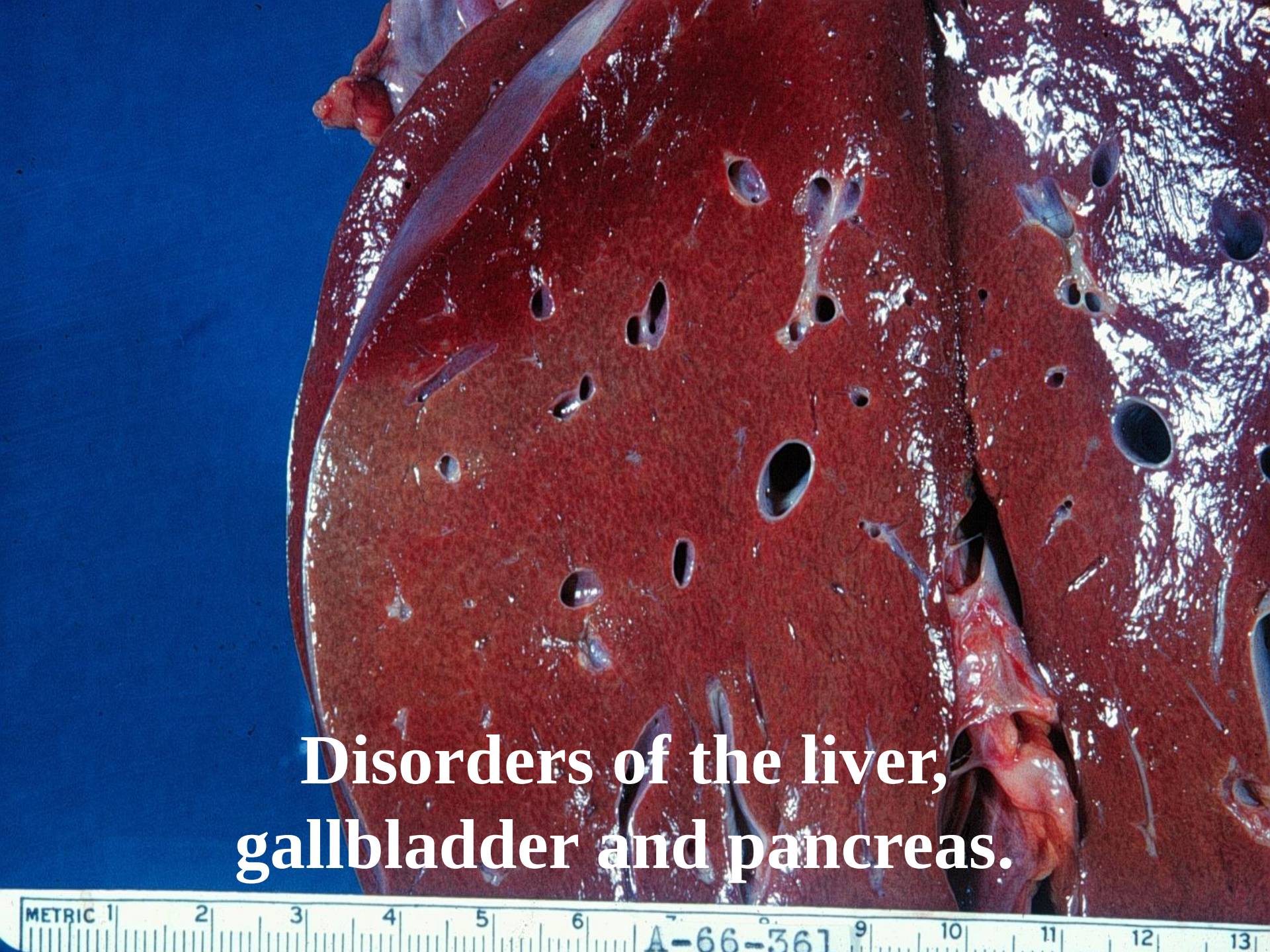


Disorders of the liver, gallbladder and pancreas.

Disorders of the liver, gallbladder and pancreas.

I. Microspecimens:

№ 89. Massive necrosis of liver. *(H-E. stain).*

Indications:

1. The extensive area of necrosis (tissue debris) in the center of the liver lobule.
2. The inflammatory infiltrate in the area of necrosis.
3. Fatty degeneration of hepatocytes at the periphery of the lobule.

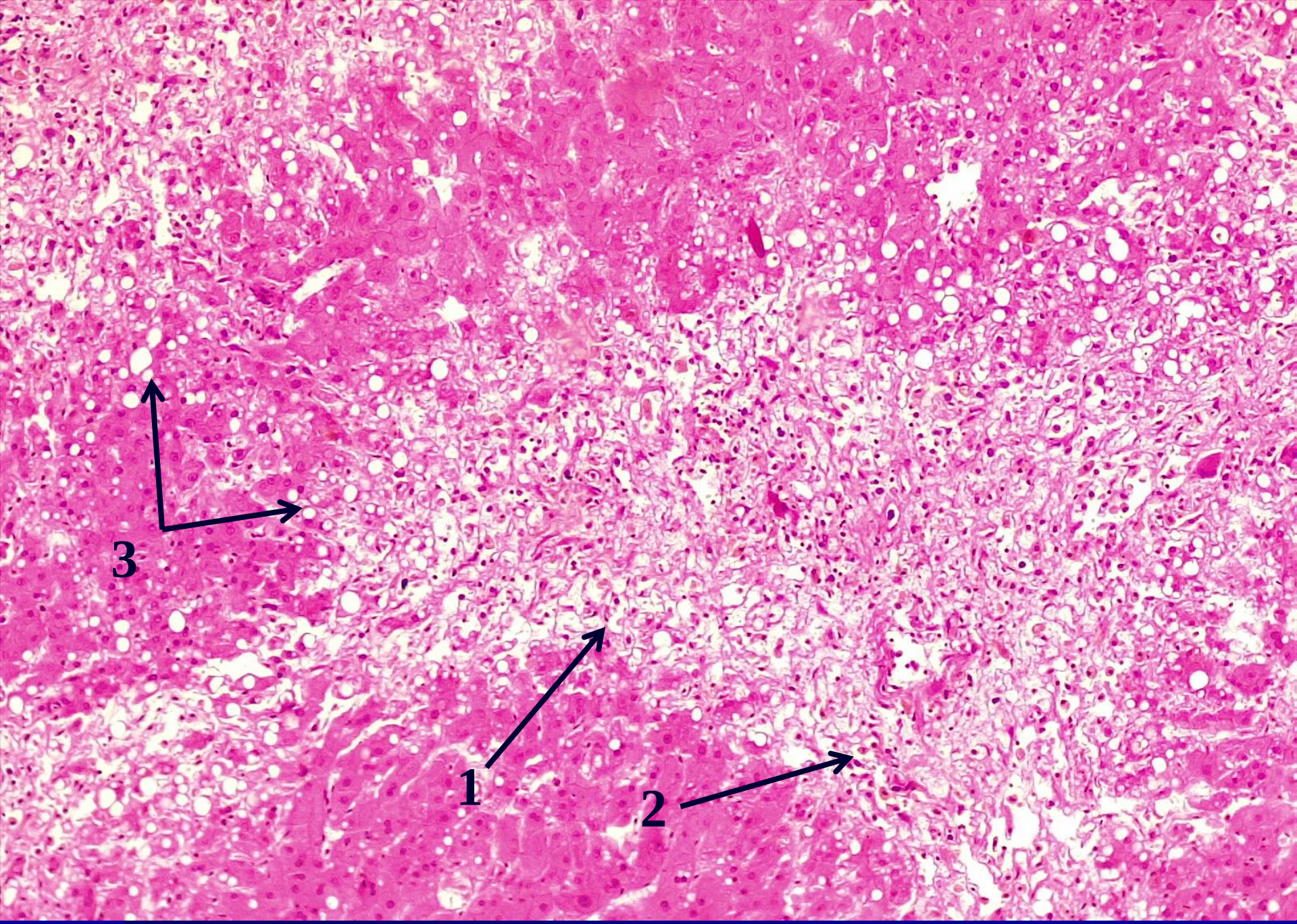
The normal structure of the liver is unclear, only parts of portal spaces are preserved, at some spaces only blood vessels. Bile ducts show cholestasis. The rest of parenchyma is transected by extensive 'bridging' necroses (centro-central, centro-portal and porto- portal). Necrotic hepatocytes are replaced by inflammatory cells – lymphocytes, macrophages, neutrophils leukocytes. Cellular debris are impregnated with bile.

№ 209. Acute viral hepatitis. *(H-E. stain).*

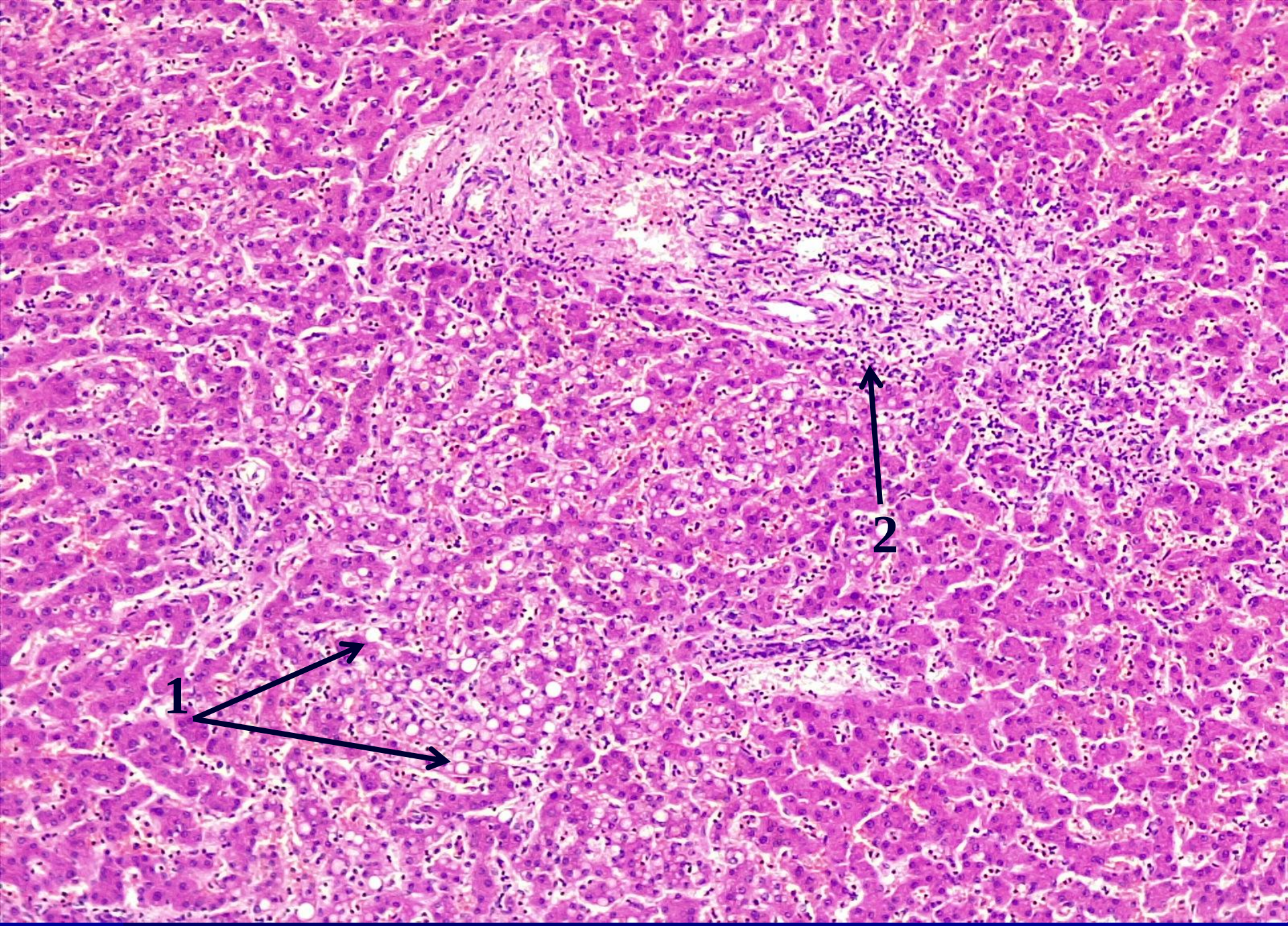
Indications:

1. Vacuolar degeneration of hepatocytes at the periphery of the lobule.
2. Lymphocytic and plasmacytic infiltration of portal tracts.

Many liver cells are swollen and vacuolated, phenomenon known as "ballooning degeneration". Infiltration of the hepatic sinusoids and portal tracts by a chronic inflammatory cellular infiltrate. The hepatic cords are also disrupted with evidence of both hepatocyte necrosis (most severely in centrilobular areas) and ongoing hepatocyte regeneration. Cells dying by apoptosis are seen as shrunken, eosinophilic Councilman bodies. Regenerating hepatocytes are large, frequently containing multiple nuclei.



№ 89. Massive necrosis of liver. *(H-E. stain).*



№ 209. Acute viral hepatitis. *(H-E. stain).*

№ 37. Micronodular cirrhosis of the liver. *(pycrofuxin by van Gieson method stain).*

Indications:

1. Thin bundles of connective tissue in the liver lobules, which join together the central veins with portals vessels.
2. "Pseudolobules".

The fibrous septa that divide the hepatic parenchyma into nodules extend from central vein to portal regions, or portal tract to portal tract, or both. The hepatocytes in the islands of surviving parenchyma undergo slow proliferation forming regenerative nodules having disorganised masses of hepatocytes. The hepatic parenchyma within the nodules shows extensive fatty change early in the disease. But as the fibrous septa become more thick, the amount of fat in hepatocytes is reduced.

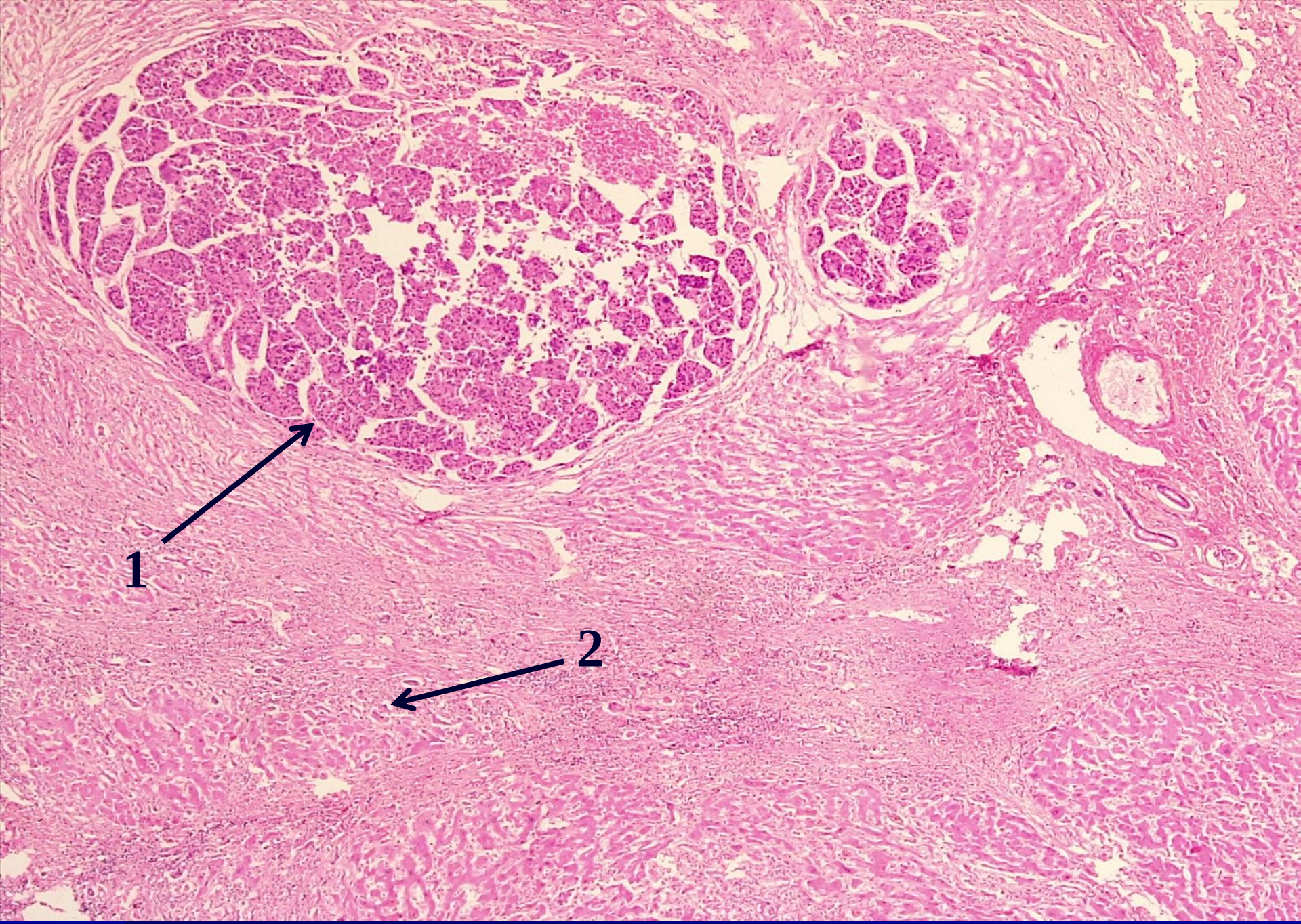
№ 157. Hepatocellular carcinoma on the background of liver cirrhosis. *(H-E. stain).*

Indications:

1. Clusters of polymorphic atypical cells with basophilic nuclei.
2. Adjacent liver tissue with cirrhotic changes.

The tumour cells in the typical hepatocellular carcinoma (HCC) resemble hepatocytes but vary with the degree of differentiation, ranging from well differentiated to highly anaplastic lesions. Most of the HCC have trabecular growth pattern. The tumour cells have a tendency to invade and grow along blood vessels. The trabeculae are made up of 2-8 cell wide layers of tumour cells separated by vascular spaces or sinusoids which are endothelium-lined. Adjacent liver tissue it is with cirrhotic changes.

