POLTAVA MEDICAL
STOMATOLOGICAL UNIVERSITY

Liver Cirrhosis

Department of Internal medicine №1

Liver Cirrhosis
(from greec. Kirrhos – orange or tan)

Chronic, progressive diffuse liver disease, characterized by:

- lesions of cytoarchitectonics of the hepatic lobe
- a significant decrease in the number of functioning hepatocytes
- increasing fibrosis
- restructuring of the normal structure of the parenchyma and the vascular system of the liver
- formation of regeneration nodes
- development of hepatic failure and portal hypertension

Introduction

- Cirrhosis is common end result of many chronic liver disorders.
- Diffuse scarring of liver – follows hepatocellular necrosis of hepatitis.
- Inflamation – healing with fibrosis - Regeneration of remaining hepatocytes form regenerating nodules.
- Loss of normal architecture and function.

Cirrhosis insidiousness

- Cirrhosis is a severe disease in which normal liver tissue is replaced with scar (connective) tissue. As a result, the human body becomes defenseless against toxic substances that the liver neutralizes, the digestion and absorption of food are disrupted, and the production and accumulation of vital substances (proteins, fats, carbohydrates, hormones) are acute reduced.

The most dangerous are alcohol-viral cirrhosis. They most often developed into liver cancer and are especially severe.

Epidemiology

- Frequency of liver cirrhosis (LC) -1-3% among all deceased
- Men are sick almost 2 times more often than women
- The average age of men is 45 years, women is 55

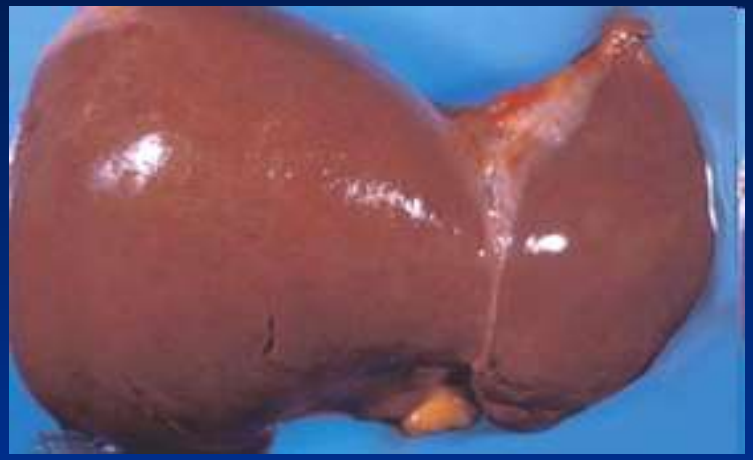
According to the World Health Organization, drinking even small doses of alcohol (more than 0.33 liters of beer per day) is an abuse.

Alcoholic liver disease development depends on the alcohol quantity in volume:

- 25 ml of vodka
- = 100 ml of wine
- = 200 ml of beer

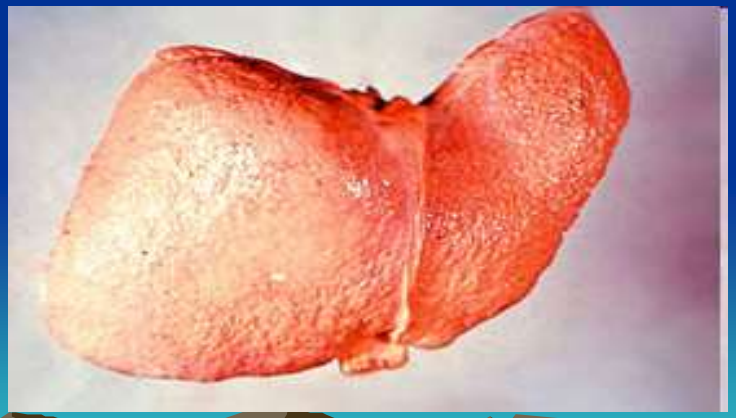
Cirrhosis develops gradually. Alcoholic LC develops over 5-7 years with daily alcohol consumption

Liver



Normal liver

Hepatitis Liver



The initial stage of liver cirrhosis

The final stage of liver cirrhosis

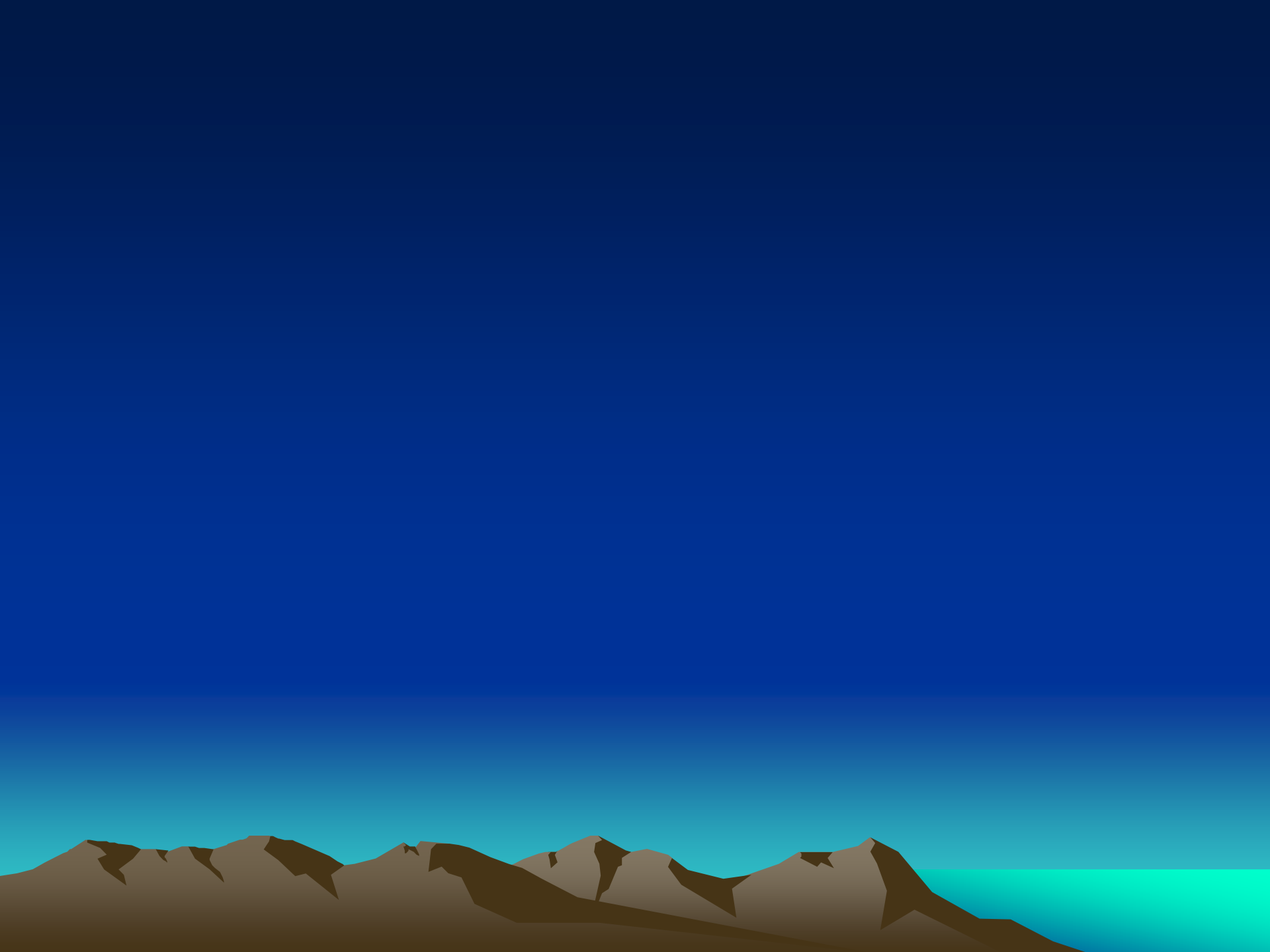
Liver Functions:

- **Metabolism** – Carbohydrate, Fat, Protein
- **Secretory** – bile, Bile acids, salts, pigments
- **Excretory** – Bilirubin, drugs, toxins
- **Synthesis** – Albumin, coagulation factors
- **Storage** – Vitamins, carbohydrates etc.
- **Detoxification** – toxins, ammonia, etc.

