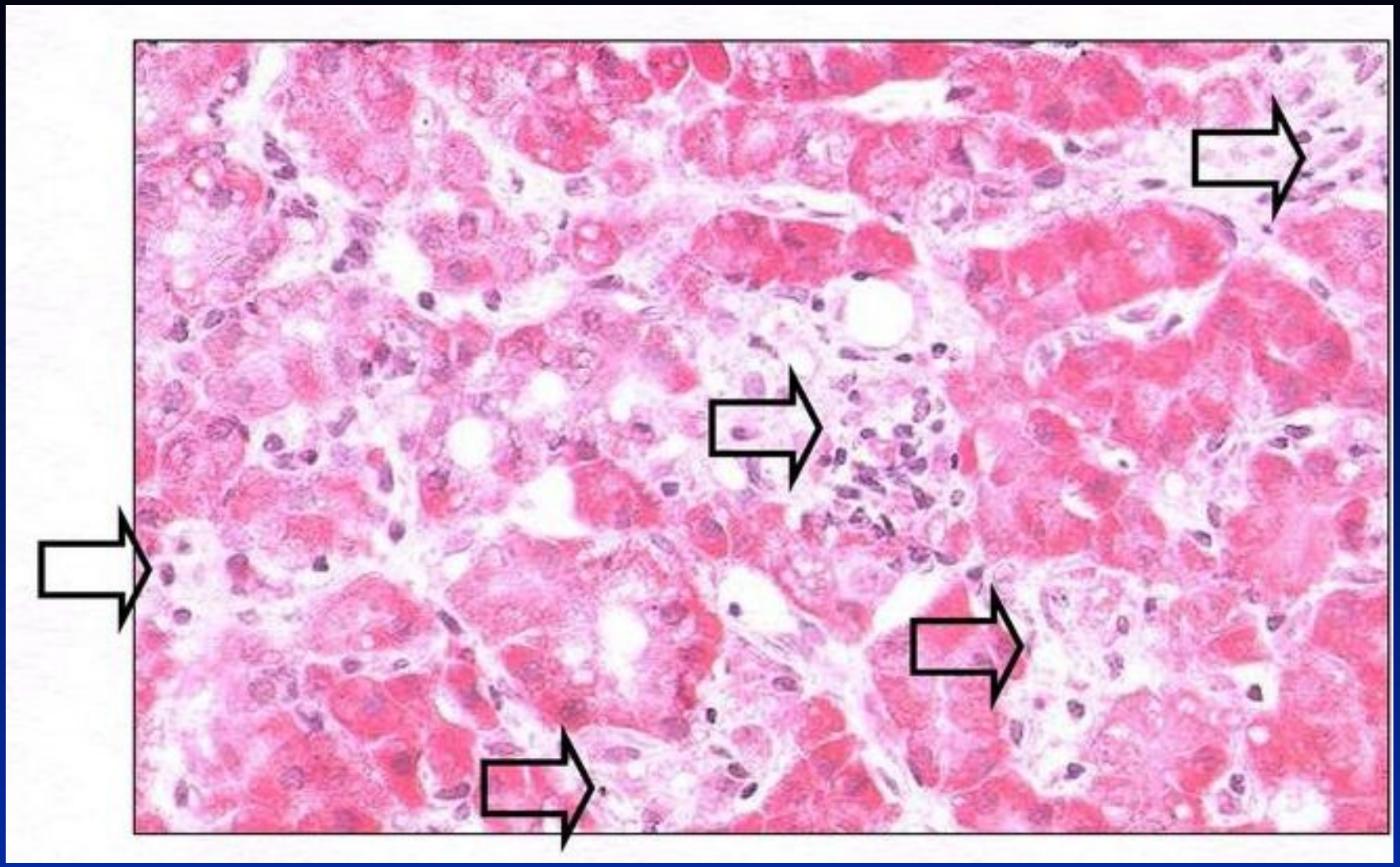
Fulminant Hepatitis:



Fulminant Hepatitis:



Fulminant Hepatitis:confluent necrosis(→).



The differential diagnosis of viral hepatitis is necessary to perform with such diseases

- **leptospirosis, yersiniosis,**
- **mononucleosis, malaria,**
- **mechanical and hemolytic jaundice,**
- **toxic hepatoses and others.**

Treatment

MILD COURSES

- Bed rest
- Dietary regimen is the basis of the therapy of viral hepatitis too. Table №5 is recommended according to Pevzner.
- Sorbents (silix, polisorbs, enterosgel, etc.).
- Enzymes (festal, mizim, pancreatini, etc.).
- Hepatoprotectors
- Duphalac

MODERATE COURSES

- **Bed rest**
- **Dietary regimen is the basis of the therapy of viral hepatitis too. Table №5 is recommended according to Pevzner.**
- **Sorbents (silix, polisorbs, enterosgel, etc.).**
- **Enzymes (festali, mizimi, pancreatini, etc.).**
- **Hepatoprotectors (carsil, legalon, etc.)**
- **Desintoxication infusive therapy (of 5% Glucose 200,0-400,0 ml, 5% solution of ascorbinic acid 10,0-15,0 ml; Reosorbilacti) 5-7 days.**
- **Duphalac**

SEVERE COURSES

- **Bed rest**
- **Dietary regimen is the basis of the therapy of viral hepatitis too. Table №5 is recommended according to Pevzner.**
- **Sorbents (silix, polisorbs, enterosgel, etc.).**
- **Enzymes (festali, mizimi, pancreatini, etc.).**
- **Hepatoprotectors (carsil, legalon, etc.)**
- **Desintoxication infusive therapy (of 5% Glucose 200,0-400,0 ml, 5% solution of ascorbinic acid 10,0-15,0 ml; Reosorbilacti) 5-7 days.**
- **Duphalac**
- **Hemostatic therapy (vicasol, aminocapronic asid, dicinoni , etc)**
- **Corticosteroids therapy**

- **Inhibitors of proteolytic ferments (trasilol, hordox or contrical)**
- **donor's albumin – 400,0-500,0 ml**
- **antibiotic therapy**


Thank you