№ 37. Micronodular cirrhosis of the liver. *(pycrofuxin by van Gieson method stain).*



№ 157. Hepatocellular carcinoma on the background of liver cirrhosis. *(H-E. stain).*

II. Macrospecimens:

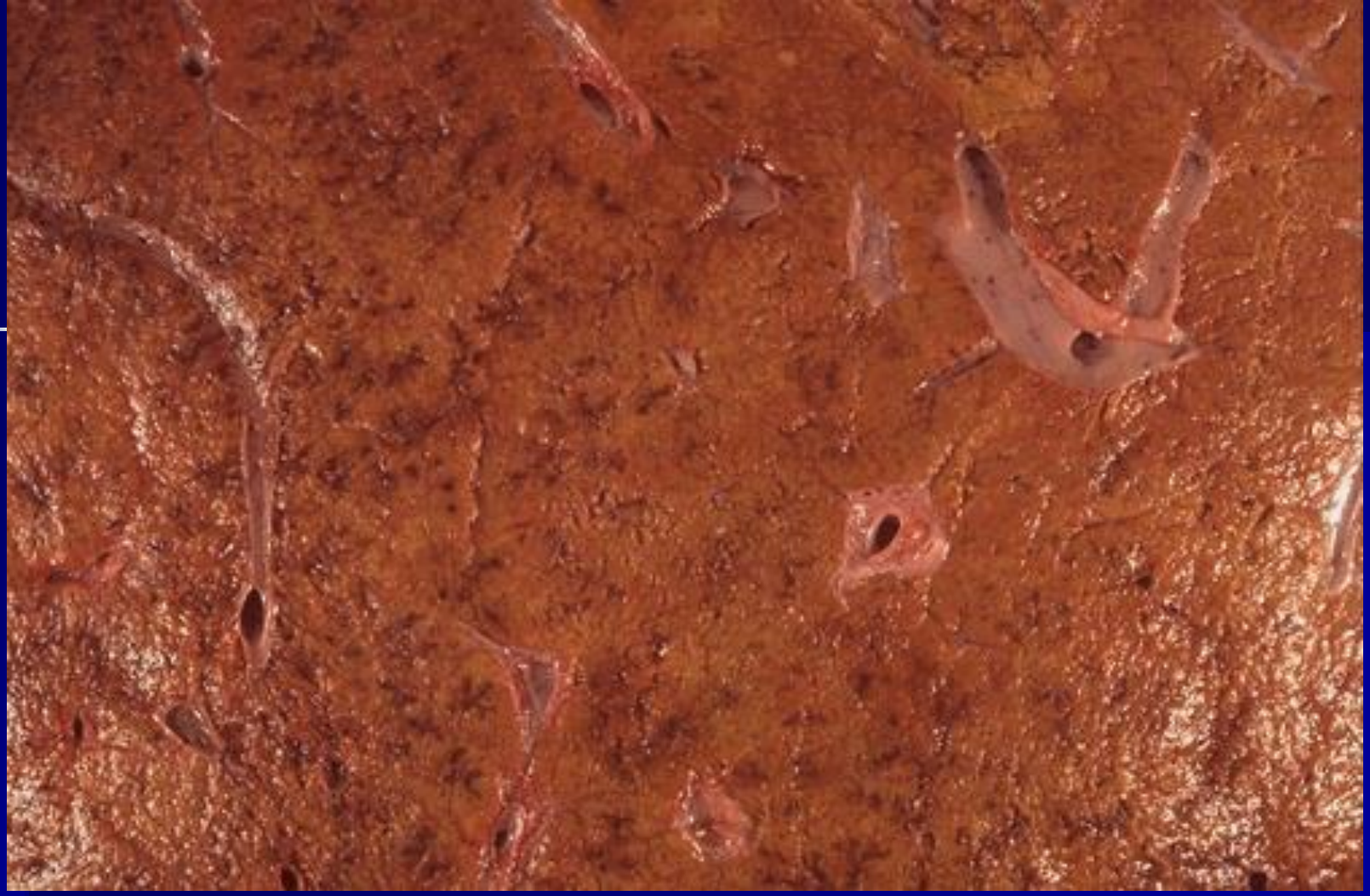
№ 72. Massive necrosis of liver.
In the first few days, the liver is slightly enlarged of dense or flaccid consistency and gets a bright yellow color both on the surface and on the section. Then, it gradually decreases in size, becomes flaccid and the capsule wrinkled; on section, the liver tissue is gray, clayey.

№ 73. Mixt (macro-micronodular) cirrhosis of the liver.
The liver is small, of hard consistency, weighing less than 1 kg, having distorted shape with irregular and coarse scars and nodules of varying size. Sectioned surface shows scars and nodules varying in diameter from 3 mm to a few centimeters.

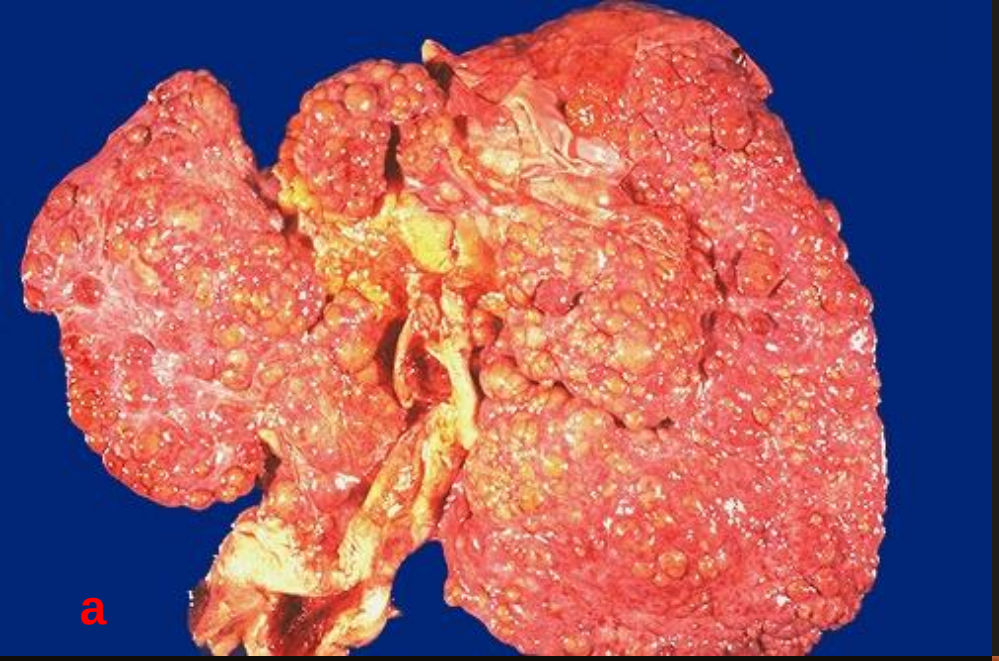
№ 74. Cancer metastasis into liver.
The liver is enlarged, on the surface and section - in all segments of the liver there are many nodules of round and oval shape, 1-4 cm in diameter, whitish of dense texture (in the center of some nodules are foci of necrosis in the form of yellowish-gray debris)

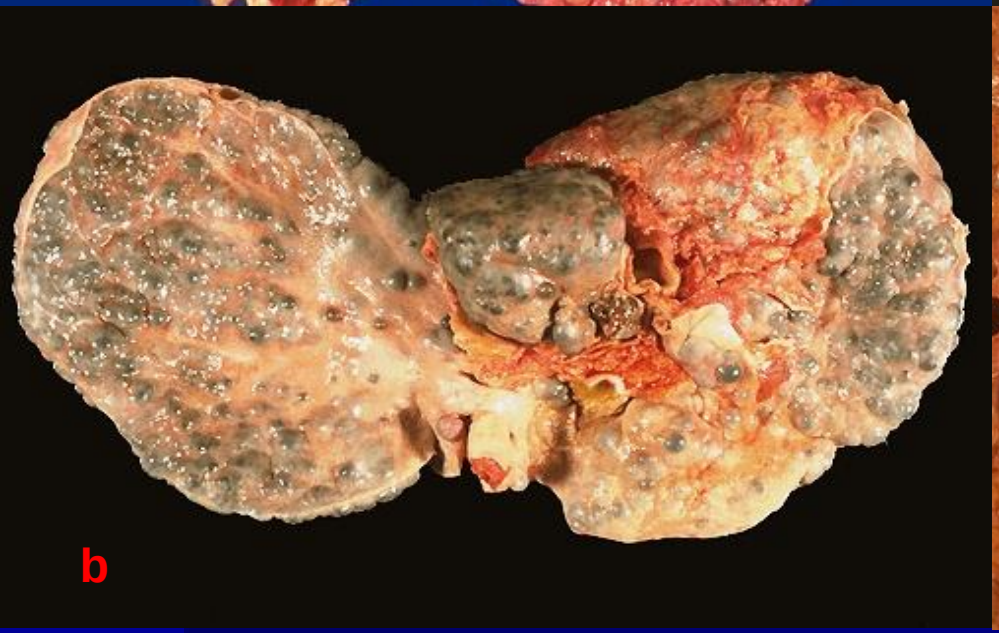
№ 76. Gallstones in the gallbladder.

The gallbladder is enlarged, the cavity expanded with multiple calculi of round shape, dark brown, gray or yellow color. The wall of the bladder is thickened, of dense consistency, the mucous membrane is smooth, often losing its softness. On the mucosa, multiple deposits of small yellowish granules (gallbladder cholesterolosis) can be observed.



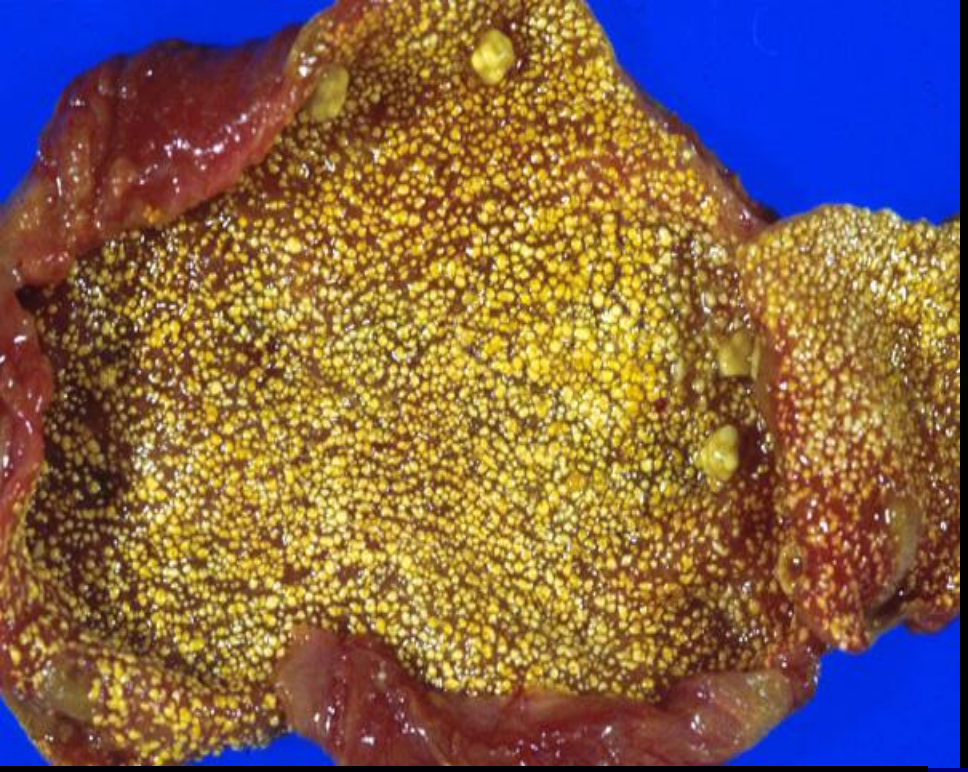
№ 72. Massive necrosis of liver.





№ 73. Mixt (macro-micronodular) cirrhosis of the liver.

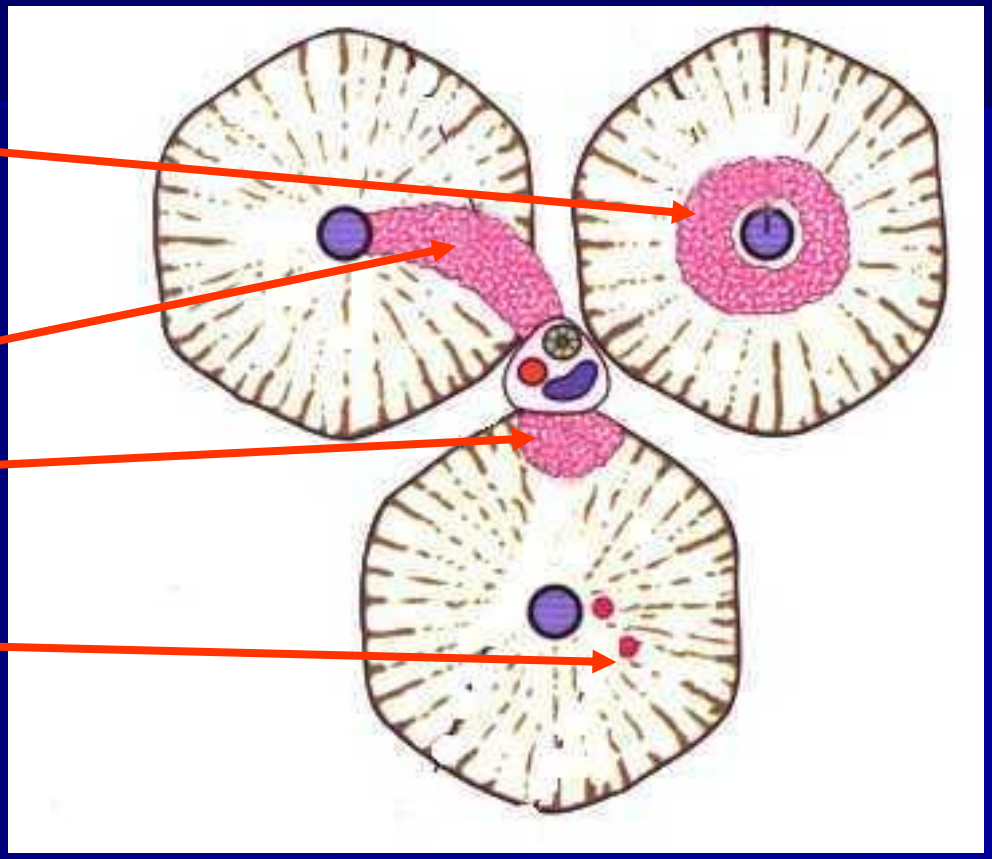
№ 74. Cancer metastasis into liver.

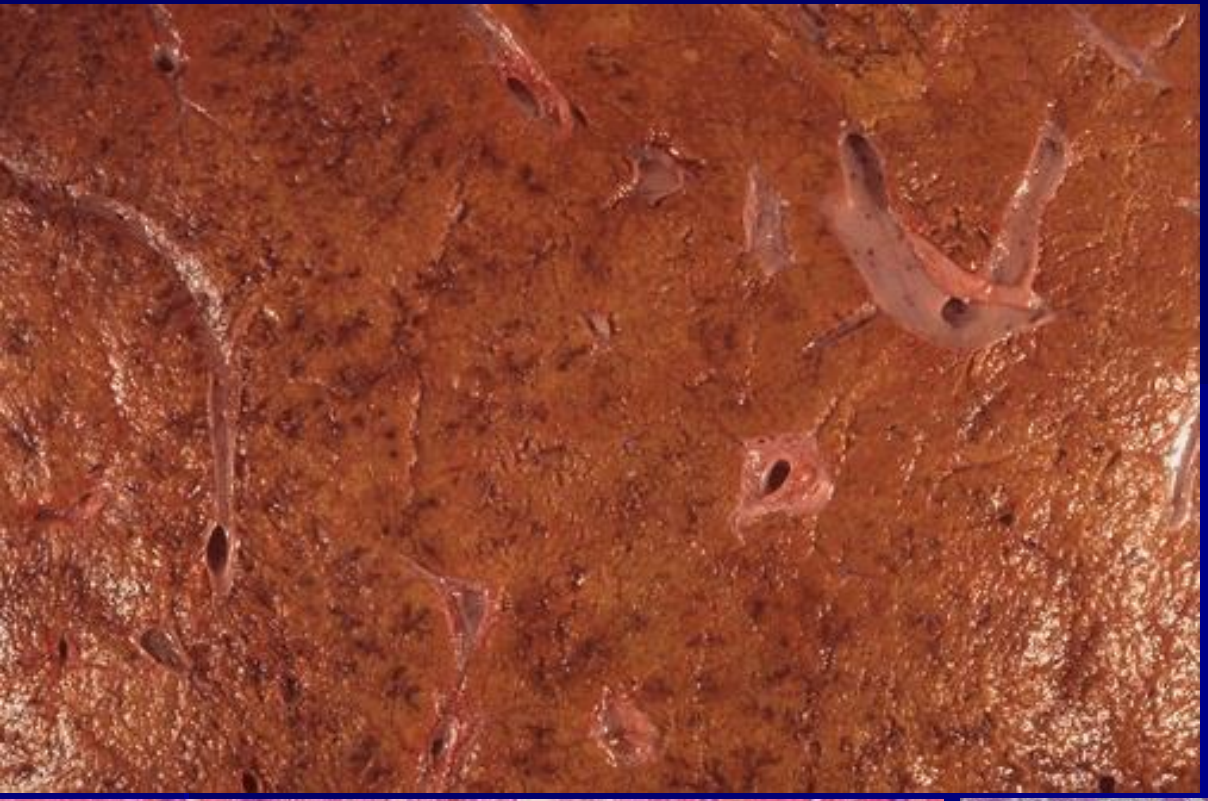


№ 76. Gallstones in the gallbladder.

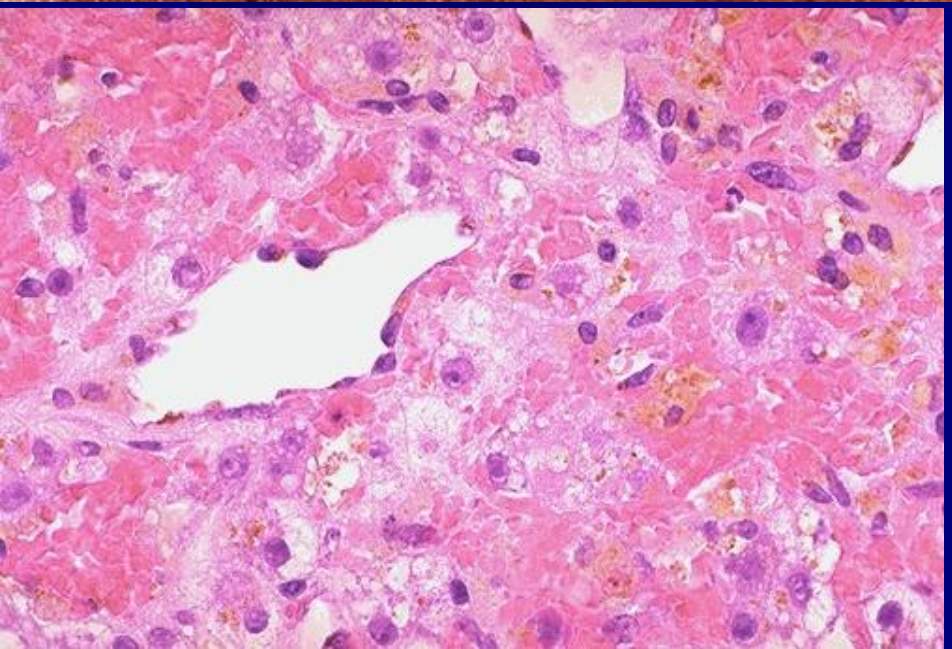
Liver injury

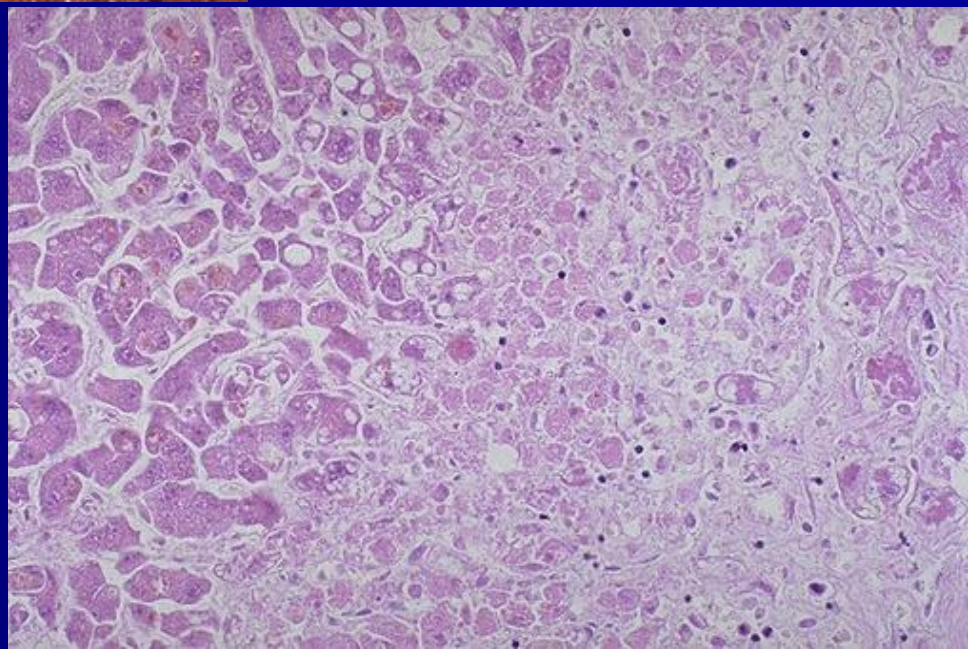
- Zonal injury – toxins / hypoxia
- Bridging type – viral etiology
- Interface (piecemeal) necrosis – autoimmune
- Apoptosis – viral etiology