Classification of cirrhosis

Etiology

- Viral - (hepatitis B, C, D, G viruses)
- Alcoholic
- Drug induced
- Toxic
- Metabolic disorder (Wilson's disease, hemochromatosis)
- Damage to the biliary tract (primary and secondary biliary disorders)
- Stagnant
- Cryptogenic



Classification of cirrhosis

According to the morphological picture:

- Micronodular (with a diameter of nodes up to 3 mm)
- Macronodular (with a diameter of nodes greater than 3 mm)
- Mixed

According to clinical signs:

- Stage of the disease (according to Child-Pugh criteria)

Initial, expressed clinical manifestations, terminal

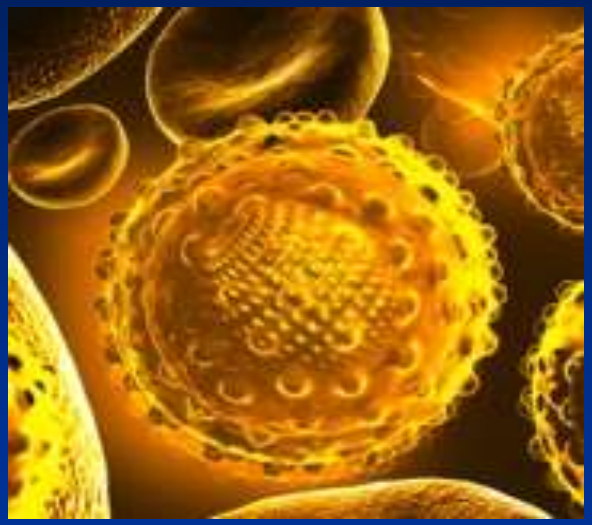
Process Activity: Active, Inactive

Complications: portal hypertension, bleeding from varicose dilated veins, hepatic encephalopathy, bacterial peritonitis, portal vein thrombosis, hepatorenal syndrome, hepatocellular carcinoma

Etiology

- Hepatities B, C, D,..viruses
- Alcohol
- Metabolic disorders (hereditary hemochromatosis, Konovalov-Wilson disease, alfa1-antitrypsin deficiency, cystic fibrosis, etc.)
- Billiary disorders (extra- and intrahepatic obstruction, including primary biliary cirrhosis and primary sclerosing cholangitis)
- Violation of the venous outflow from the liver (Budd-Chiari syndrome, veno-occlusive disease, right ventricular heart failure)
- medicines, toxins, chemicals (methotrexate, amiodarone)
- immune disorders
- Other causes (syphilis, schistosomatosis, sarcoidosis…)

Hepatitis C and B viruses



Hepatitis C virus multiplies in the liver and circulates in the blood of infected people

Hepatitis B is a dangerous viral disease transmitted by hematogenous pathway.

Morphological classification of cirrhosis

Micronodular (alcoholic, primary and secondary biliary cirrhosis, hemochromatosis)

Macronodular(viral, α1-antitrypsin deficiency)

Mixed

Мелкоузловой цирроз

Крупноузловой цирроз

Pathogenesis

- It develops slowly (over the years), with an unfavorable course for 0.5-1.5 years.
- Alcoholic cirrhosis develops in 7-8 years with daily use by men - 60 ml, women -30-40 ml of ethanol
- Alcohol is the most common hepatotropic poison

LC is very quickly formed in the AIH

- 6-10% in patients with HBV
- 50-60% in patients with HCV

Pathogenesis

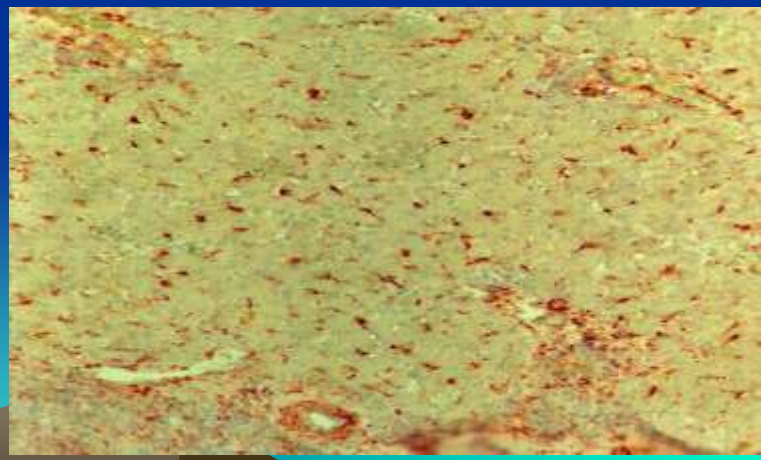
- Hepatocyte injury leading to necrosis.
  - **Alcohol, virus, drugs, toxins, genetic etc..**
- Chronic inflammation - (hepatitis).
- Bridging fibrosis.
- Regeneration of remaining hepatocytes Proliferate as round nodules.
- Loss of vascular arrangement results in regenerating hepatocytes ineffective.

Pathogenetic mechanisms

- 1. The action of the etiological factor
- 2. Activation of HSC (Ito cells), their proliferation and chemotaxis
- 3. Violation of the blood supply to the liver parenchyma (nodal regeneration)
- 4. The inclusion of immune and autoimmune mechanisms of hepatocytes cytolysis

Ito Cell

- Stellate liver cell, fat storage cell, lipocyte, eng. Hepatic Stellate Cell, HSC, Cell of Ito, Ito cell) - pericytes contained in the perisinusoidal space of the hepatic lobule, capable of functioning in two different states - calm and activated. Activated Ito cells play a major role in fibrogenesis - the formation of scar tissue with liver damage.



Clinical Syndromes in Cirrhosis

- ✓ **Astheno-vegetative**
- ✓ **Dyspeptic**
- ✓ **Cytolytic**
- ✓ **Immunoinflammatory**
- ✓ **Portal hypertension**
- ✓ **Cholestatic**
- ✓ **Hypersplenism**
- ✓ **Hepatic cell failure**

Clinical Syndromes

• Asthenic (fatigue, weakness, depression)	90%
• Hepato-lienal	80-90%
• Pain in the right hypochondrium	80
• Dyspeptic (nausea, vomiting, weight loss, flatulence)	60-80
• Jaundice	60-70
Ascites	50-60
• Hemorrhagic (increased bleeding, nasal,	
• gastric bleeding, hemorrhagic diathesis)	30-40
•	
• “Hepatic signs” - “hepatic palms”, telangiectasia	30-40
• Subfebrile condition	30-50
• Endocrine disorders (gynecomastia, feminization, violation	
• libido, hyperaldosteronism)	30-40
• Skin itching	30
• Hepatic encephalopathy	20-30%

Clinical Features

- Hepatocellular failure.
  - Malnutrition, low albumin and clotting factors, bleeding.
  - Hepatic encephalopathy.
- Portal hypertension.
  - Ascites, Porta systemic shunts, varices, splenomegaly.

Cirrhosis Features

- Hepatocellular Failure
- Portal obstruction, Porta systemic shunts…
- Portal hypertension, Splenomegaly
- Jaundice, Coagulopathy, hypoproteinemia, toxemia, Encephalopathy

Hepatic cell failure

- Violation of inactivation, binding and elimination of toxic substances
- Violation of the synthetic function of the liver

Clinical manifestations of hepatic cell failure

Hyperestrogenemia

- Teleangiectasias ("spider veins")
- Palmar erythema (“hepatic palms”)
- Gynecomastia, amenorrhea
- Alopecia

Clinical manifestations of hepatic cell failure

The entry into the bloodstream of toxic protein metabolism products (ammonia, indole, skatol, etc.)