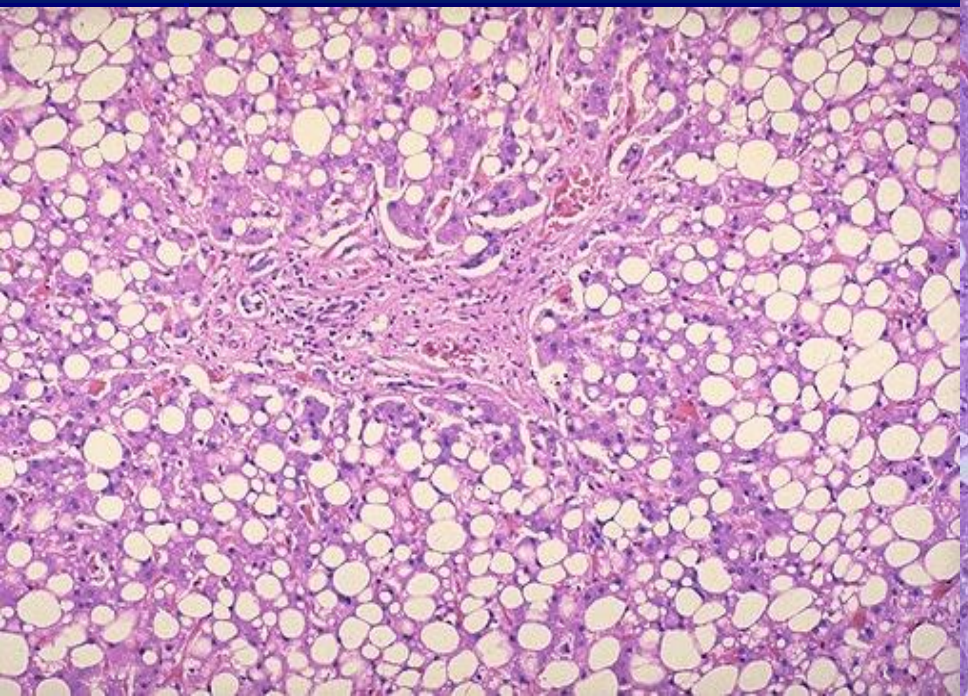


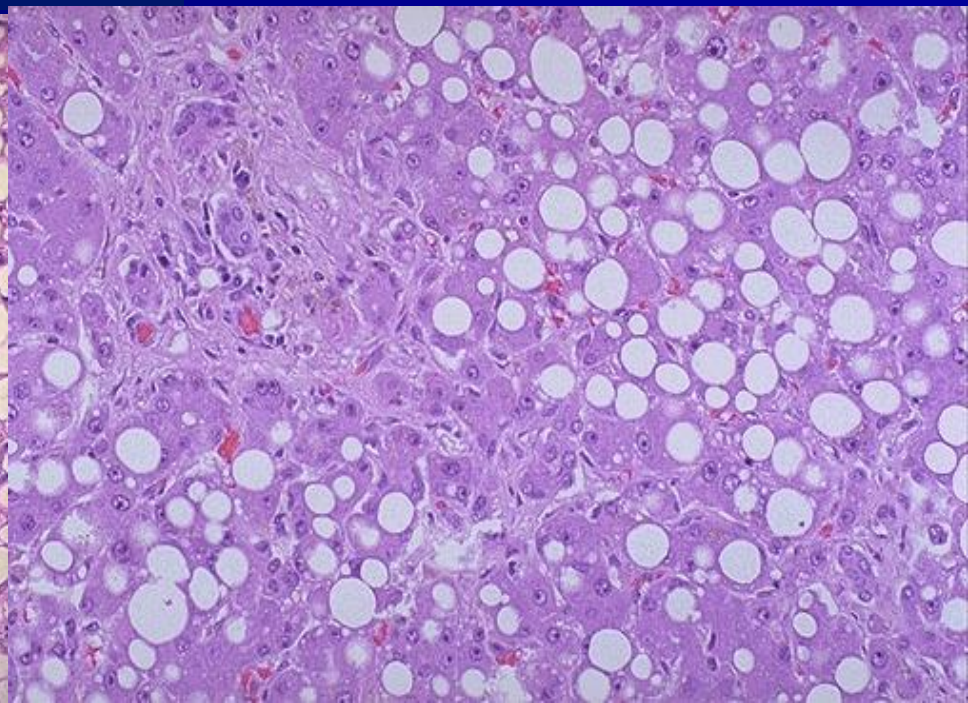
Massive necrosis of the liver.



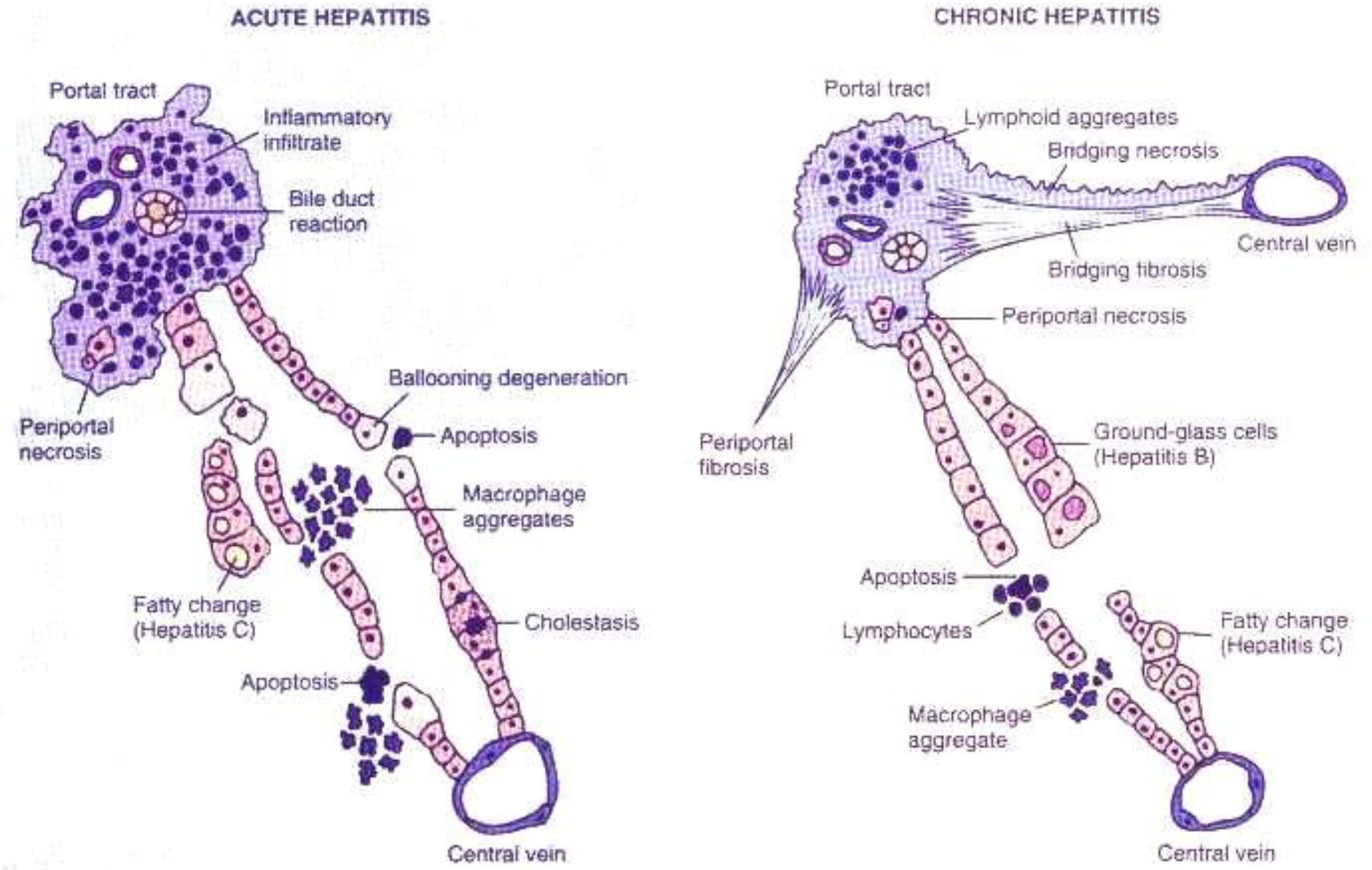


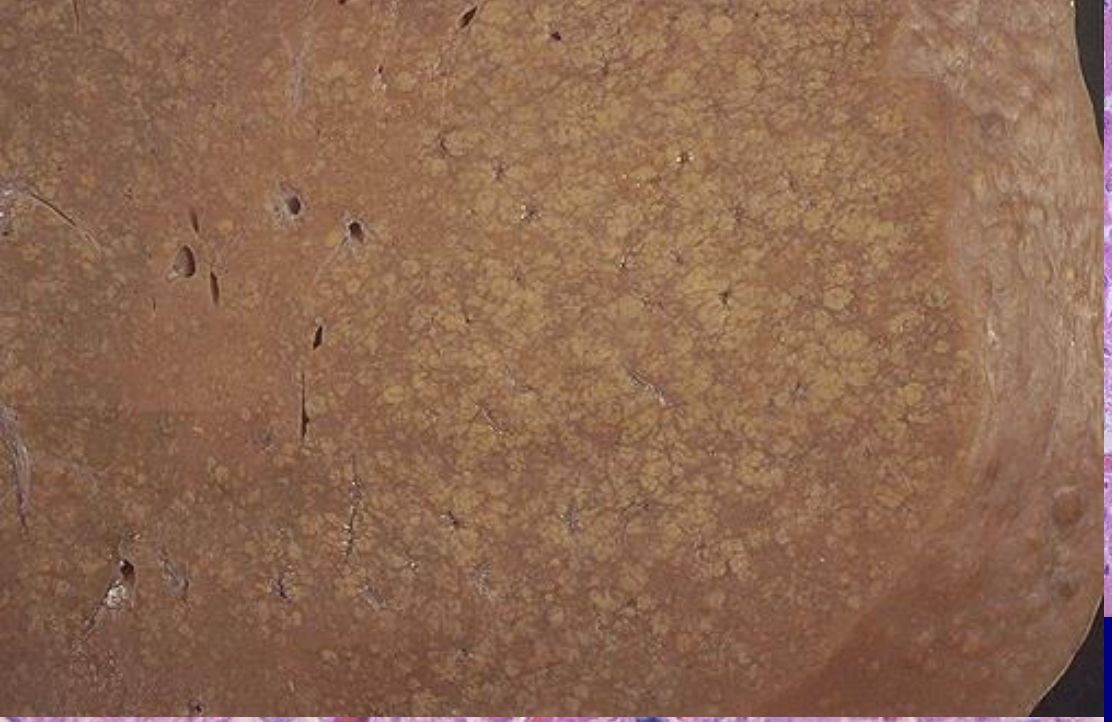
Liver steatosis.

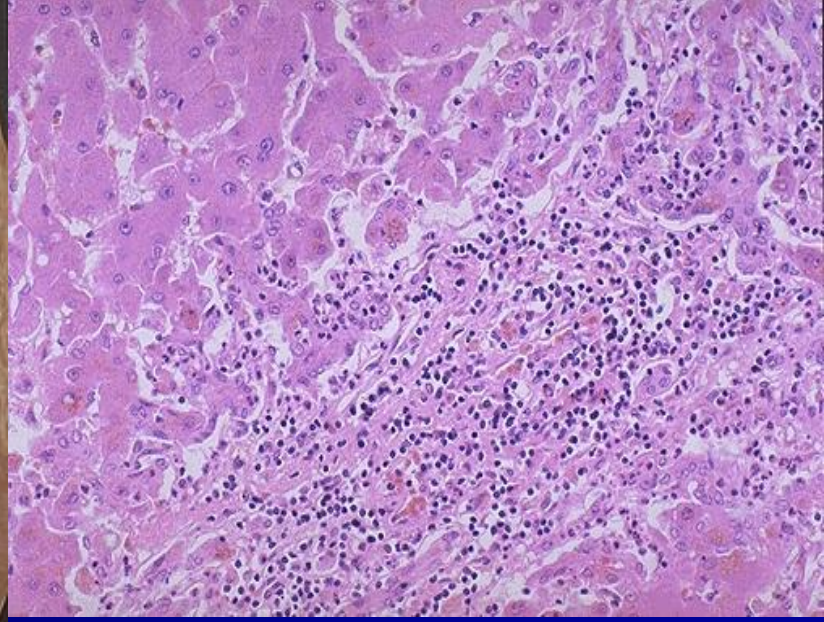


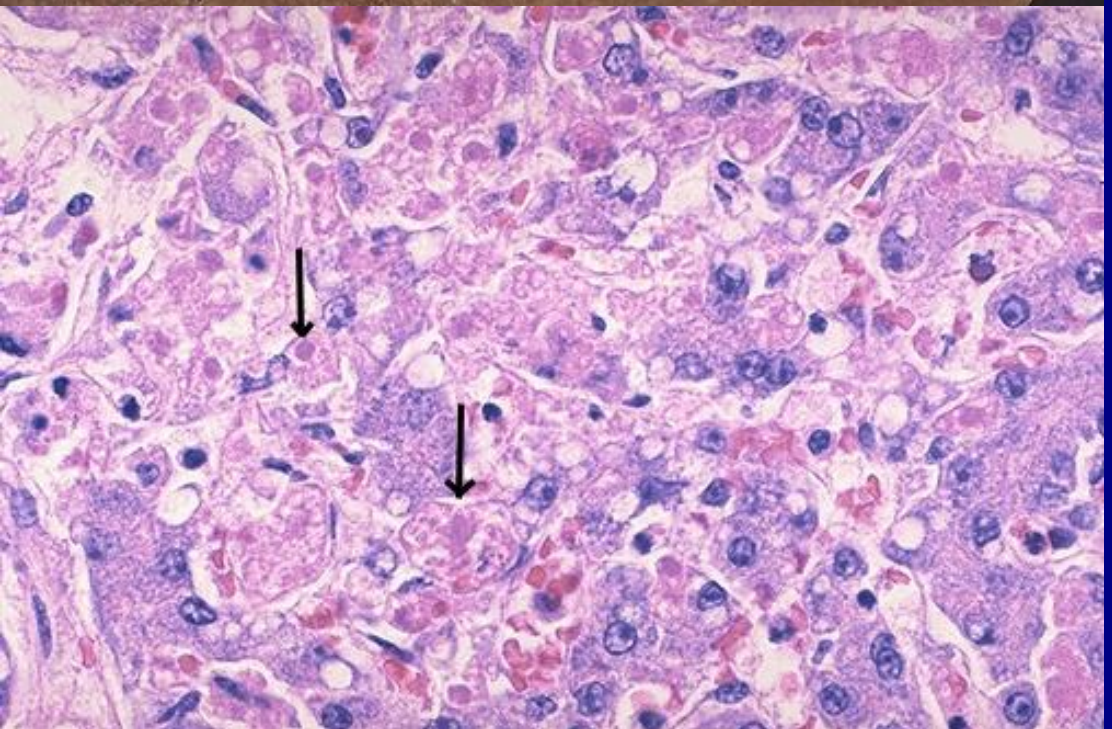


Acute hepatitis vs. chronic.



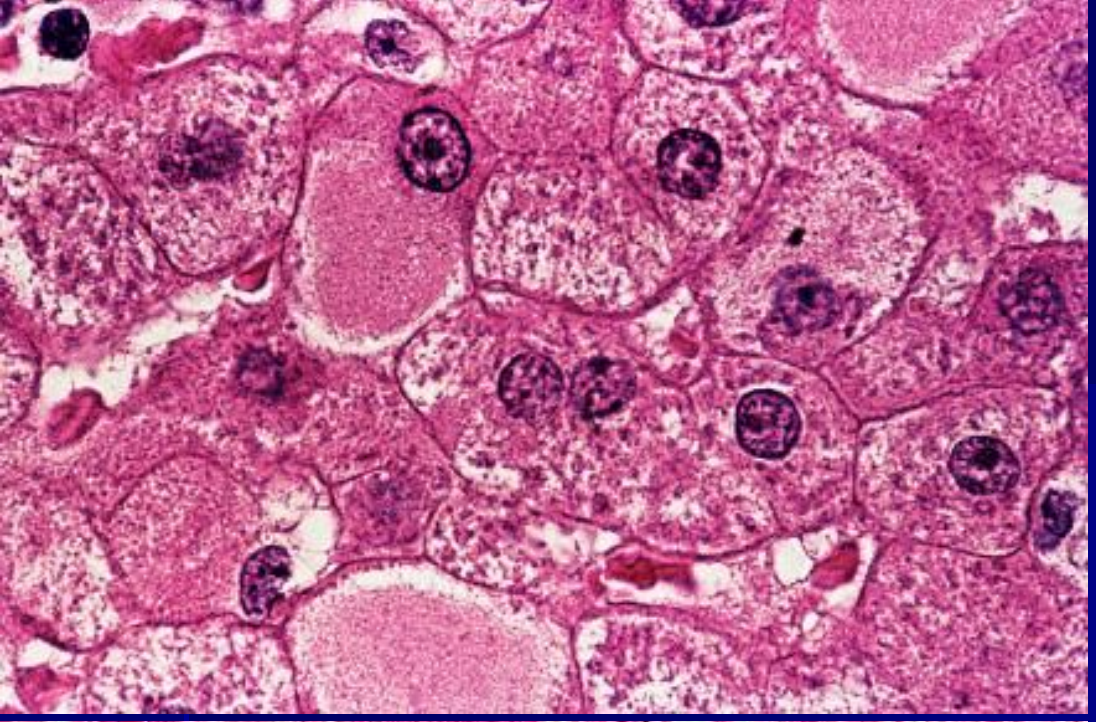




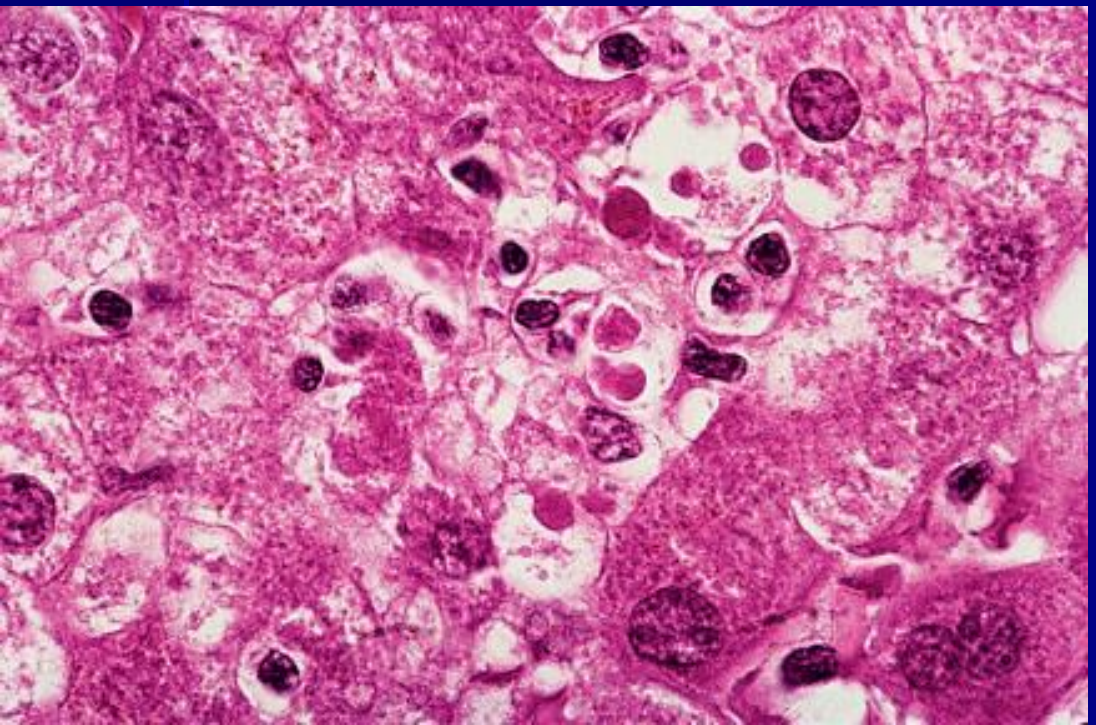


Viral hepatitis,

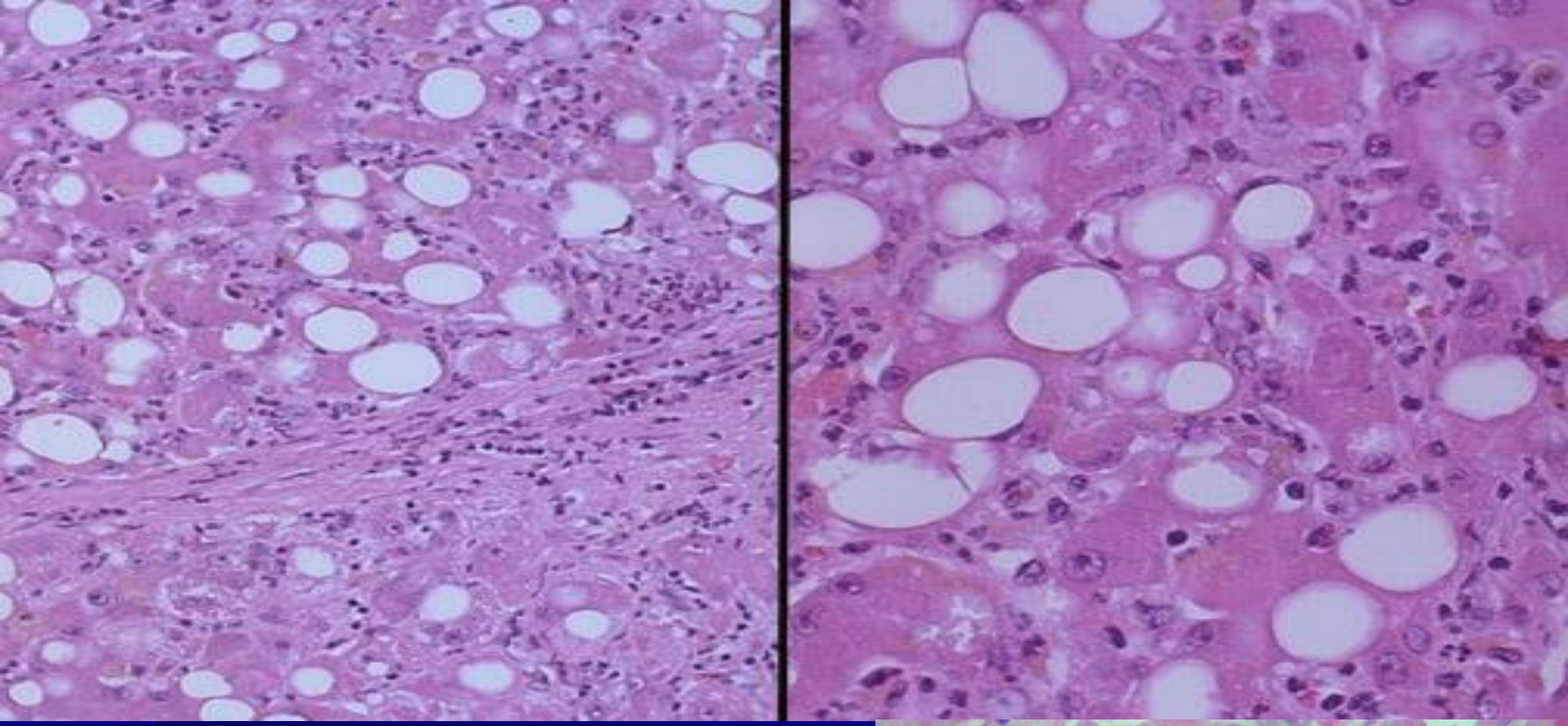
hydropic degeneration and Councilman corpuscles ←, lymphoid infiltration of the portal tracts (pariportal piecemeal necrosis) ↑



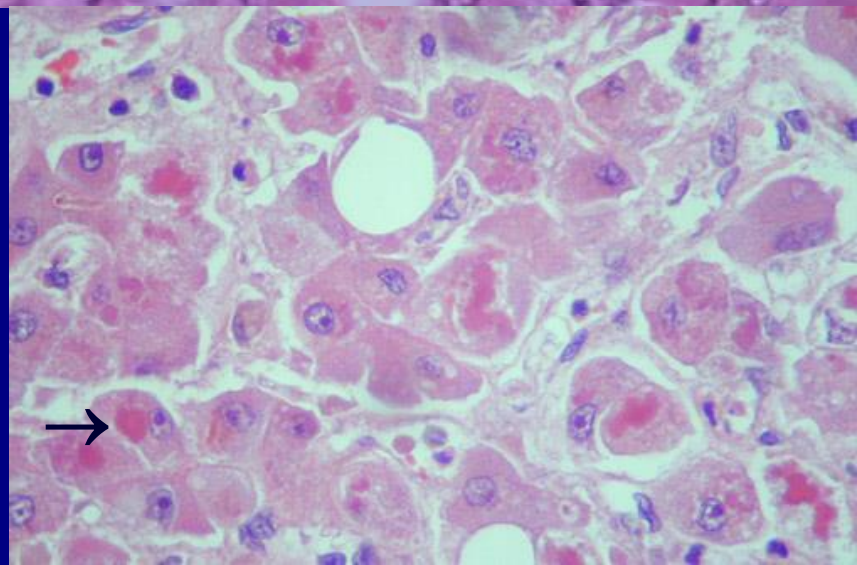
← ***"Ground glass hepatocytes"*** *with homogenized cytoplasm* (*massive accumulations of HBsAg)*

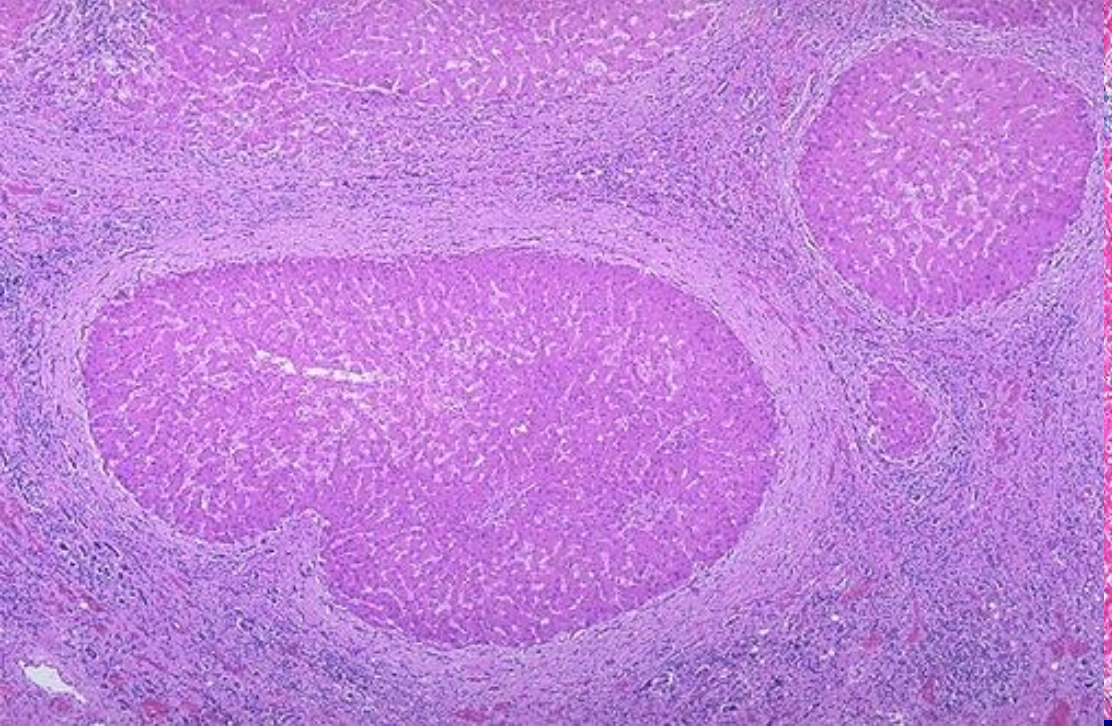


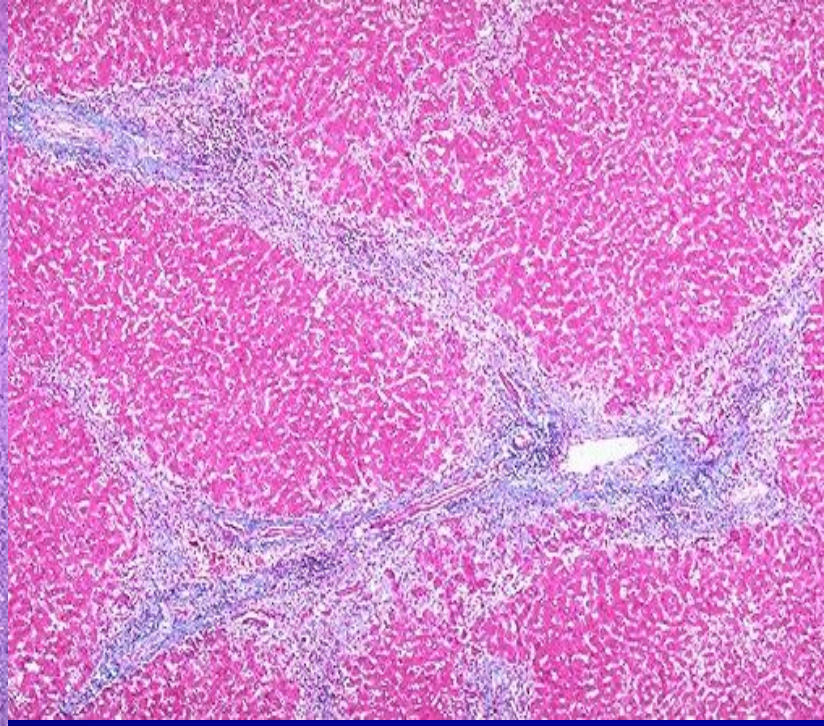
← **Rounded eosinophil fragments of apoptotic hepatocyte**– ***Councilman*** *bodies*

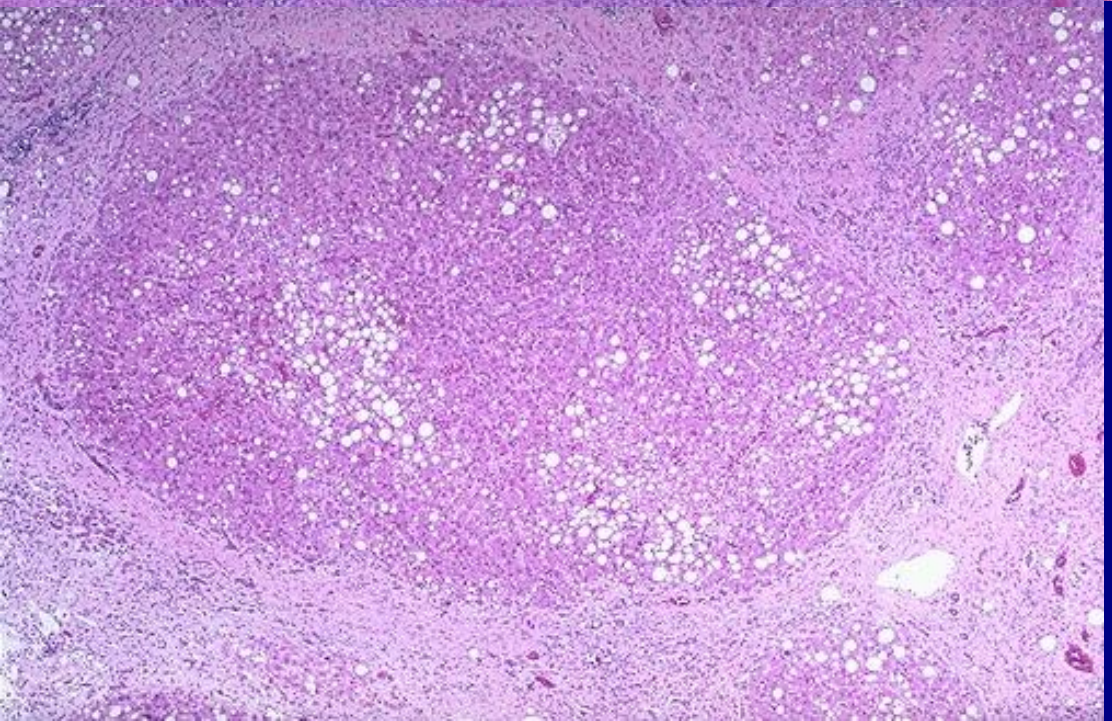


Alcoholic hepatitis,
steatosis and Mallory corpuscles









Cirrhosis of the liver, microscopic patterns.

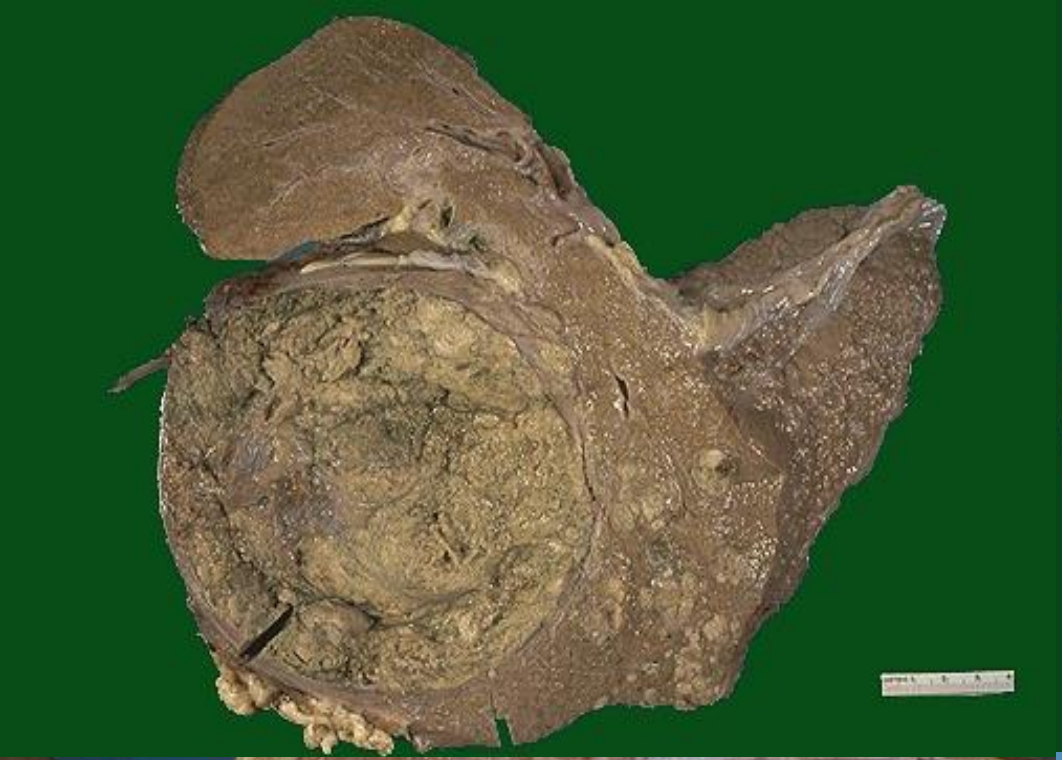
Varicose veins dilation

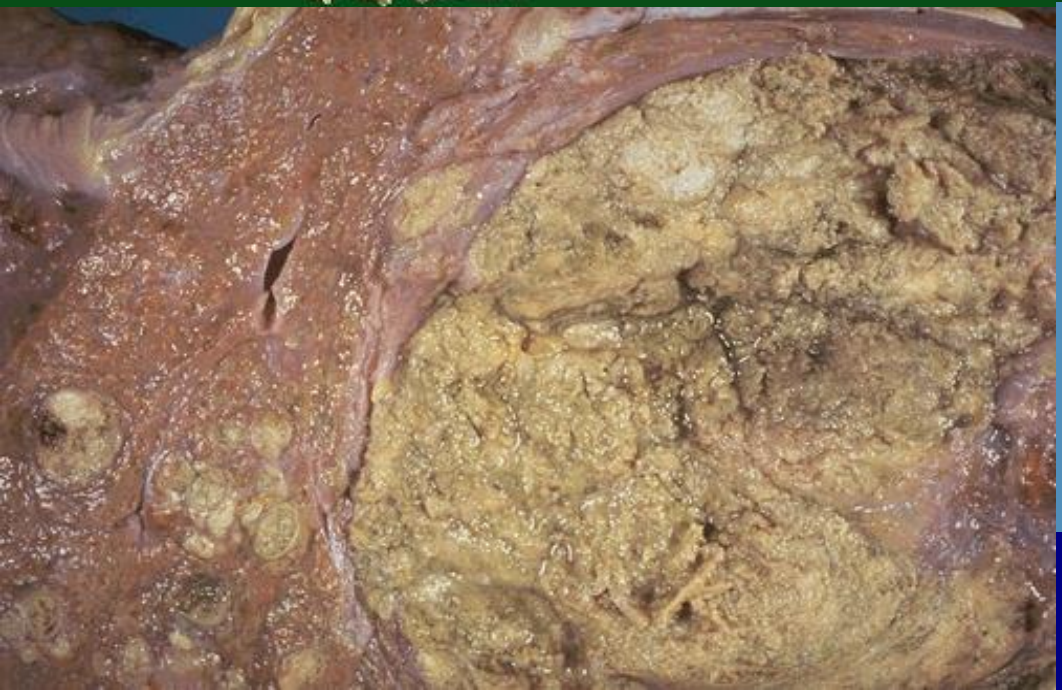
esophageal ↑

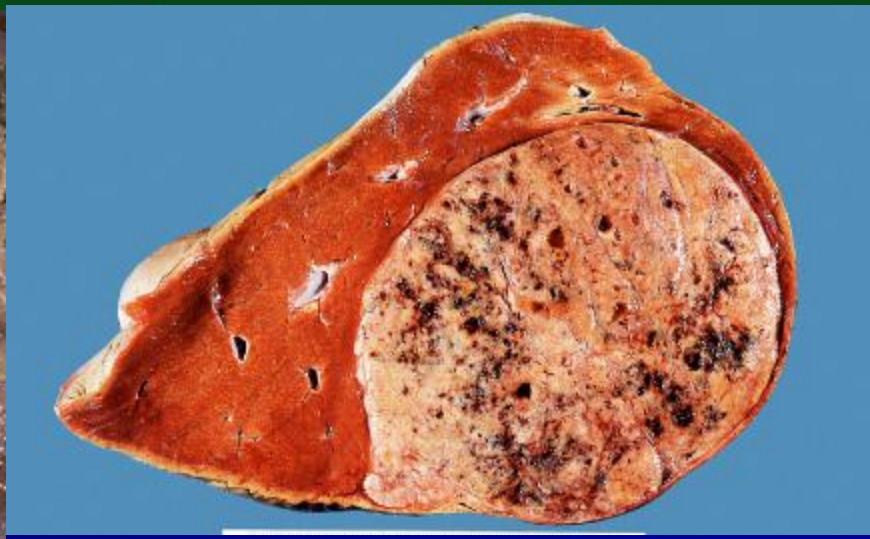
and of anterior abdominal wall → →

Splenomegaly.