- Cerebro-toxic effect (encephalopathy)
- Acidosis
- Increased hypokalemia

Clinical manifestations of hepatic cell failure

Decreased protein-synthetic function

- Hypoalbuminemia
- Hypocholesterolemia
- β-Lipoprotein Decrease
- Hypoprothrombinemia
- Hypofibrinogenemia
- Reduced angiotensin
- Impaired binding to plasma proteins of iron, bilirubin

Clinical manifestations of hepatic cell failure

Violation of intestinal pyrogens neutralization

- Fever
- Leukocytosis

Portal hypertension

- Venous enlargement

  inflow to the spleen - Splenomegaly
- Reticulolymph-hyperplasia

  histiocytic elements – Splenomegaly+Hypersplenism

  Hypersplenism:
- thrombocytopenia
- leukopenia
- anemia

Portal hypertension

Venous collateral veins with vena cava inferior development

Varicose veins of the esophagus, stomach, intestines, hemorrhoidal, anterior abdominal wall

Complications of Cirrhosis

- Ascites
- Spontaneous bacterial peritonitis
- Varicose veins bleeding
- Hepatic encephalopathy
- Hepatocellular carcinoma
- Hepatorenal syndrome

Ascites

- Portal hypertension
- Increased extracellular osmolarity-increased secretion of vasopressin, ADH
- Decreased liver albumin synthesis - decrease in plasma colloid osmotic pressure
- Decreased inactivation of aldosterone in the liver+increased aldosterone production - Secondary hyperaldosteronism
- Decreased renal blood flow- impaired renal function
- Increased liver lymph production

Spontaneous bacterial peritonitis

- **The most common infectious complication of cirrhosis (8-32%)**
- **Total mortality during the year after the first episode 61-78%**
- **Pathogenesis: penetration of bacteria into the abdominal cavity by translocation and hematogenous pathway against the background of a decrease in nonspecific resistance of the body**
- **The main risk factor is low protein content in ascitic fluid.**
- **The main causative agent is Escherichia coli**
- **Clinical picture: diffuse abdominal pain, fever, vomiting, diarrhea, intestinal paresis, septic shock**
- **Diagnosis: ascitic fluid culture**
- **Treatment: III generation cephalosporins (cefotaxime) or amoxicillin + clavulanic acid**
- **Prevention: fluoroquinolones, trimethoprimsulfamethoxazole**

Hepatoma - a malignant tumor of the liver - often develops in patients suffering from cirrhosis, especially after hepatitis B.

Hepatorenal syndrome

- **Progressive renal failure, developing on the background of portal hypertension**
- **Pathogenesis: pronounced spasm of the renal arteries, leading to a decrease in perfusion of mainly cortical matter, which leads to a decrease in glomerular filtration rate**
- **Diagnostic criteria:**
- **increased creatinine> 150 mcmol/l**
- **GFR <40 ml / min**
- **the absence of other causes of renal failure**
- **lack of improvement in renal function after discontinuation of diuretics and intravenous compensation of plasma volume**
- **The forecast is unfavorable**
- **(75% of patients die within 3 weeks, 90% within 8 weeks)**

Hepatic encephalopathy

- **Neuropsychiatric disorders that develop with hepatic cell failure and / or porto systemic blood bypass**
- **Pathogenesis: 1) “glia hypothesis”; 2) ammonia hypothesis; 3) the mercaptan hypothesis; 4) amino acid imbalance**
- **Clinical manifestations: sleep rhythm disturbances, decreased attention, irritability, handwriting changes, disorientation in time and space, asterixis, lethargy, inappropriate behavior, coma**
- **Treatment: 1) a decrease in the formation of ammonia in the intestine (lactulose, ornithine-aspartate, ornithine-α-ketoglutarate); 2) a decrease in inhibitory processes in the central nervous system (flumazenil); 3) amino acids, zinc**

COMA

- Portocaval Bypass - Shunt coma
- Hepatic cell failure - Hepatic cell coma

Factors provoking the development of hepatic coma

- **1. Bleeding from varicose veins of the esophagus, stomach (anemia, hypoproteinemia, absorption of blood breakdown products in the stomach)**
- **2. Taking hepatotoxic drugs (psychotropic drugs, narcotic analgesics, antibiotics, hypoglycemic drugs)**
- **3. Intercurrent infection**
- **4. Stress, surgery**
- **5. Violation of the diet (ammonia intoxication)**
- **6. Drinking alcohol**
- **7. Violation of the water-electrolyte balance (overdose of diuretics, excessive removal of ascitic fluid, profuse vomiting and diarrhea)**

Complaints:

- Weakness, fatigue, decreased performance
- Pain and a feeling of heaviness in the right hypochondrium, bloating, nausea, vomiting, diarrhea, poor tolerance of fatty foods and alcohol
- Nosebleeds
- Fever
- Sleep disturbance, irritability
- Sexual dysfunction

Anamnesis:

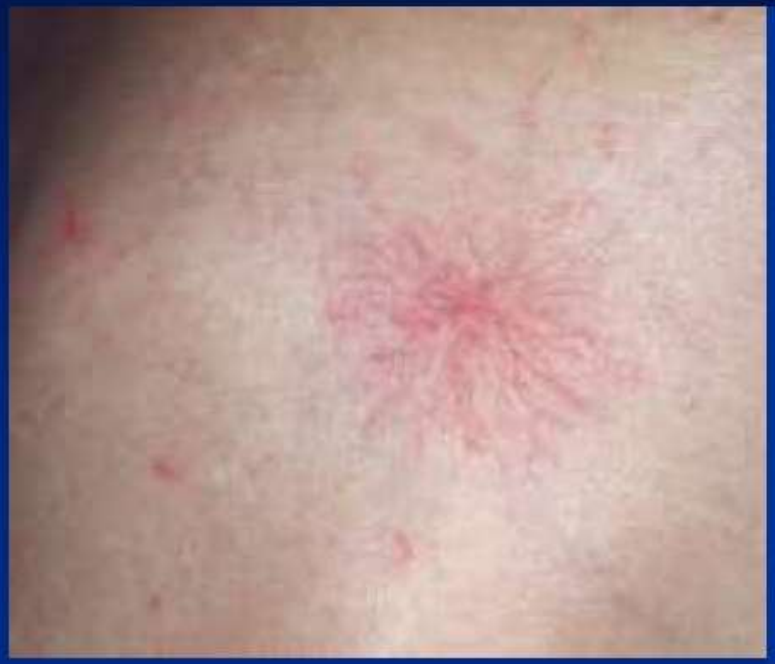
- Alcohol abuse
- Data on the transferred hepatitis (jaundice)
- Hepatotropic Poisoning
- Reception of hepatotoxic drugs (anti-tuberculosis, psychotropic, etc.)

Objectively

- “Hepatic” signs: telangiectasias, palmar erythema, gynecomastia, impaired “secondary” hair growth
- Signs of an alcoholic disease: Dupuytren's contracture, enlargement of the parotid glands, muscle atrophy, polyneuritis
- Jaundice
- Hemorrhages, petechiae

Objectively

- Ascites
- Peripheral edema
- Venous collaterals (“jellyfish head”, expansion of hemorrhoidal veins)
- Hepatomegaly
- Splenomegaly
- Decreased attention, loss of fine motor skills (change in handwriting), drowsiness, tremor

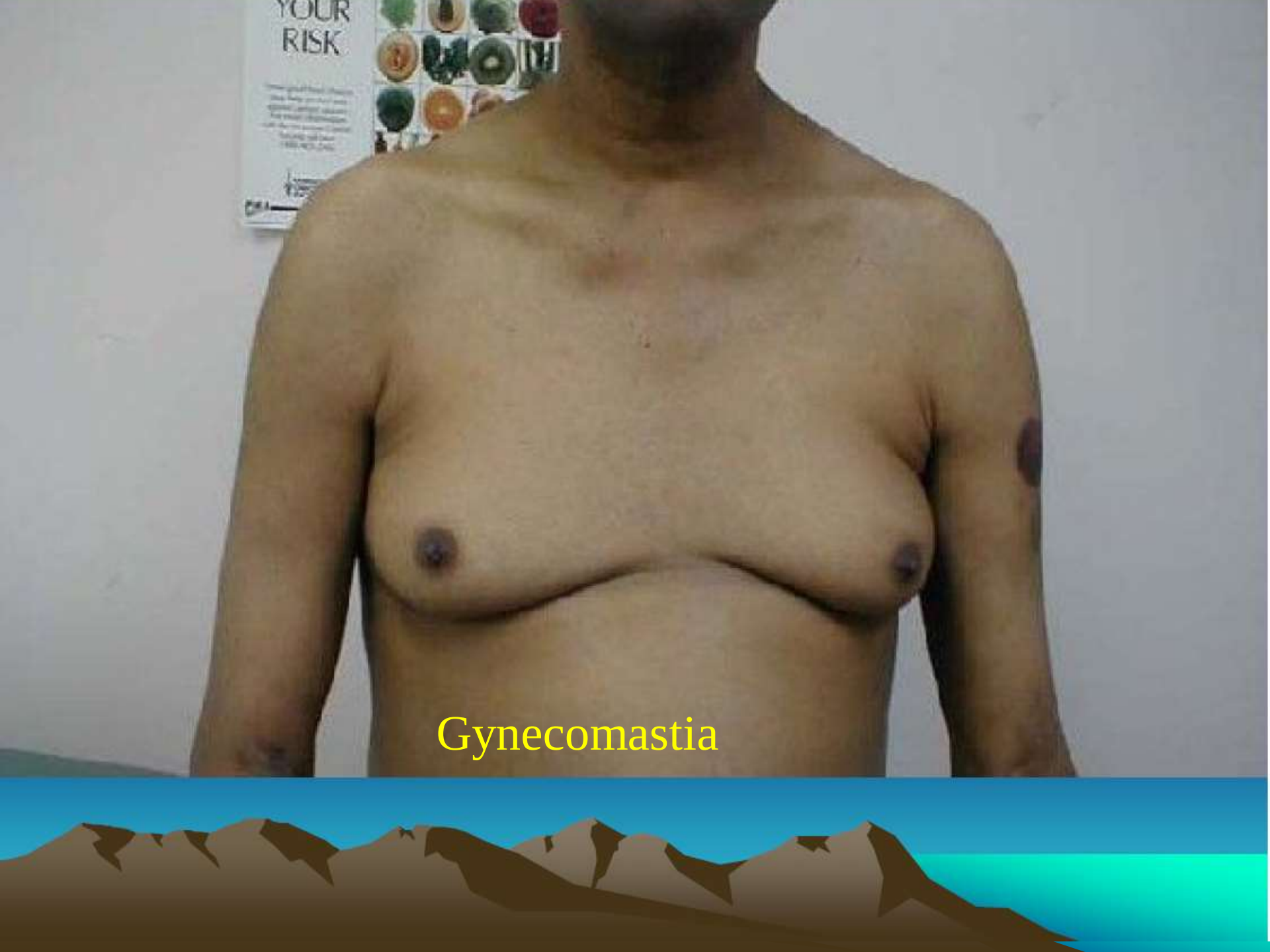


Teleangioectasia

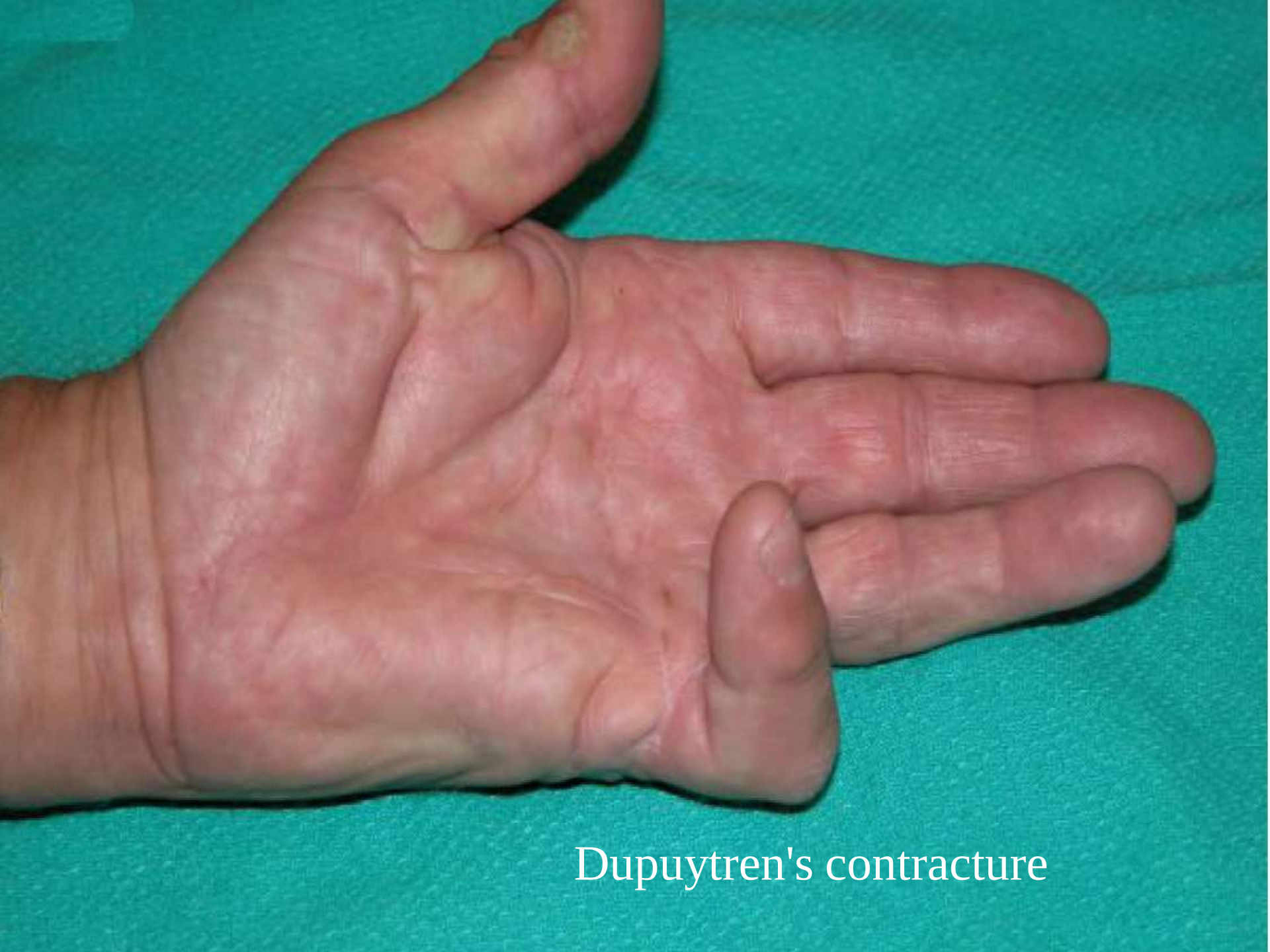
Xantoma

Xanthelasma


Palmar erythema: the skin of the palm is thinned, shiny, hyperemic.