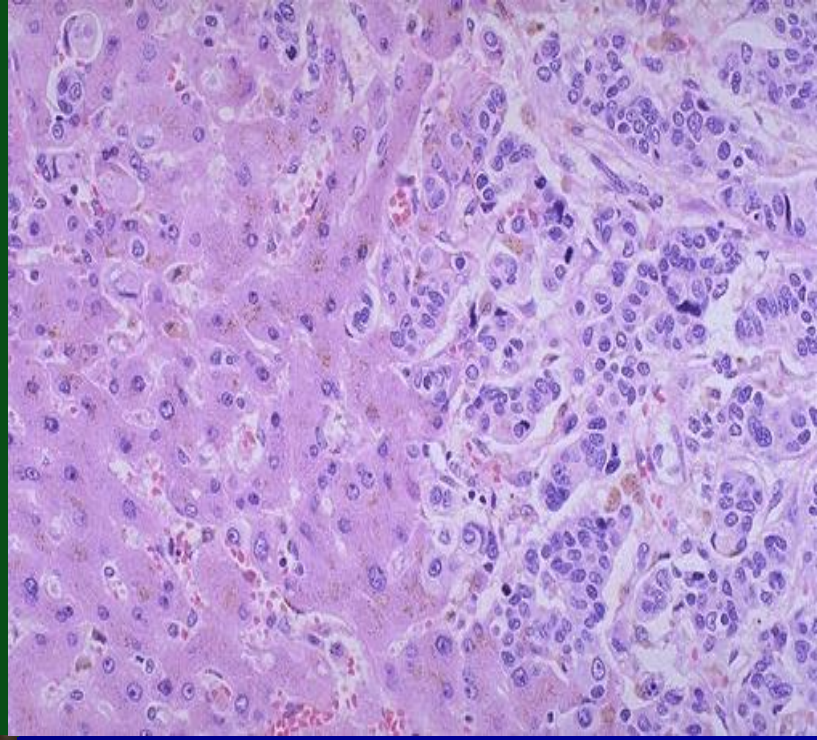
Jaundice.



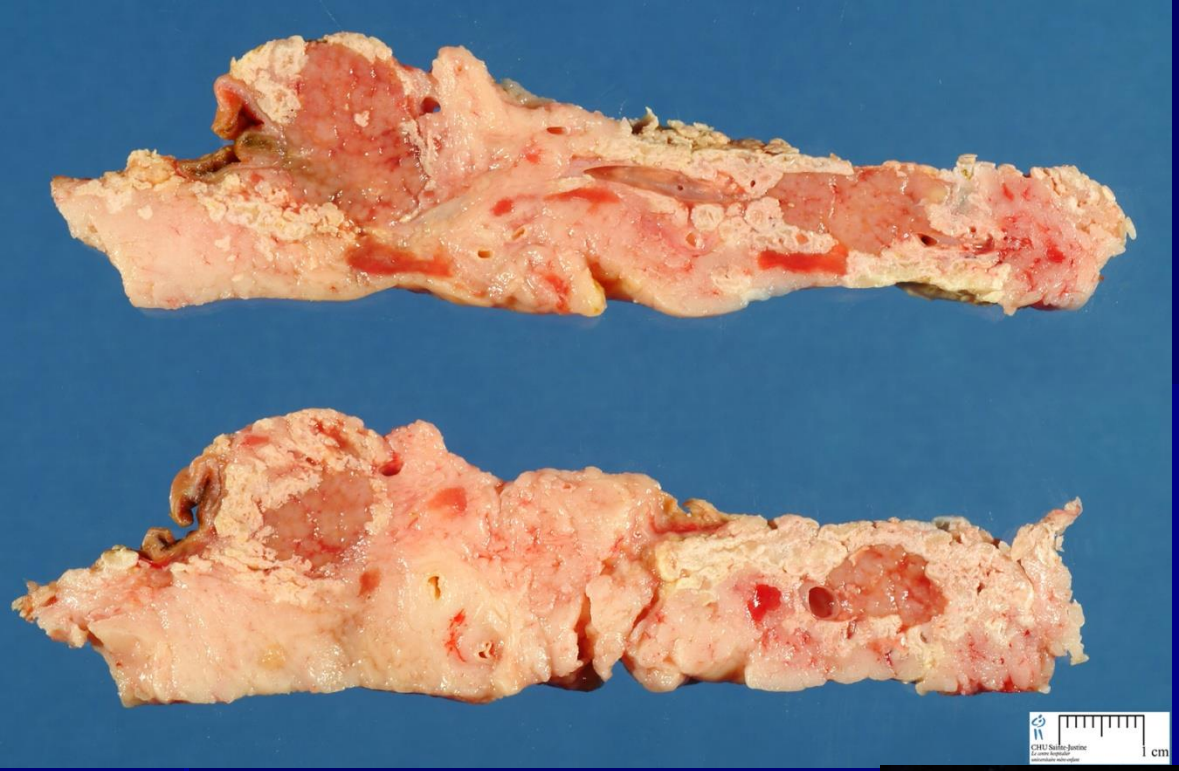




Nodular liver carcinoma.



Carcinoma metastases in liver.

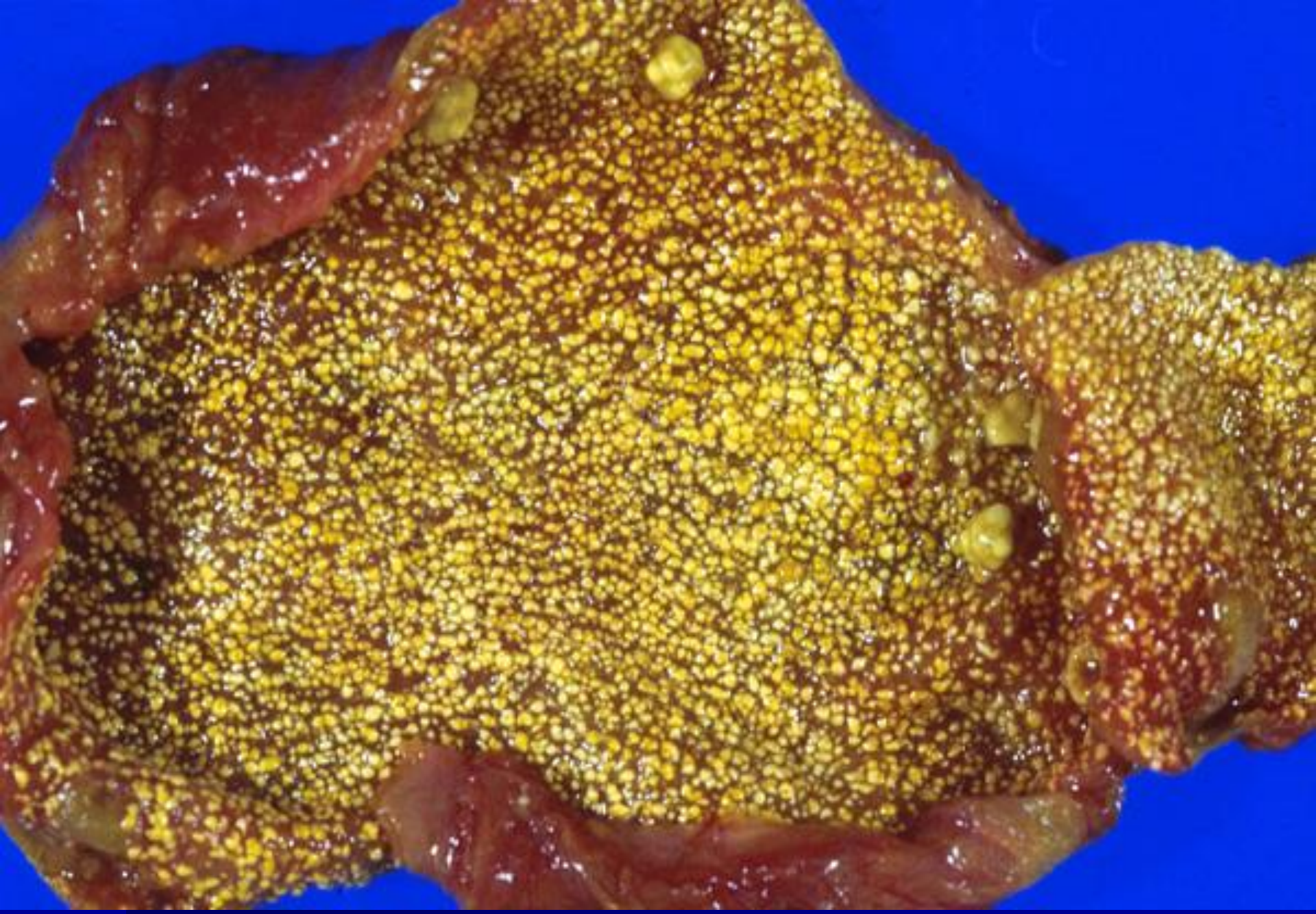


Chronic pancreatitis.

↓

↑

Acute pancreatitis.



Cholesterolosis of the gallbladder mucosa.

Types of liver injuries

Regardless of different existing causes that lead to liver lesions, there are only few variants of tissue response. The most common ones are as following:

- Hepatocyte injury and intracellular accumulations
- Necrosis and apoptosis
- Inflammation
- Regeneration
- Fibrosis

Various clinical liver diseases have different syndromal manifestations:

- Hepatic failure,
- Liver cirrhosis,
- Portal hypertension
- bilirubin metabolism disturbance causing jaundice and cholestasis.

Hepatic failure

The most severe liver disease manifestation is hepatic failure. It can happen due to sudden and massive acute hepatocyte destruction (fulminant hepatic failure), but most commonly is a consequence of successive waves of injury or progressive chronic damage (end stage liver disease).

Hepatic failure

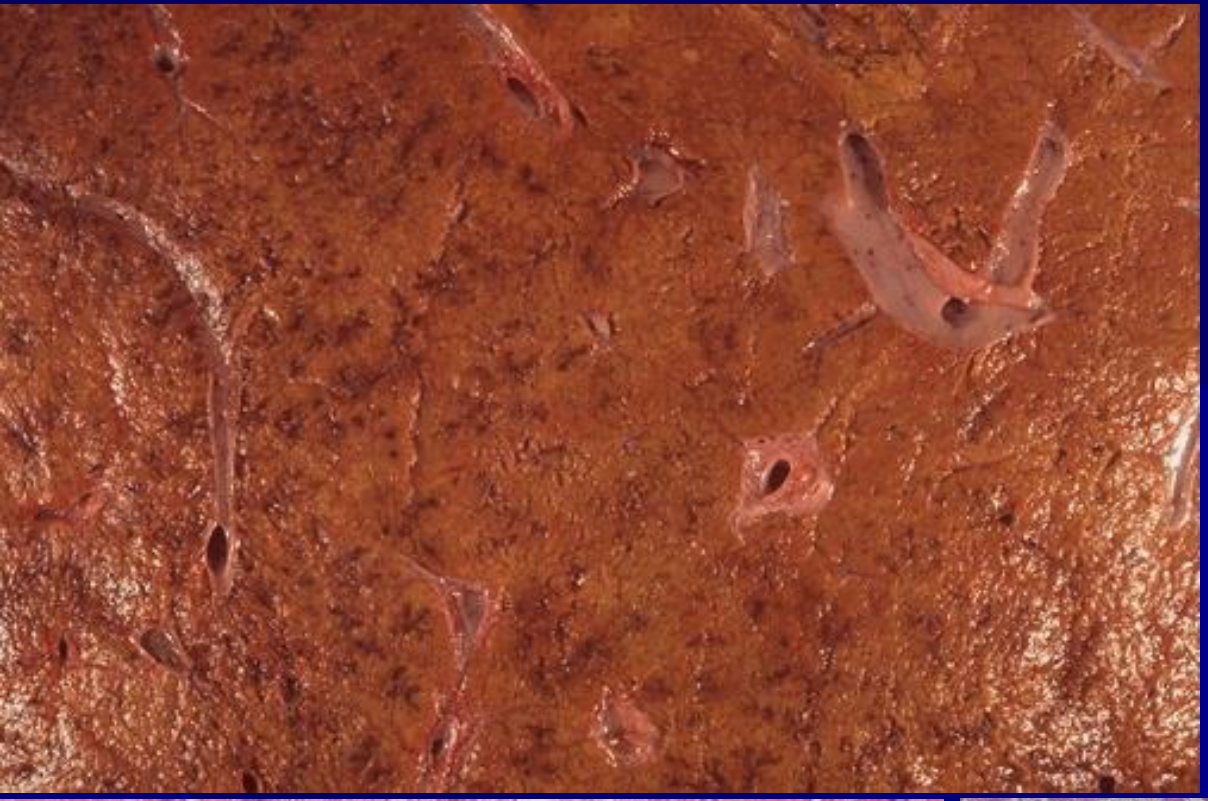
Regardless of the cause hepatic failure occurs when greater than 80% to 90% of hepatic

function is lost; Due to the incapacity of the liver to maintain homeostasis only liver transplant treatment option can be life saving. If liver transplant is not performed the mortality rate is approximately 80% . Lesions causing hepatic failure can be divided into 3 categories.

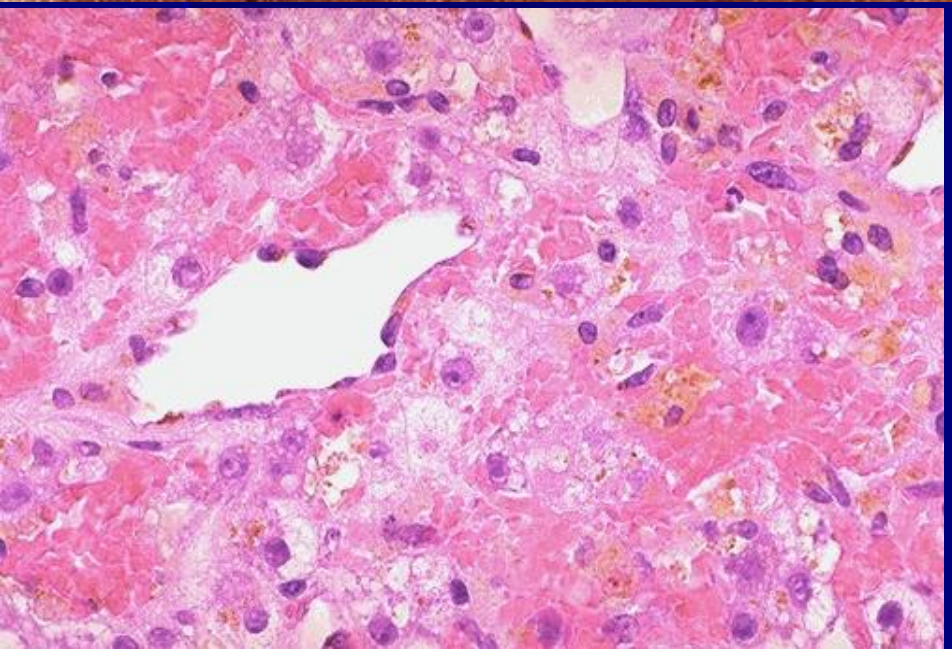
1. **Acute liver injury.**

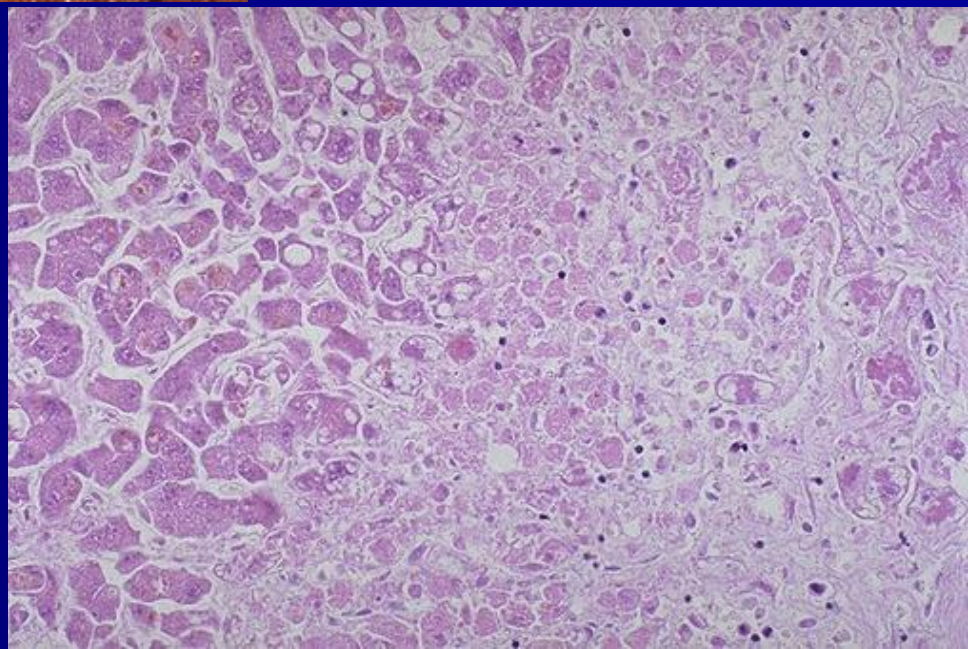
This condition is accompanied by encephalopathy. If encephalopathy is developed after 2 weeks from the appearance of jaundice then this can be considered as fulminant hepatic failure. If it is developed after 3 months after the jaundice appears it can be considered a subfulminant hepatic failure. Acute renal failure may occur due to massive liver necrosis most commonly caused by medication or toxins.

- accidental or conscious use of acetaminophen (paracetamol)
- Halothane
- Medication used for mycobacterium tuberculosis infection treatment (Rifampicin, Isoniazid)
- Antidepressants
- Mushroom poisoning (бледная поганка)
- Hepatitis A virus can cause acute hepatic failure in 4% of the cases,
- Hepatitis B virus — 8% of the cases.
- Autoimmune hepatitis and hepatitis of unclear/unknown etiology - 15% of the cases.
- Hepatitis C virus very rarely can lead to massive liver necrosis.



Acute hepatosis
toxic liver injury.





2. Chronic liver disease is the most common cause of failure The endpoint of relentless chronic hepatitis ending in cirrhosis.

3. Hepatic dysfunction without overt necrosis: Viable hepatocytes unable to perform normal metabolic functions (e.g., tetracycline Toxicity, Acute fatty liver of pregnancy).

Clinical features:

- jaundice
- Hypoalbuminemia – underlying cause of systemic oedemas
-Hyperammonemia – plays an important role in damage to brain function
- Fetor hepaticus, an odor (sweet-sour) related to mercaptan formation by intestinal bacteria during methionine (sulfur containing amino acids.) breakdown.
- Hyperestrogenemia due to impaired estrogen metabolism with palmar erythema, spider angiomata, hypogonadism, and gynecomastia

Clinical features:

Estrogen metabolism disturbance and hyperestrogenemia lead to : :

- palmar erythema formation on skin due to local and и , spider angiomata. In the center of each angiomata there is a dilated pulsating arteriole with radiating small vessels .
- In men hyperestrogenemia leads to hypogonadism and gynecomastia.
- Coagulation factor synthesis disturbance causes coagulopathy, that can lead to massive gastro-intestinal hemorrhage.

Three complications associated with hepatic failure need to be discussed separately due to the high lethality rate in patients who develop them:

1. Hepatic encephalopathy, a life-threatening disorder of CNS and neuromuscular transmission; it is caused by porto-systemic shunting and loss of hepatocellular function. Resulting excess ammonia in the blood impairs neuronal function and causes brain edema, leading to disturbances in consciousness (confusion to coma), limb rigidity, hyperreflexia, and asterixis.

2. Hepatorenal syndrome – a form of hepatic failure causing renal failure; the etiology is decreased renal perfusion pressure, followed by renal vasoconstriction, with sodium retention, impaired free-water excretion and high concentration of residual nitrogen and creatinine in the blood. The incidence is high in patients with chronic liver disease.