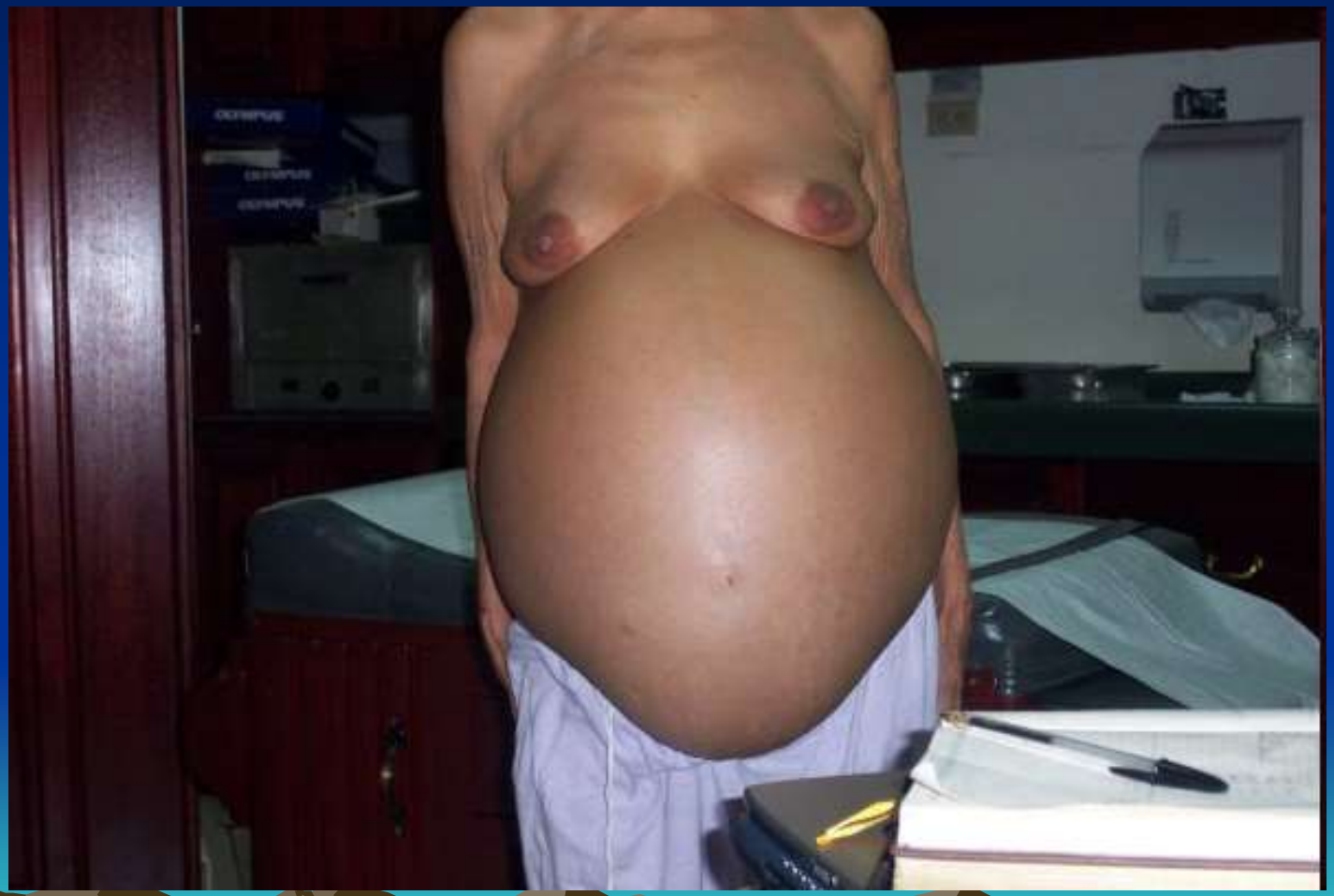


Gynecomastia

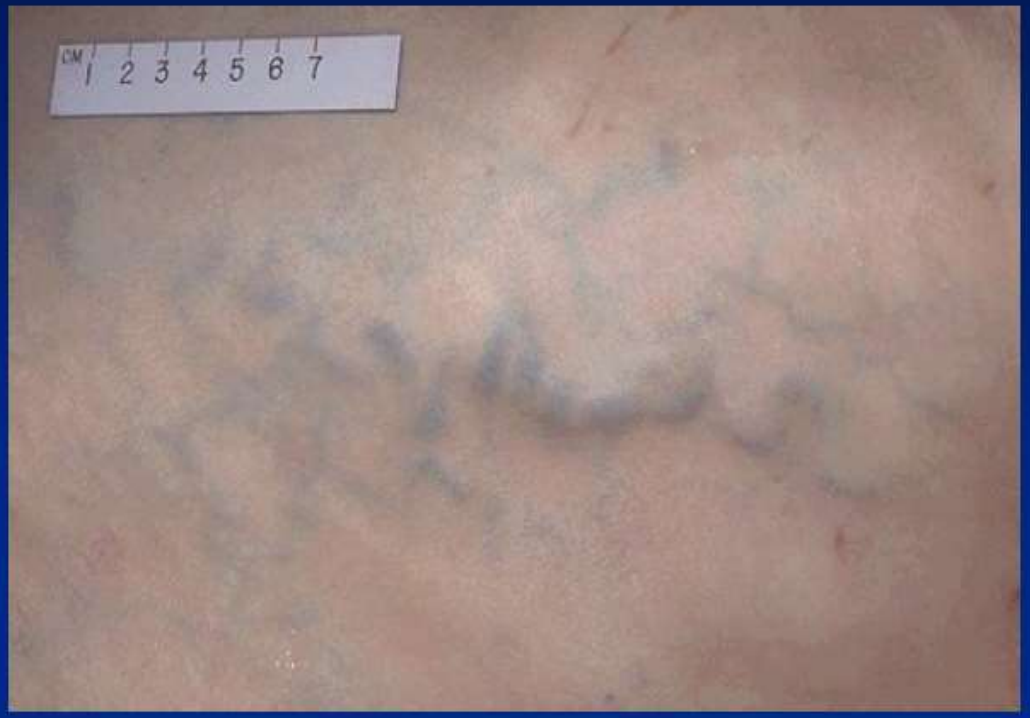


Dupuytren's contracture

Ascites

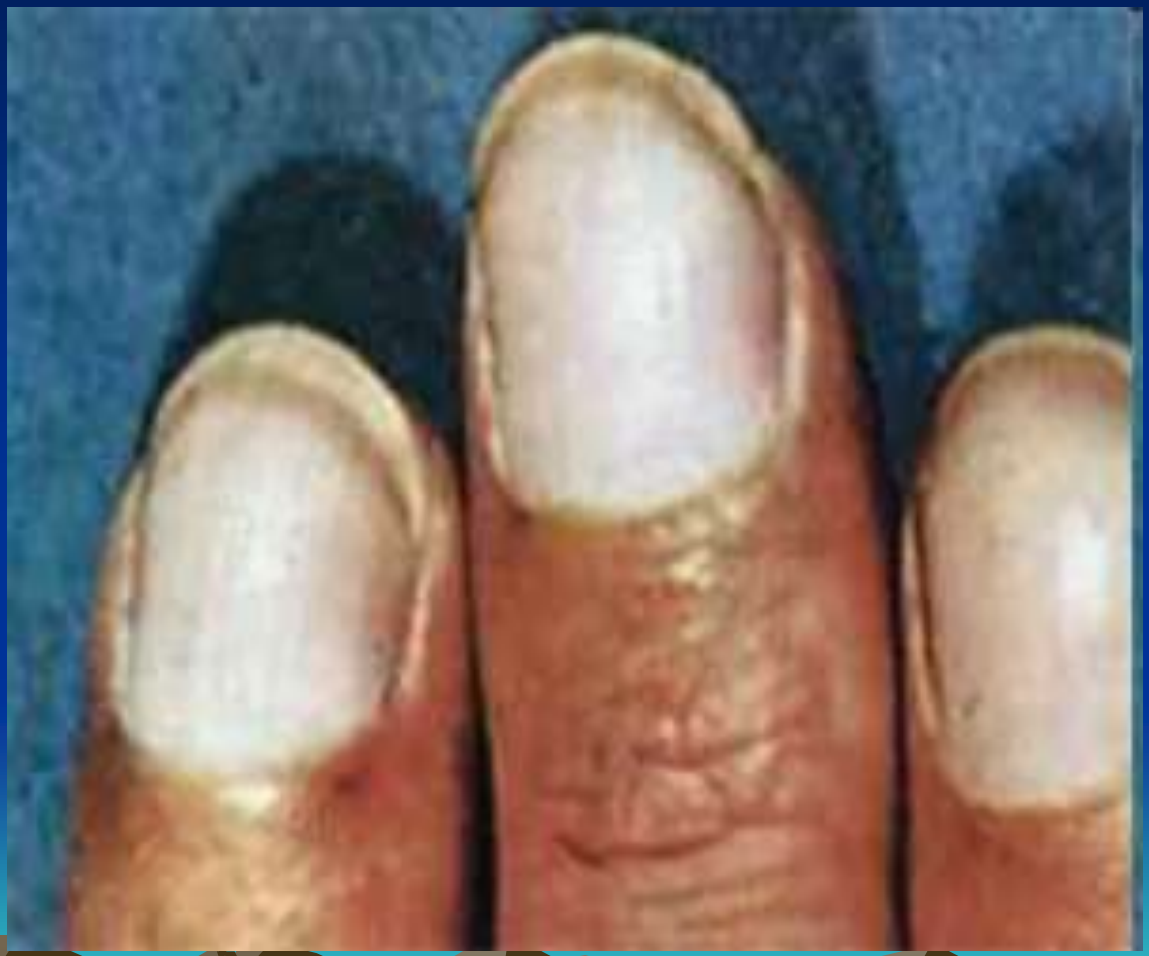


Живот при декомпенсированном циррозе печени: увеличение живота за счет асцита, бугристость кожи, обусловленная отеком дермы, расширение подкожных вен.



“jellyfish head”

White nails, total leukonychia



Laboratory data

General blood analysis:

- Anemia
- Leukocytosis / Leukopenia
- Thrombocytopenia
- ESR increase

General urine analysis:

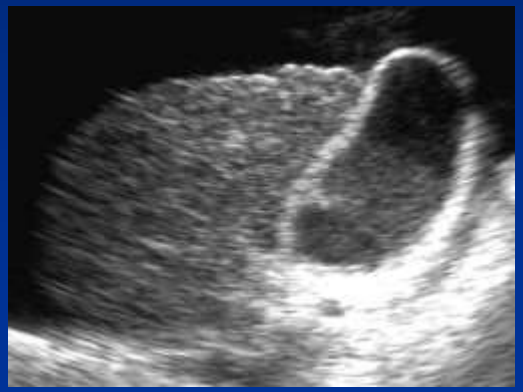
- Urobilin level increase
- Proteinuria

General analysis of feces: steatorrhea

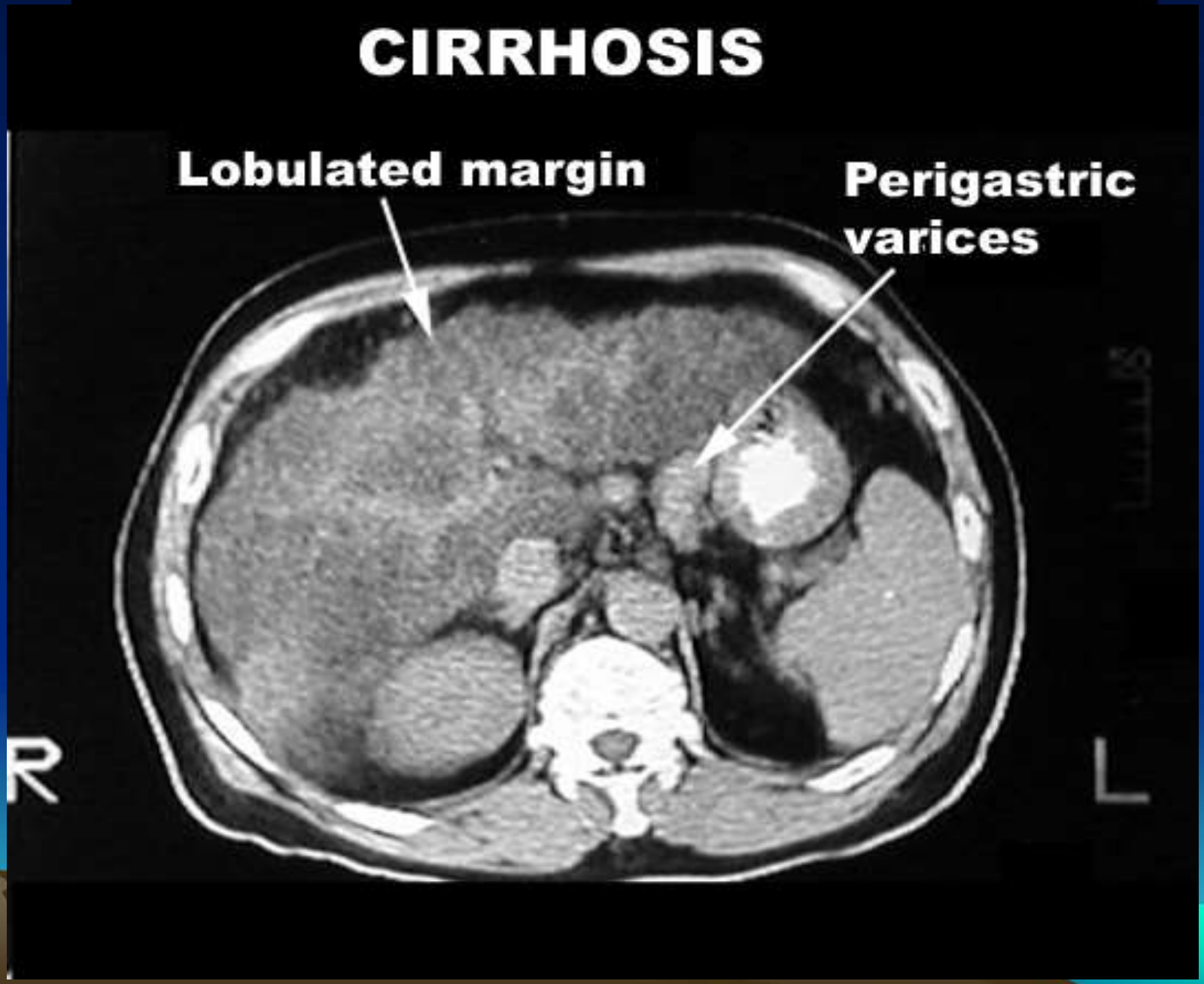
Instrumental Studies

Ultrasound / CT scan of the abdominal cavity:

- Enlargement / reduction of the liver
- Uneven contours
- Structure heterogeneity
- Signs of portal hypertension
- Splenomegaly
- Abdominal fluid



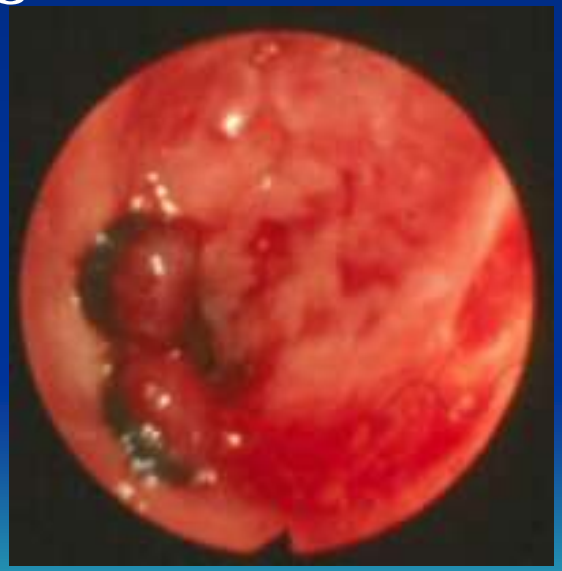
CIRRHOSIS



Instrumental Studies

Endoscopy, radiography of the esophagus, stomach:

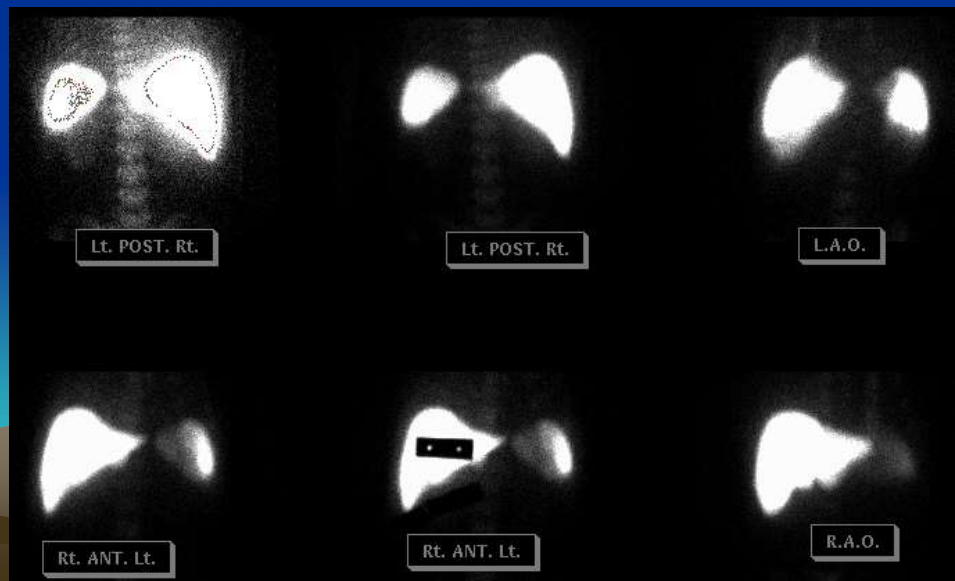
- *Varicose veins of the esophagus and cardiac part of the stomach*



Instrumental Studies

Liver scintigraphy:

- Heterogeneous accumulation and delay in the elimination of pharmaceuticals in the liver
- Intensive accumulation of pharmaceuticals in the spleen

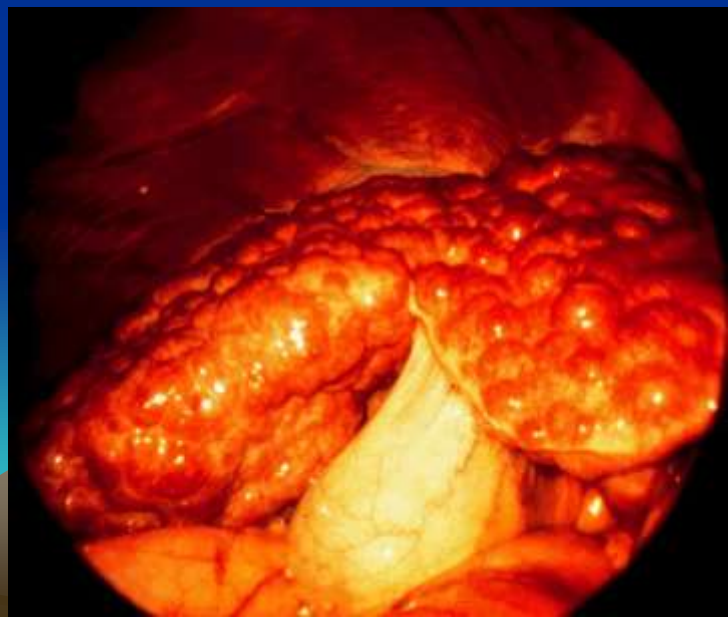


Liver biopsy

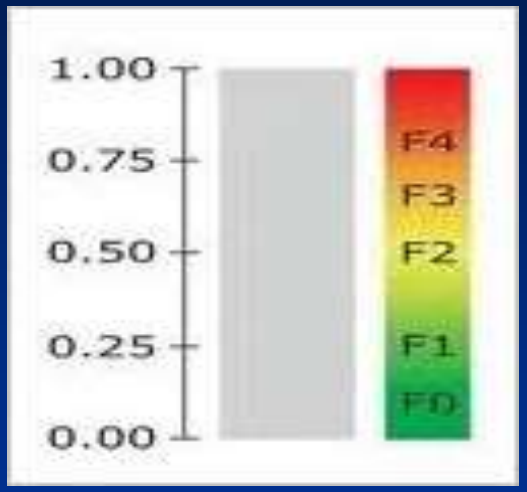
is the main method for verifying liver cirrhosis.

Liver biopsy

- Puncture
- Laparoscopy with targeted biopsy



Modern diagnostics



Ultrasensitive test: HBV and HCV confirmation of the virus carrier of HCV, HBV.

Fibrotest
fibrosis determination method
Determining the early stages of the disease when there are no morphological changes ...

Liver fibroscan (elastometry)
apparatus for non-invasive diagnosis of the liver, determines the degree of fibrosis.

The predominant syndromes in cirrhosis of alcoholic etiology

With subcompensation:

- dyspeptic
- asthenovegetative

With decompensation:

- portal hypertension

The predominant syndromes in cirrhosis of viral etiology

- With subcompensation and decompensation:
- cytolytic
- dyspeptic

Classification of the severity of liver cirrhosis by Child-Pugh

The Pugh-Child score is determined by scoring five clinical signs (indexes) of liver disease. A score of 1, 2, or 3 is given to each index, with 3 being the most severe.

These five clinical signs are:

- total bilirubin: yellow compound in bile from hemoglobin destruction
- serum albumin: blood protein produced in the liver
- prothrombin time, prolongation(s) or INR: time for blood to clot
- ascites: fluid in peritoneal cavity
- hepatic encephalopathy: brain dysfunction

For example:

For example:

- If the ascites result is “none,” that measure would be scored with 1 point.
- If the ascites result is “mild/diuretic responsive,” that measure would be scored with 2 points.
- If the ascites result is “moderate/diuretic refractory,” that measure would be scored with 3 points.

Once scores are available in each of the five clinical measures, all scores are added and the result is the Child-Pugh score.

- So, 5-6 points – A (compensation)
- 7-9 B (subcompensation)
- 10-15 C (decompensation)

Banty Syndrome

Splenomegaly with cirrhosis

- The main clinical manifestations: splenomegaly with severe hypersplenism (anemia or panhemocytopenia, hemorrhagic diathesis); later, the size of the cirrhotically altered liver increases, jaundice appears;
- In the terminal stage, the liver shrinks, free fluid accumulates in the abdominal cavity (ascites), and esophageal-gastric bleeding occurs.
- The course is progressive - anemia, cachexia, and liver failure are increasing.

Diagnostic criteria

- 1. The presence of hepatolienal syndrome (detected clinically and with instrumental diagnosis)
- 2. Small hepatic syndrome
- failure:
- -Disruption of the detoxification function of the liver (telangiectasia, erythema palmaris, impaired drug metabolism, hormonal disorders: impotence, gynecomastia in men, menstrual irregularities in women)
- - Violation of the protein-synthetic function of the liver (increased bleeding, slight bruising, trophic disorders - weight loss, development of cachexia)

Diagnostic criteria

- 3.Ascites
- 4. Big liver syndrome
- insufficiency (hepatic
- encephalopathy, coma) in the terminal
- stages:
- -Nonspecific mental disorders
- the activities
- - Neuromuscular disorders ("clapping tremor")
- - Hepatic breath
- - Respiratory failure (hyperventilation)
- - Changes to the electroencephalogram

Diagnostic criteria

- 5. Violations of functional liver samples (corresponding laboratory manifestations of hypersplenism, cytolysis, cholestasis, mesenchymal inflammatory, hepatoprivial syndrome, hypertension)
- 6. Instrumental confirmation of portal hypertension (detection of varicose veins with ultrasound, endoscopy, rectoscopy)
- 7. Morphological signs of LC (fibrotic changes and nodal regeneration)

Diagnosis

- 1. Etiology (alcoholic, viral, biliary, toxic, etc.)
- 2. Morphology (small, large, mixed)
- 3. Morphogenesis (post-necrotic, portal, biliary, mixed)
- 4. Clinical and functional condition (compensation, sub- or decompensation, indicating the severity of portal hypertension, liver failure)
- 5. Indication of syndromes (with severe hemorrhagic syndrome, hypersplenism)
- 6. Process activity (active, inactive or remission)
- 7. The course (progressive, stable)

Differential diagnosis

- Primary liver cancer
- Liver fibrosis
- Autoimmune hepatitis
- Acute viral hepatitis
- Alveolar echinococcosis
- Constrictive pericarditis
- Hemochromatosis
- Wilson-Konovalov disease
- Amyloidosis
- Benign subleukemic myelosis
- Waldenstrom macroglobulinemia

Activity criteria

2-4 times increase in signs of inflammation - necrotic active indicators: (AlAT, AsAT,

γ - globulins, IgM, IgG, conjugated bilirubin, ESR)

Principles of management of the patients with cirrhosis

- 1. Categorical alcohol intake cessation
- 2. Prevention of acute viral hepatitis
- 3. Protection against hepatotoxic drugs
- 4. Physiotherapeutic procedures, insolation, vaccination are contraindicated
- 5. Gentle regime of work and rest compliance
- 6. Diet

  the exception of fatty, fried foods

  with ascites - restriction of fluid and sodium chloride

  with hepatic encephalopathy - protein restriction
- 7. Vitamin therapy

Drug therapy for cirrhosis

1. Treatment of portal hypertension and edematous ascites syndrome

- Non-selective β-blockers (propranolol) and vasodilators (prolonged nitrates)
- Water-salt regime, diuretics (spironolactone +/- furosemide)

2. Treatment of hepatic encephalopathy

- Protein restriction in food, antibiotics to suppress intestinal flora, lactulose, α-ketoglutaric acid

Treatment for Compensated Cirrhosis (Childe-Pugh Class A)

- Exclusion of alcohol and hepatotoxic factors
- Balanced diet
- With viral cirrhosis B and C - lavimudine or IFN-alpha in usual doses
- With alcoholic cirrhosis - a complete rejection of alcohol
- With primary biliary cirrhosis:
- UDCA preparations (ursofalk 250-500 mg 2-3 r per day, depending on body weight)
- Ademethionine (heptral) 400-800 mg per day intramuscularly or intravenously for 2-3 weeks, maintenance therapy - 1-2 months
- With itching, cholesterol 4-12 g per day, bilignin (bile acids bind)
- Calcium preparations (calcium glycerophosphate at 0.5x3 per day)

Treatment of subcompensated and decompensated liver cirrhosis (class B, C according to Child-Pugh)

- Diet with a limited amount of protein (0.5 g / kg body mass) and salt
- Basic therapy:
- Hepatoprotectors:
- Vegetable (Hepabene, Silymarin, Carsil F) - 1-2 months
- Preparations of essential phospholipids (essenteale forte H-IV 5-10 ml on autologous blood for 5-10 days, then in capsules:
- 2 caps. x 3 per day for 1-2 months, Enerliv — 1–2 capsx3 per day 1-2 months)
- Lipoic acid (berlition) 600 mg in the morning 1 month
- Ademethionine (heptral): 400 - 800 mg once a day, IV slowly or IM, then 800-1600 mg / day PO - 1-2 months maintenance dose
- Enzyme preparations (mesim-forte, pangrol, creon) with concomitant chronic pancreatitis and exocrine pancreatic insufficiency

- With edematous ascites syndrome:
- Spironalactone (Veroshpiron 100-400 mg)
- in combination with furosemide 40-100 mg
- With bacterial ascites-peritonitis:
- Antibacterial drugs
- (cephalosporins III generations : rifaximin (alpha normix) 400-800 mg / day orally for 5-10 days)
- With cholestasis:
- Ursodeoxycholic acid (ursofalk, ursolysin, ursosan) 250 mg 3 times a day, PO, (10-20 mg / kg body weight), for 3 months. Ademethionine (heptral)
- Calcium and Fat Soluble Vitamins A, E (400 mg / day), D, K
- With portal hypertension:
- Portal pressure reduction
- (Non-selective B-blockers-propranolol
- 10 mg 3 / day, bisoprolol for a long time), nitrates - isosorbide mononitrate 30-60 mg / day, surgical methods
- Liver transplantation

Treatment of hepatic encephalopathy

- Restriction of protein in the diet to 40 g per day
- IV administration of a 5% glucose solution,
- hepasol A, hepasteril
- Reducing the formation of ammonia in the intestines - lactulose (dufalac) 10 ml 2-3 r daily inside, rifaximin 1200 mg / day or ciprofloxacin 500 mg 2 r per day
- Ammonia neutralization in the liver and muscles -
- L-ornithine - L-aspartate (Hepa-Merz) (20-40 g / day i / v slowly), glutargin-4% 50 ml per physical solution, citrarginine 10 ml of drinking solution 3 r / day
- Efferent treatments
- Liver transplantation

Detoxification therapy in the treatment of liver cirrhosis

- Elimination of dyspeptic disorders and constipation (in order to reduce the absorption of toxic substances formed in the colon), enzyme preparations that do not contain bile acids (mesim forte, creon, pancreatin) are prescribed for this.
- Adsorbents (for intestinal cleansing - enterosorbent, activated carbon, bowel lavage)

With bleeding from varicose veins of the esophagus and stomach:

- - Somatostatin preparations (100 mg every 2 hours 2 days), intravenous glipressin,
- - Hemostatic therapy (aminocaproic
- acid, etamzilate, vikasol, etc.)
- - Proton pump inhibitors (pantoprozol,
- omez, nexium) or H2-histamine blockers (kvamatel)
- - Balloon therapy (Blackmore balloon)
- - Endoscopic treatment (vein ligation)
- - Surgery

Stem cells in the treatment of liver cirrhosis

A new, promising direction in the treatment of cirrhosis is the use of stem cell transplantation.

Advances in stem cell biology, which have shown the multipotent capabilities of their tissue differentiation, have made tissue regeneration a clinical reality.

The use of stem cells in the treatment of liver cirrhosis makes it possible to repair, restore, replace or regenerate a damaged organ in cirrhosis and is more effective than pharmacological drugs.

Currently, only stem cell transplantation can save a patient dying from cirrhosis.

Cell therapy using autologous stem cells can solve the problem of cirrhosis.

Treatment program

- Etiological treatment (with alcohol, "cardiac", viral)
- Clinical nutrition and regimen
- Improving the metabolism of hepatocytes (vitamins, hepatoprotectors)
- Detoxification therapy
- Pathogenetic treatment (glucocorticoids)
- Treatment of edematous ascites syndrome (diuretics, protein preparations, paracentesis, surgical methods)
- Treatment of bleeding from varicose veins of the esophagus (transfusion therapy, somatostatin, vasopressin, balloon tamponade)
- Treatment of chronic hepatic encephalopathy (lactulose, antibiotics, ornicetil, detoxification)
- Treatment of cholestasis (ursodeoxycholic acid)
- Surgery

Pharmacotherapy

- Antiviral therapy - 3-6-12 months
- Hepatoprotectors - 3-6 months
- Metabolic drugs -1 month
- Anti-cholestatic drugs -3-6 months
- Diuretics - for life
- Detoxification therapy - for life
- Dyspepsia correction - on demand
- Dysbacteriosis correction - on request.

Some words from comedy novel…

- "… I had the (over all liver disease) symptoms, beyond all mistake, the chief among them being "a general disinclination to work of any kind." What I suffer in that way no tongue can tell. From my earliest infancy I have been a martyr to it. As a boy, the disease hardly ever left me for a day. They did not know, then, that it was my liver. Medical science was in a far less advanced state than now, and they used to put it down to laziness.
- ”
- JK Jerome “Three Men in a Boat ”