3. Hepatopulmonary syndrome, presenting with hypoxia. The likely cause is intrapulmonary vascular dilation (due to increased nitric oxide) and functional shunting of blood from pulmonary arteries to veins; altered blood flow causes ventilation-perfusion mismatch.

Liver cirrhosis

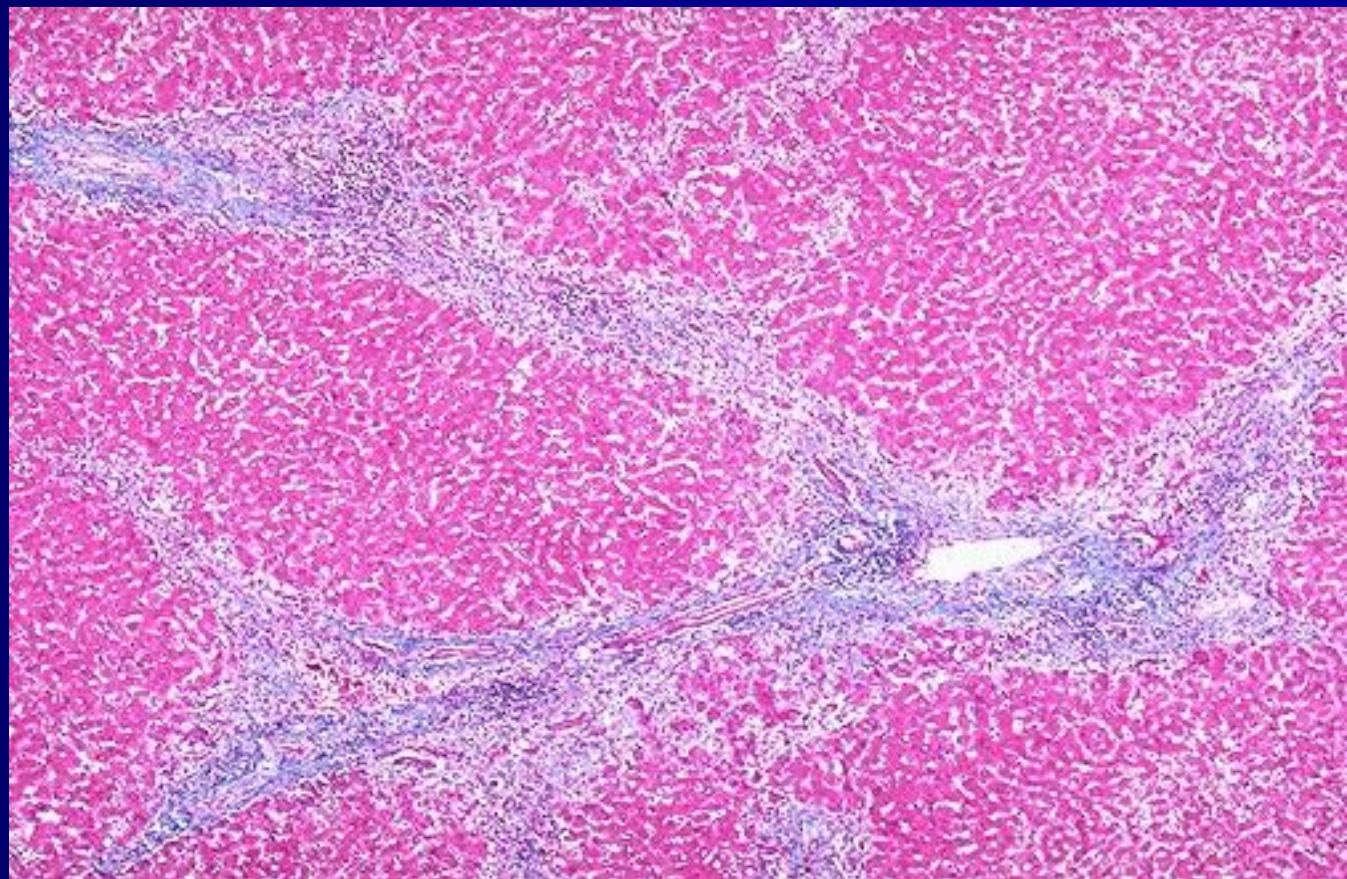
Most common causes of liver cirrhosis are:

- Alcohol abuse
- viral hepatitis
- non-alcoholic steatohepatitis

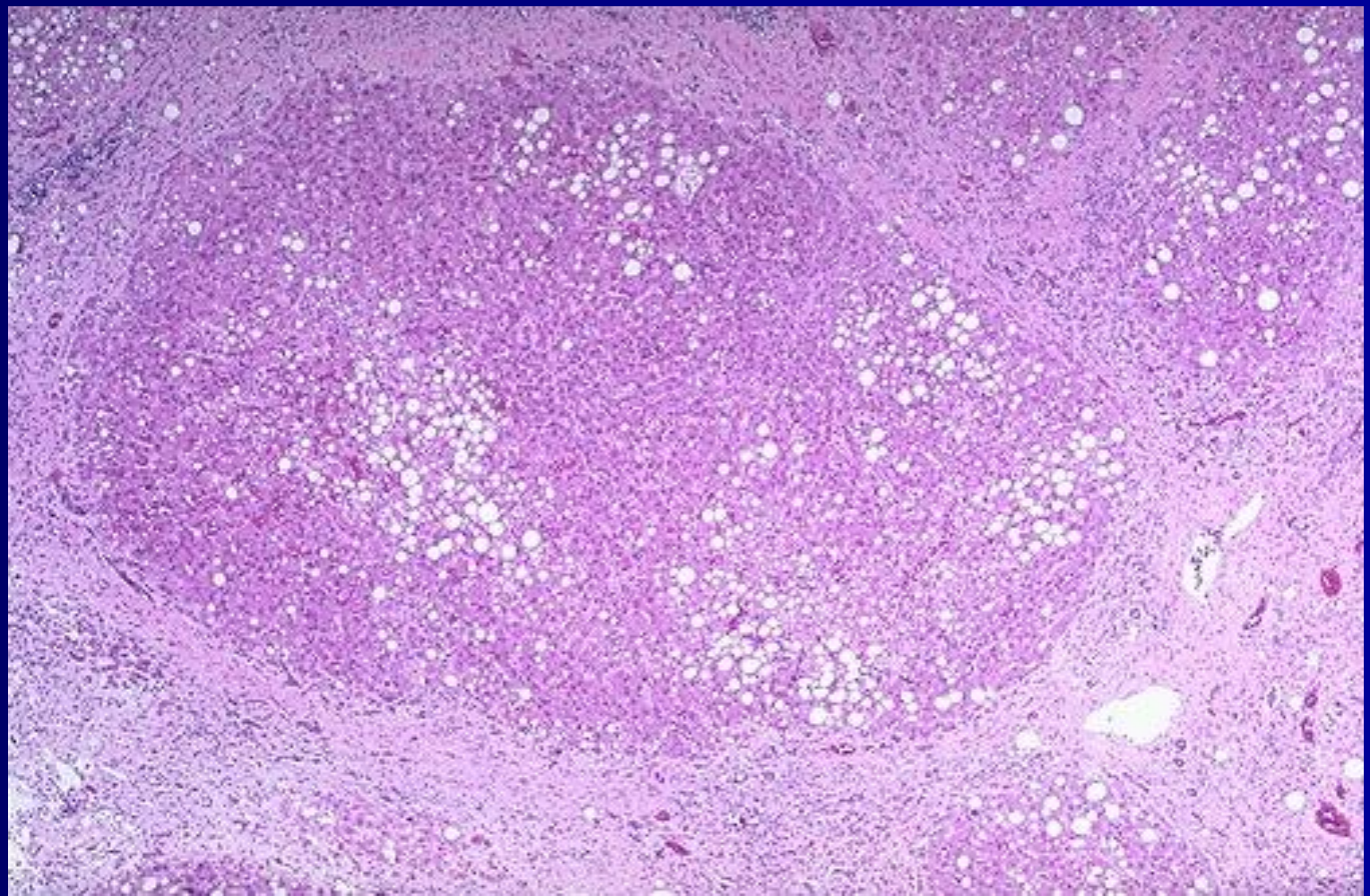
Biliary disease and hemochromatosis are less frequent causes.

Cirrhosis as the end stage of chronic liver diseases has three morphologic characteristics :

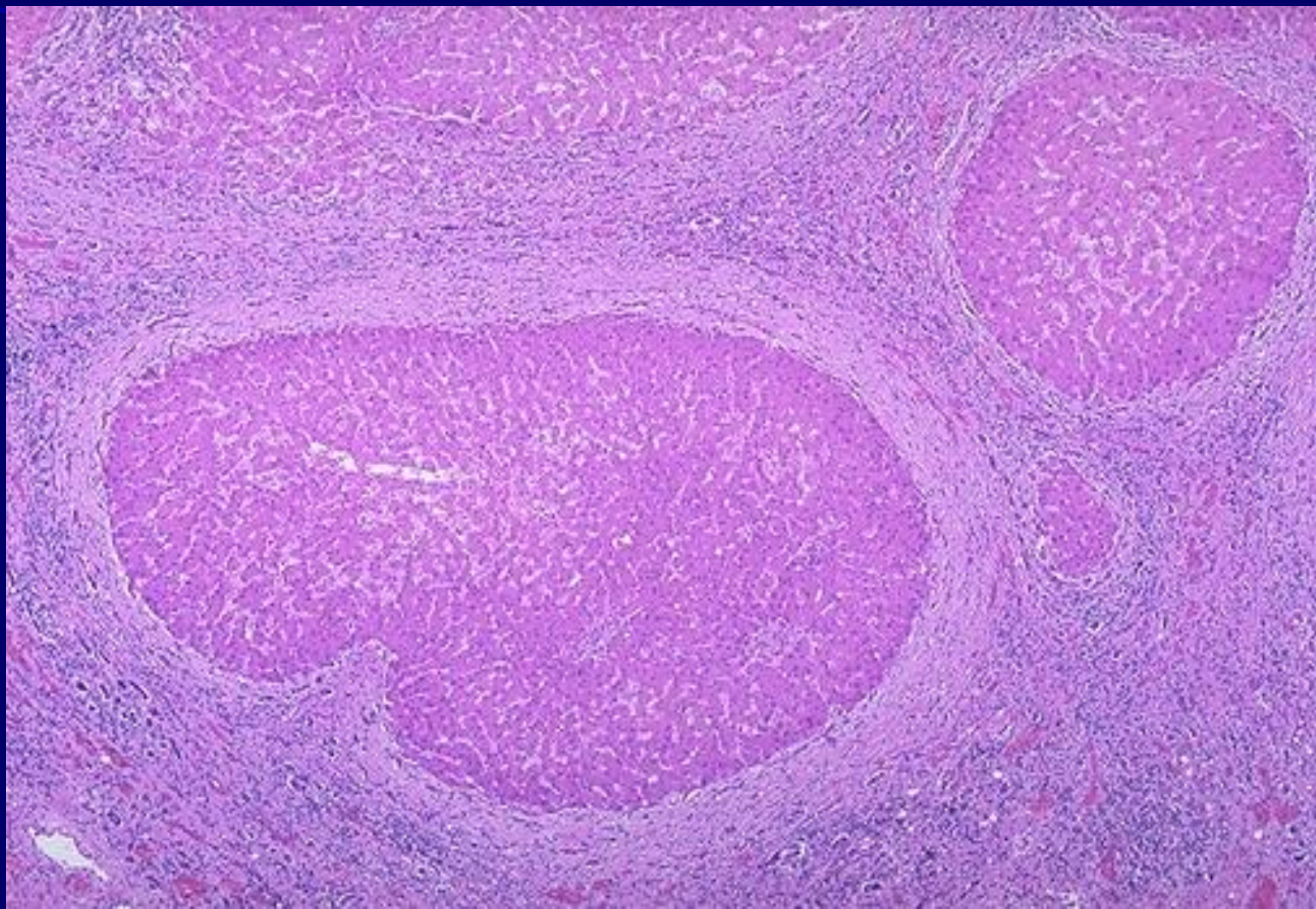
- Bridging fibrosis (collagen remodeling and accumulation – the main sign of perogressive liver damage) linking portal tracts to each other and to central
Veins.

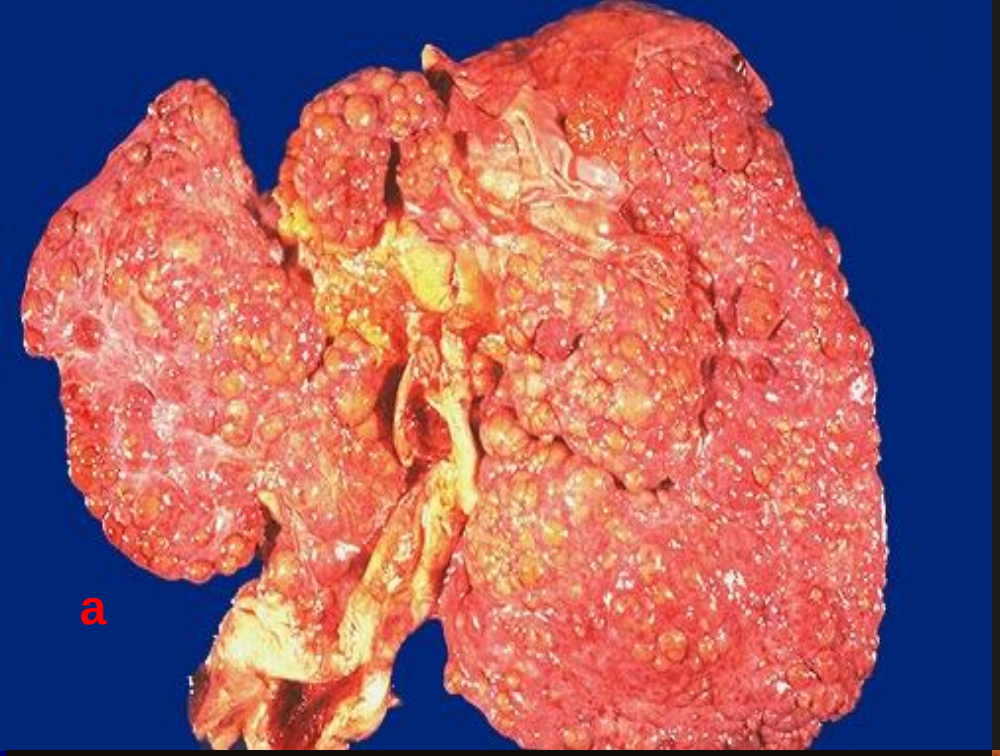


- Parenchymal nodules resulting from hepatocyte regeneration when encircled by fibrosis. A process of pseudolobule formation is observed The diameter of the lobules varies from small (<0.3 cm – micronudular cirrhosis of the liver) to large (a few centimeters – macronodular cirrhosis).



- Disruption of hepatic parenchymal architecture – parenchymal damage with subsequent fibrosis is diffuse and involves the entire liver tissue. Focal damage with subsequent scarring does not lead to cirrhosis of the liver, nor to diffuse nodular transformation without fibrosis.

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Clinical Features

Cirrhosis in 40% of the cases is clinically silent until far advanced. Death can be caused by:

- Progressive liver failure (as discussed previously)
- Complications of portal hypertension (see later discussion)
- Hepatocellular carcinoma (see later discussion).

Portal Hypertension

Portal hypertension results from a combination of increased flow into the portal circulation and/or increased resistance to portal blood flow; causes are:

- *Prehepatic: Thrombosis, portal vein narrowing, increased splanchnic arterial circulation, or massive splenomegaly with increased splenic vein blood flow*
- *Intrahepatic: Cirrhosis (most common), schistosomiasis, massive fatty change, granulomatous disease, or nodular regenerative hyperplasia*
- *Posthepatic: Right-sided heart failure, constrictive pericarditis, or hepatic vein obstruction.*

Major clinical consequences of portal hypertension are:

- *Ascites - a collection of excess serous fluid in the peritoneal cavity*
- *Portosystemic shunts which arise as portal pressures rise flow is reversed from the portal into the systemic circulation where there are shared capillary beds:*

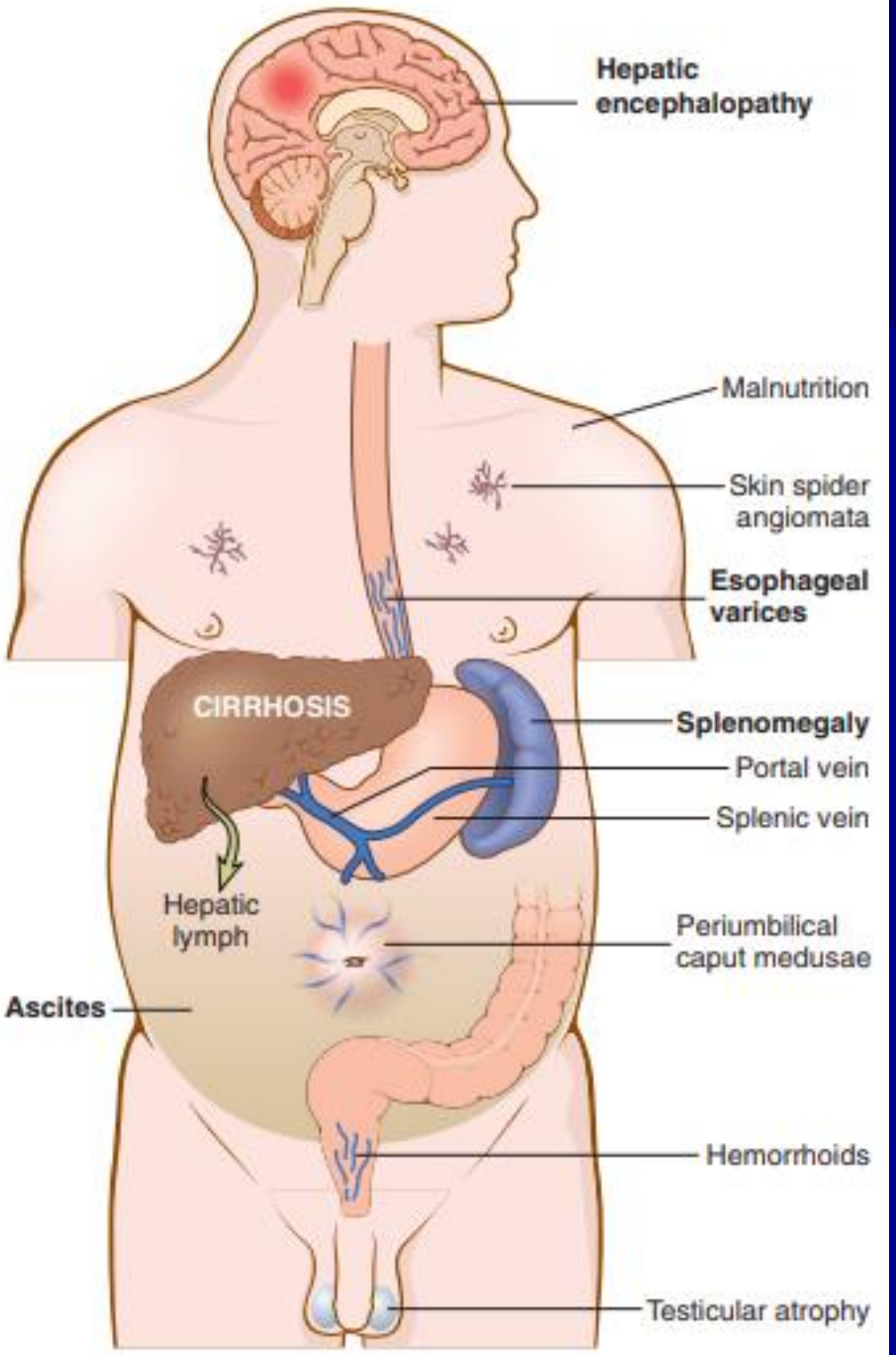
a. Esophagogastric varices (most significant), which occur in 40% of patients with advanced cirrhosis; these rupture and can cause massive hematemesis; each bleed has a 30% mortality.

b. Rectum (hemorrhoids).

c. Falciform ligament and umbilicus (caput medusa).

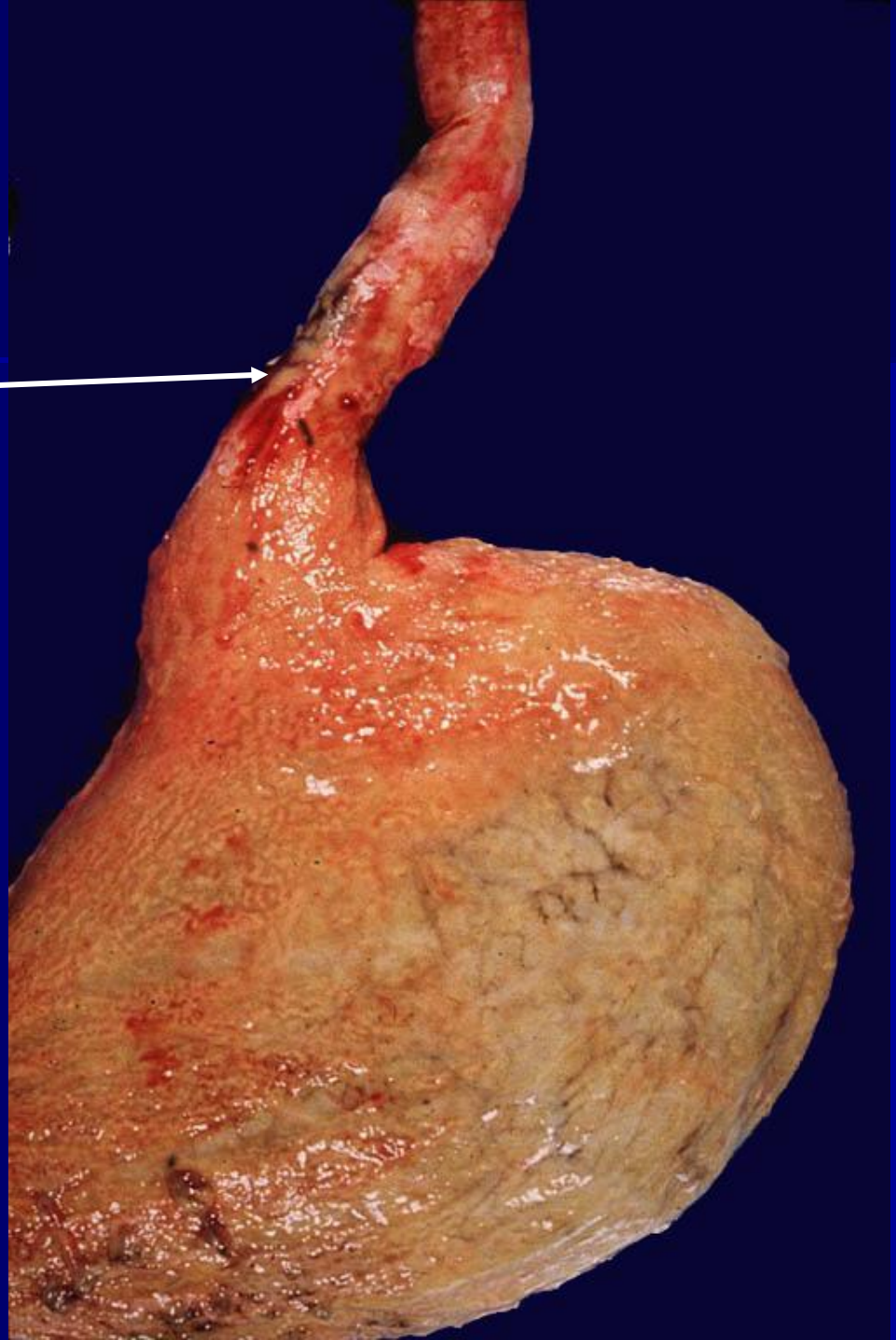
Major clinical consequences of portal hypertension are:

- Splenomegaly , caused by long-standing congestion.The degree of enlargement of the spleen is variable, the mass can reach 1 kg, however, this indicator does not necessarily correlate with other signs of portal hypertension. Massive splenomegaly can secondarily lead to thrombocytopenia and even pancytopenia due to hypersplenism.


Hepatic encephalopathy
Malnutrition
Skin spider angiomata
Esophageal varices
CIRRHOSIS
Splenomegaly
Portal vein
Splenic vein
Hepatic lymph
Periumbilical caput medusae
Ascites
Hemorrhoids
Testicular atrophy

cm 1 2 3 4

CM 1 2 3 4 5 6 7



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Infectious liver diseases

Viral Hepatitis

Liver damage occurs in the following viral diseases:

(1) infectious mononucleosis, the acute phase may be accompanied by mild hepatitis;

(2) cytomegalovirus infection, especially in newborns or immunocompromised patients;

(3) yellow fever, which is the leading cause of hepatitis in tropical countries. Occasionally, in children and immunocompromised patients, liver damage can develop in rubella, adenovirus, herpes virus and enterovirus infections.

In most cases, the term "viral hepatitis" is used to refer to an infectious liver lesion caused by a group of hepatotropic viruses (hepatitis A, B, C, D and E viruses) that have high affinity for liver cells.

Infectious liver diseases

Viral Hepatitis A

HAV causes a prognostically benign, self-limited disease with an incubation period of 3-6 weeks. HAV does not cause chronic hepatitis and also extremely rare causes fulminant hepatitis, and therefore mortality from HAV infection is -0.1%. AV is ubiquitous and is the causative agent of endemic disease in countries with poor sanitary and hygienic conditions, many of whose residents already have antibodies to HAV by the age of 10.

Infectious liver diseases

Viral Hepatitis A

HAV infection occurs with the use of contaminated water or food, and the virus is present in feces 2-3 weeks before and within 1 week after the development of jaundice. Thus, most infections occur after close contact with an infected person or as a result of the fecal-oral route of transmission of the pathogen over a specified period of time, which explains the outbreaks of the disease in schools and kindergartens.

Infectious liver diseases

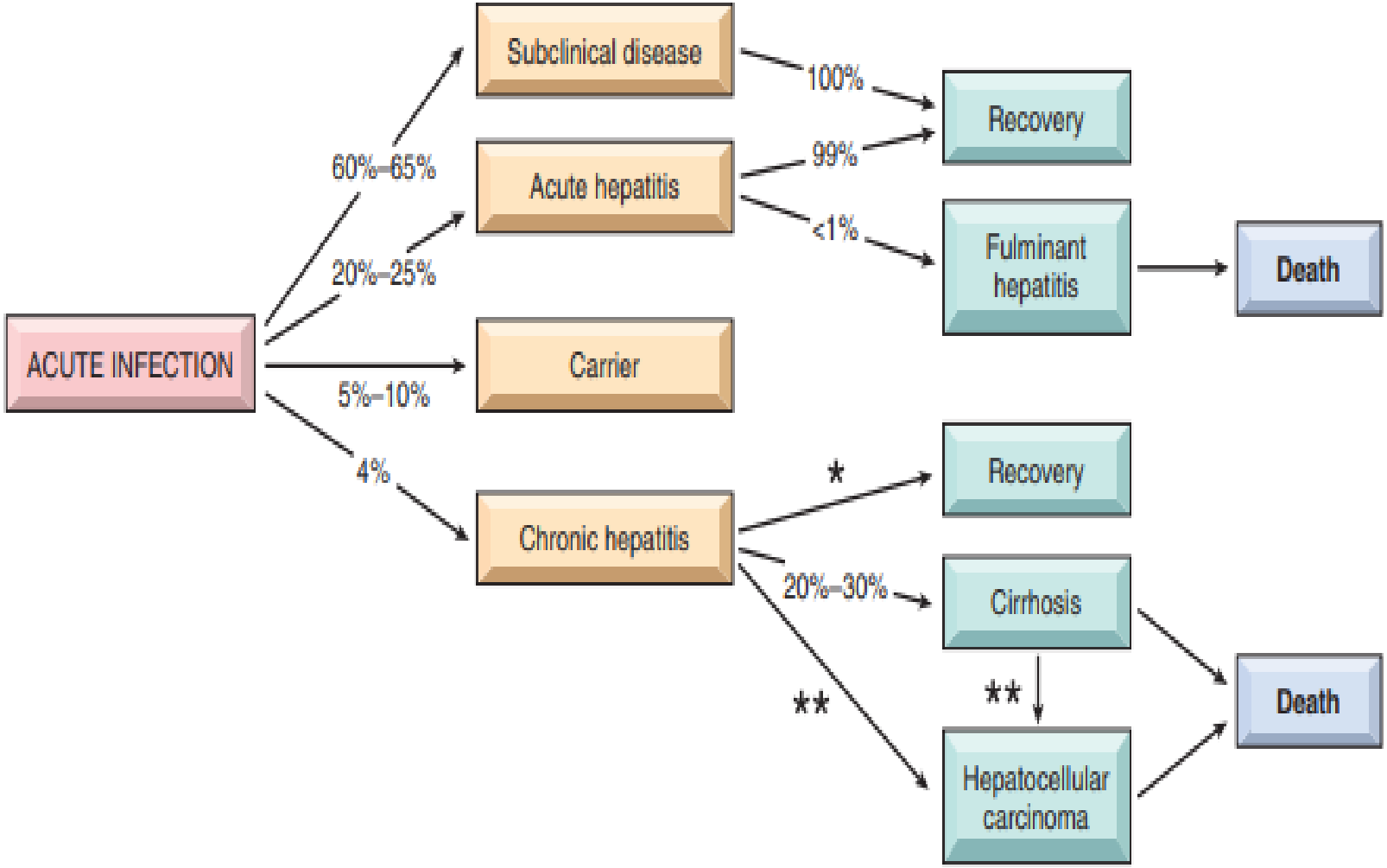
Hepatitis B Virus

HBV may cause:
(1)acute hepatitis with subsequent recovery and elimination of the virus;
(2) non-progressive chronic hepatitis;
(3) Progressive chronic disease culminating in cirrhosis (and increased risk of hepatocellular carcinoma)
(4) fulminant hepatitis with massive liver necrosis;
(5) asymptomatic carriage.

HBV-induced chronic liver disease precedes HCC.

Infectious liver diseases

Hepatitis B Virus



Infectious liver diseases

Hepatitis B Virus

The route of HBV transmission varies by geographic region. So, in areas with a high prevalence of HBV in 90% of cases, a vertical transmission route of the virus (during childbirth) is noted.

In regions with moderate prevalence, the main route of transmission is horizontal (in contact with the patient). In areas of low prevalence, such as the United States, the virus is mainly transmitted through unprotected heterosexual or homosexual intercourse and intravenous drug use (with the sharing of needles and syringes). **The spread of infection through blood transfusion has declined significantly in recent years due to screening of donated blood and donors for HBsAg.**

Infectious liver diseases

Hepatitis B Virus

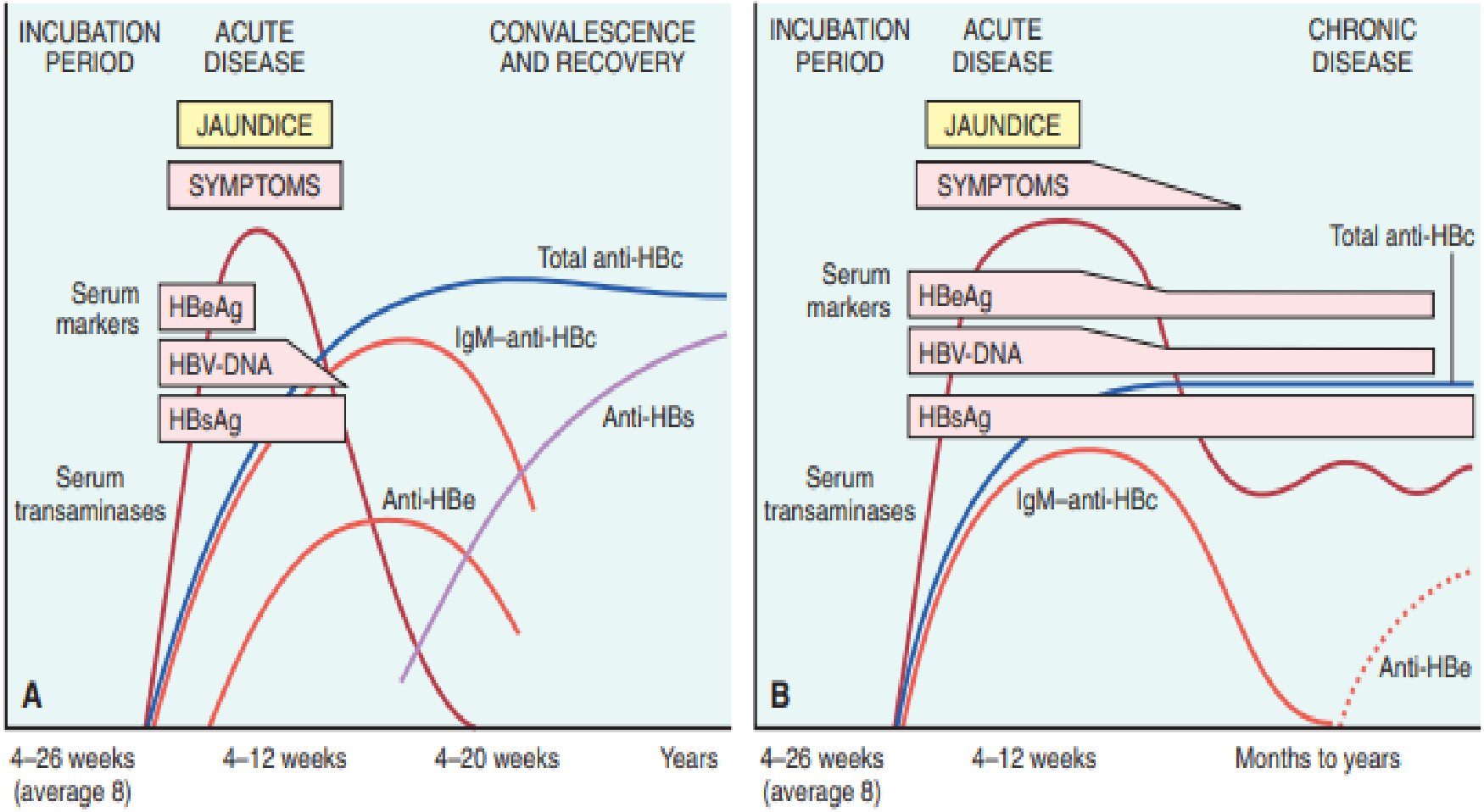
HBV infection is characterized by a long incubation period (4-26 weeks). Unlike HAV, HBV is detected in the blood both before and during the active phase of acute and chronic hepatitis. **The HBV genome has four open reading frames:**

- **Nucleocapsid core antigen (HBcAg), plus a longer polypeptide transcript (HBeAg) that is secreted into the bloodstream.**
- **Hepatitis B surface antigen (HBsAg) envelope glycoproteins (large, middle, and small);**
- **Polymerase with both DNA polymerase and reverse transcriptase activity; viral replication occurs through an intermediate RNA template: DNA ! RNA ! DNA.**
- **Hbx protein, a transcriptional transactivator of host and viral genes, necessary for viral replication.**

Infectious liver diseases

Hepatitis B Virus

In the natural course of the disease, the following serum markers can be determined:



Infectious liver diseases

Hepatitis B Virus

It is believed that damage to hepatocytes occurs as a result of cytotoxic T-lymphocytes CD8 + effect on the infected cells.

Infectious liver diseases

Hepatitis B Virus

HCV is the leading cause of liver disease worldwide: -170 million people are infected.
The incubation period of viral hepatitis C varies from 2 to 26 weeks, averaging 6-12 weeks. In almost 85% of patients, acute infection is asymptomatic and often not diagnosed.

In symptomatic acute HCV infection, anti-HCV antibodies are detected only in 50-70% of patients, in the remaining anti-HCV antibodies appear 3-6 weeks after infection.

Infectious liver diseases

Hepatitis B Virus

The clinical course of acute viral hepatitis C is milder than hepatitis B. Occasionally, a severe course is observed that is indistinguishable from that of viral hepatitis A and B.

Persistent infection and chronic hepatitis are typical signs of chronic HCV infection (acute infection is usually asymptomatic). The persistence of the virus and the chronic form of the disease are noted in 80-85% of cases. Liver cirrhosis can develop 5-20 years after acute infection in 20-30% of patients with persistent infection.

Infectious liver diseases

Hepatitis B Virus

In chronic HCV infection, HCV RNA for a long time circulates in the blood of many patients (more than 90% of patients with chronic disease), despite the presence of neutralizing antibodies.

A characteristic clinical sign of chronic HCV infection is a periodic increase in the level of transaminases in the blood serum, alternating with periods of decrease in their concentration to normal. Fulminant liver failure develops with HCV infection quite rarely.

Infectious liver diseases

Hepatitis B Virus

Hepatitis D virus (HDV), also called delta virus, is a unique RNA-containing virus whose life cycle depends on HBV. Different variants of the course of infection determines the type of HDV infection.

- acute co-infection develops when infected with blood serum containing both viruses, HDV and HBV. In this case, HBV replication must begin first so that HBsAg is formed, which is necessary for the development of HDV virions.
The frequency of disease progression to a chronic form corresponds to that of classical acute hepatitis B.

Infectious liver diseases

Hepatitis B Virus

- superinfection develops when HDV enters the host organism of a chronic HBV infection carrier. This leads to the development of the disease after 30-50 days. Superinfection of HDV in HBsAg carriers can occur in the form of severe acute hepatitis in a previously unrecognized HBV carrier or in the form of exacerbation of already existing chronic hepatitis B. Chronic HDV infection develops in 80-90% of these patients

Infectious liver diseases

Clinical and pathological syndromes of viral hepatitis:

1. asymptomatic acute infection with recovery (determined only serologically) - The disease is detected by chance, and the diagnosis is made on the basis of a minimal increase in the level of transaminases in the blood serum or the presence of antiviral antibodies in it (confirmation of an already transmitted infection).

Infectious liver diseases

Clinical and pathological syndromes of viral hepatitis:

2. **acute hepatitis with recovery (icteric or anicteric form) – Infection by any of the hepatotropic viruses has a similar clinical picture with 4 phases:**

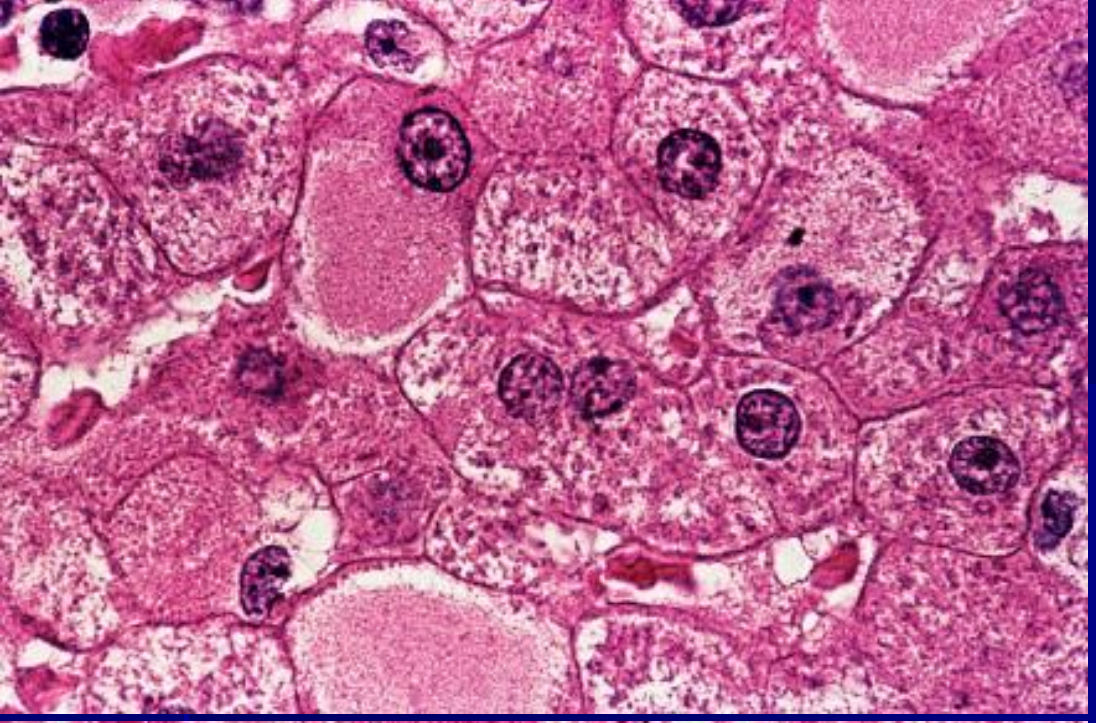
- **incubation period**
- **symptomatic preicteric phase**
- **-symptomatic icteric phase**
- **recovery**

Infectious liver diseases

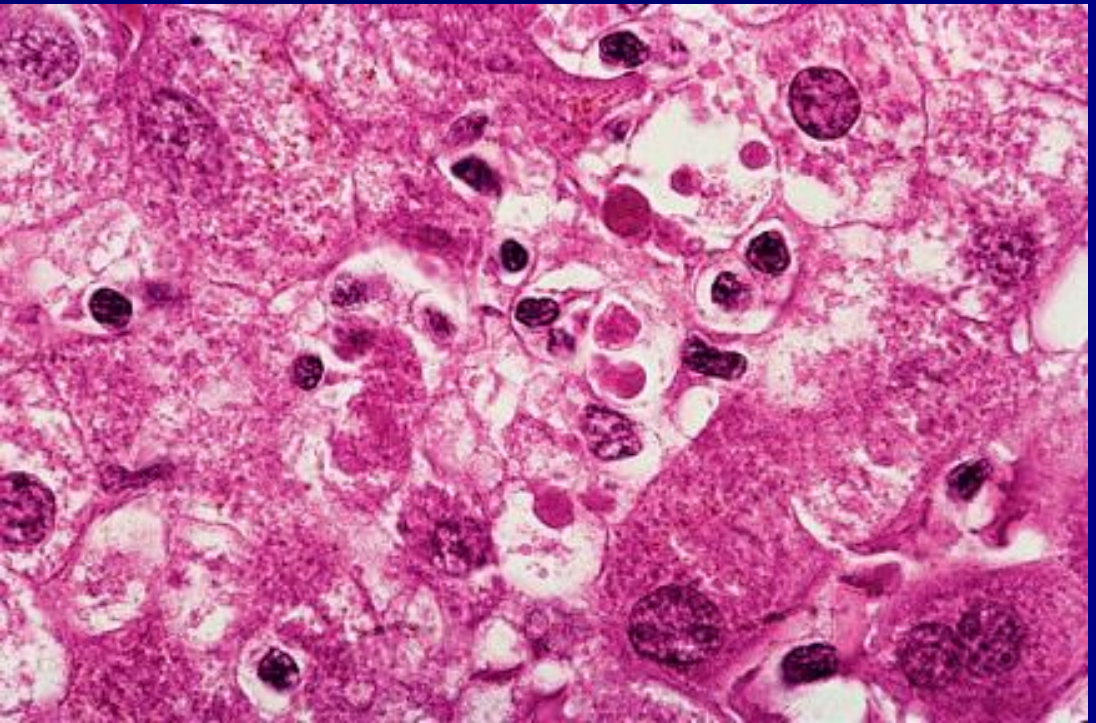
Clinical and pathological syndromes of viral hepatitis

3. chronic hepatitis (progressive or non-progressive to cirrhosis)

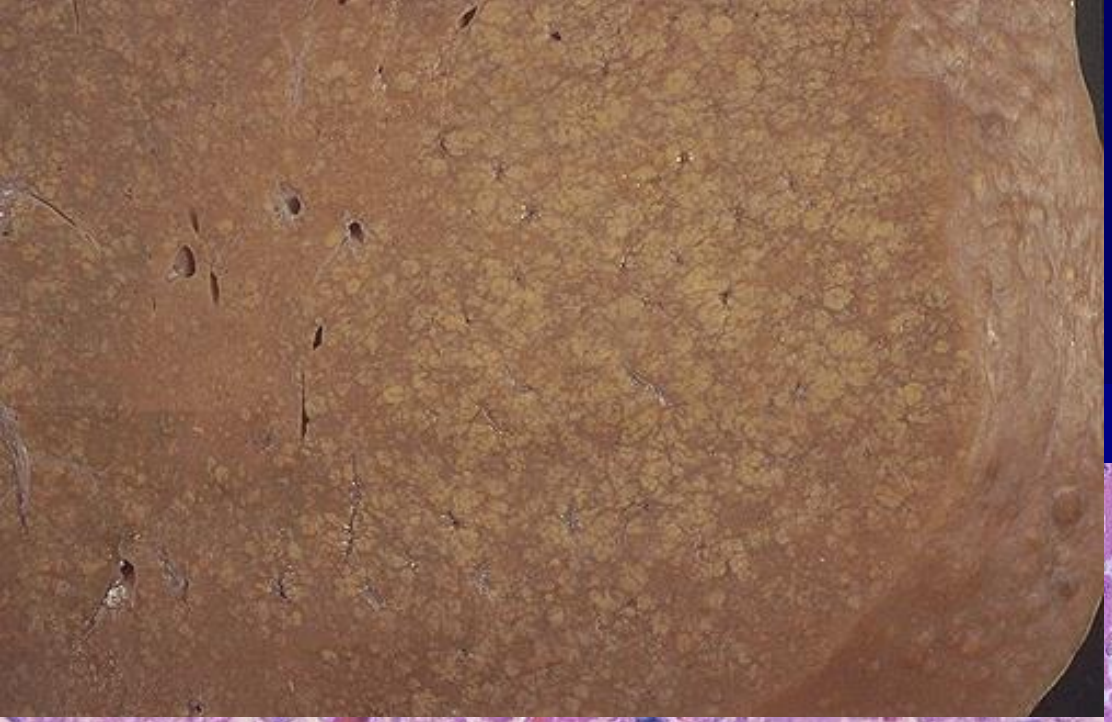
4. fulminant hepatitis (with massive or submassive liver necrosis)

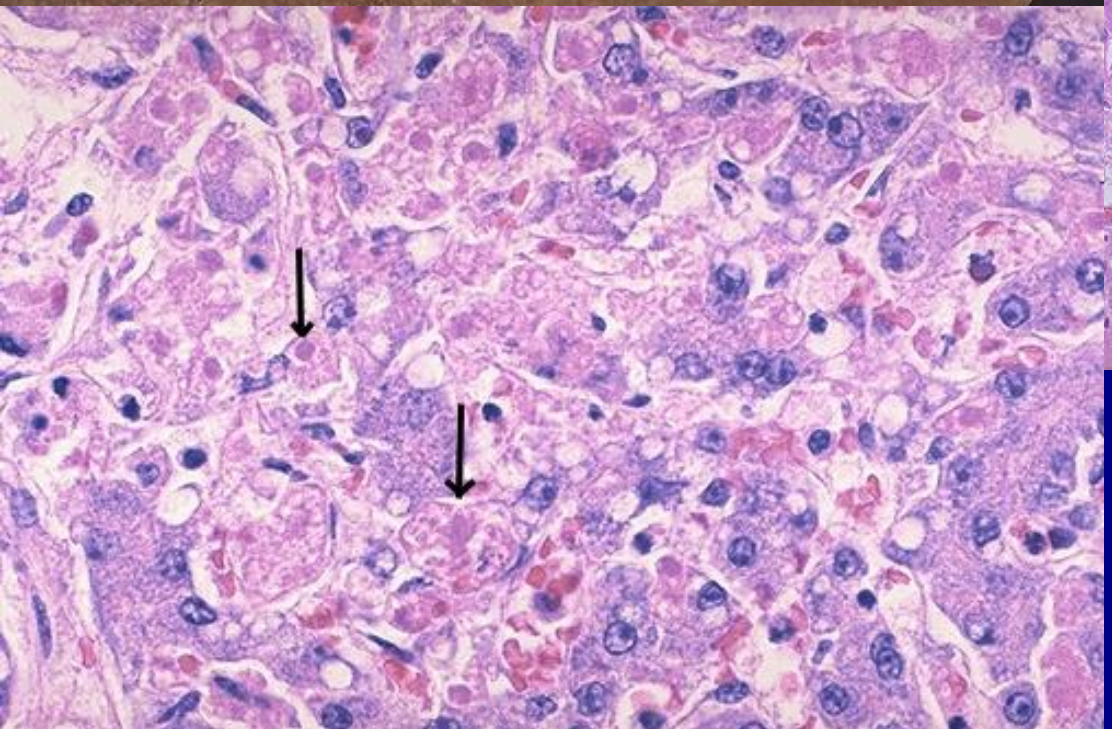


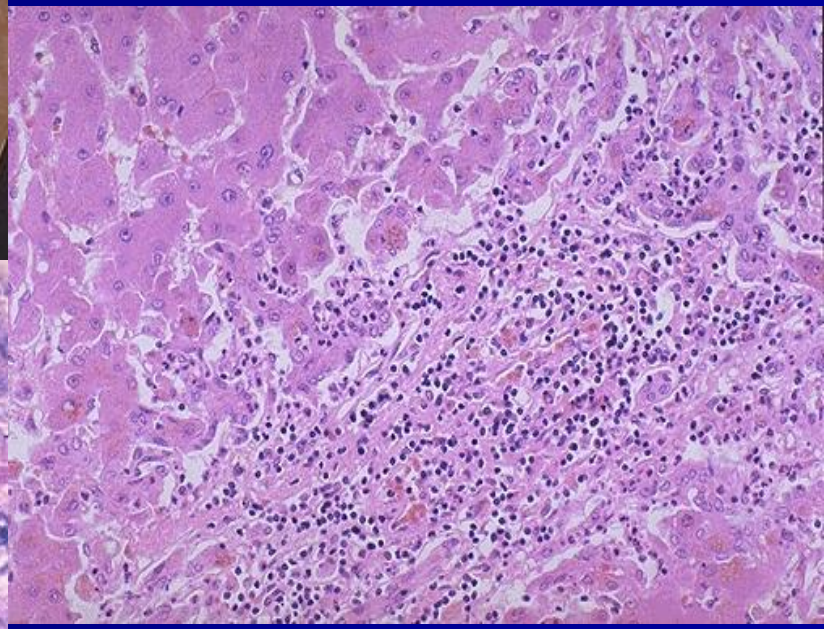
← the cytoplasm of hepatocytes affected by HBV has a soft-granular structure due to the presence of spherical particles and HBsAg tubes (opaque vitreous hepatocytes) in it.

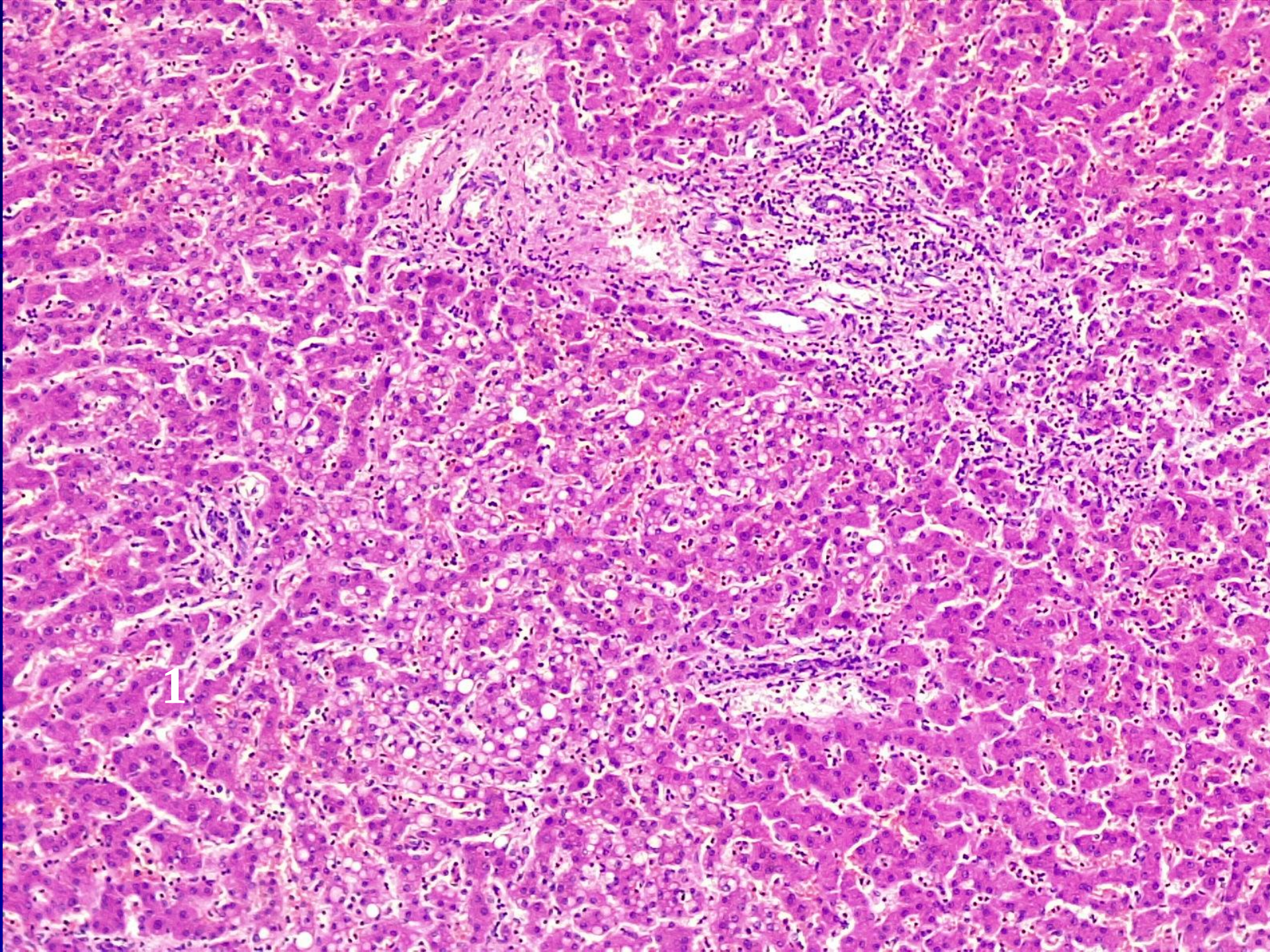


← Councilman corpuscles





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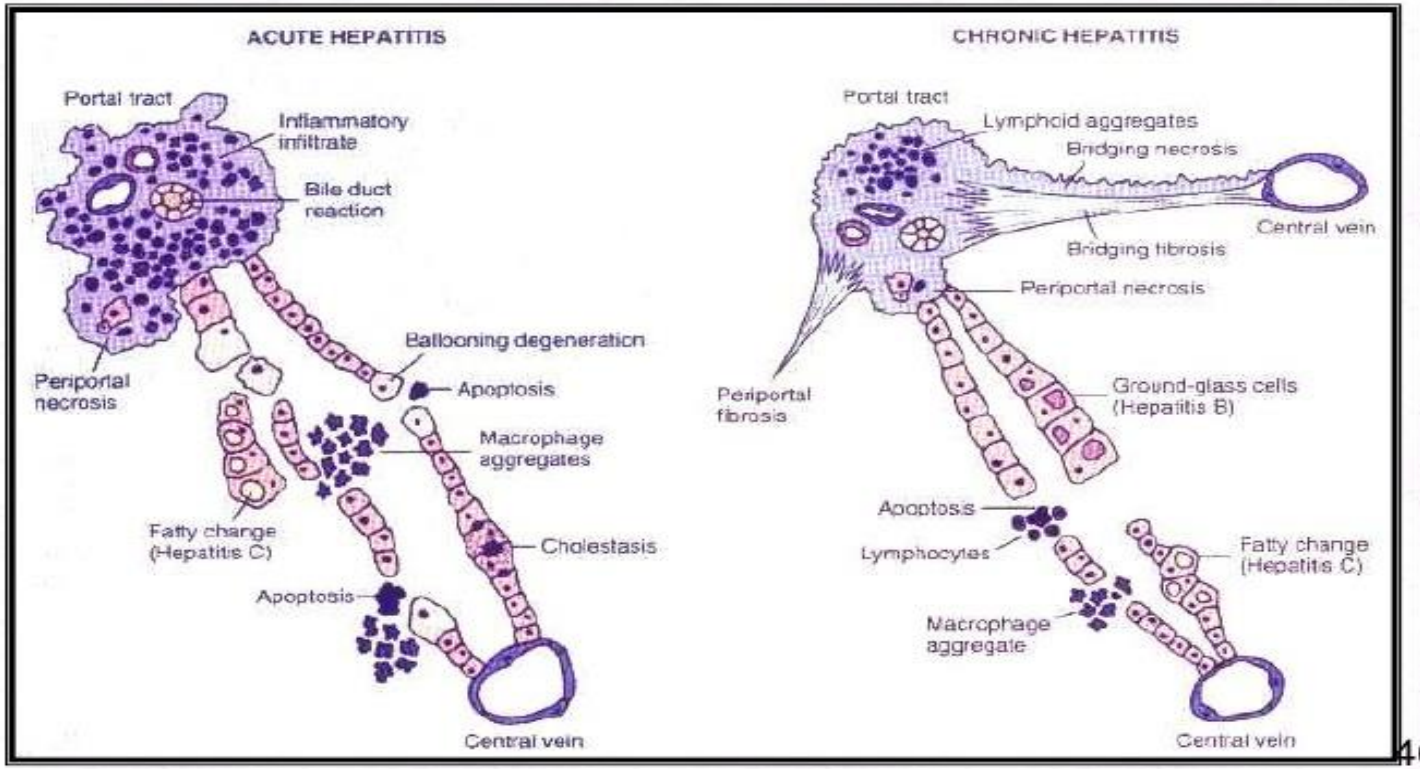
Infectious liver diseases

Morphological signs of acute and chronic viral hepatitis.

Acute - **Hepatitis** - Chronic



Alcoholic Liver Disease

There are 3 partially overlapping forms of alcoholic liver disease:

(1) fatty hepatosis;

(2) alcoholic hepatitis;

(3) cirrhosis.

Alcoholic Liver Disease

Morphology. Microvesicular lipid droplets within hepatocytes and can occur with even moderate alcohol intake. With chronic alcohol intake, lipid accumulates in macrovesicular droplets, displacing the nucleus. The liver becomes enlarged, soft, greasy, and yellow. There is little to no fibrosis (at least initially) and the condition is reversible.

Alcoholic Liver Disease

Macroscopically, the liver in case of fatty hepatosis caused by chronic alcoholism is increased in size (its mass reaches 4-6 kg), soft consistency, yellowish, on the cut section has lardaceous appearance.

At the initial stage of the disease, fibrosis is absent or slightly expressed, but with continuous alcohol abuse, fibrous tissue grows around the central veins with its spread to adjacent sinusoids. Fatty hepatosis is completely reversible if alcohol consumption is stopped.

Alcoholic Liver Disease

The following symptoms are characteristic for alcoholic hepatitis (alcoholic steatohepatitis):

- swelling and necrosis of hepatocytes. Single cells or their clusters swell and die. Cell swelling occurs as a result of intracellular accumulation of fats, fluids and proteins, which normally should be released from the cell.

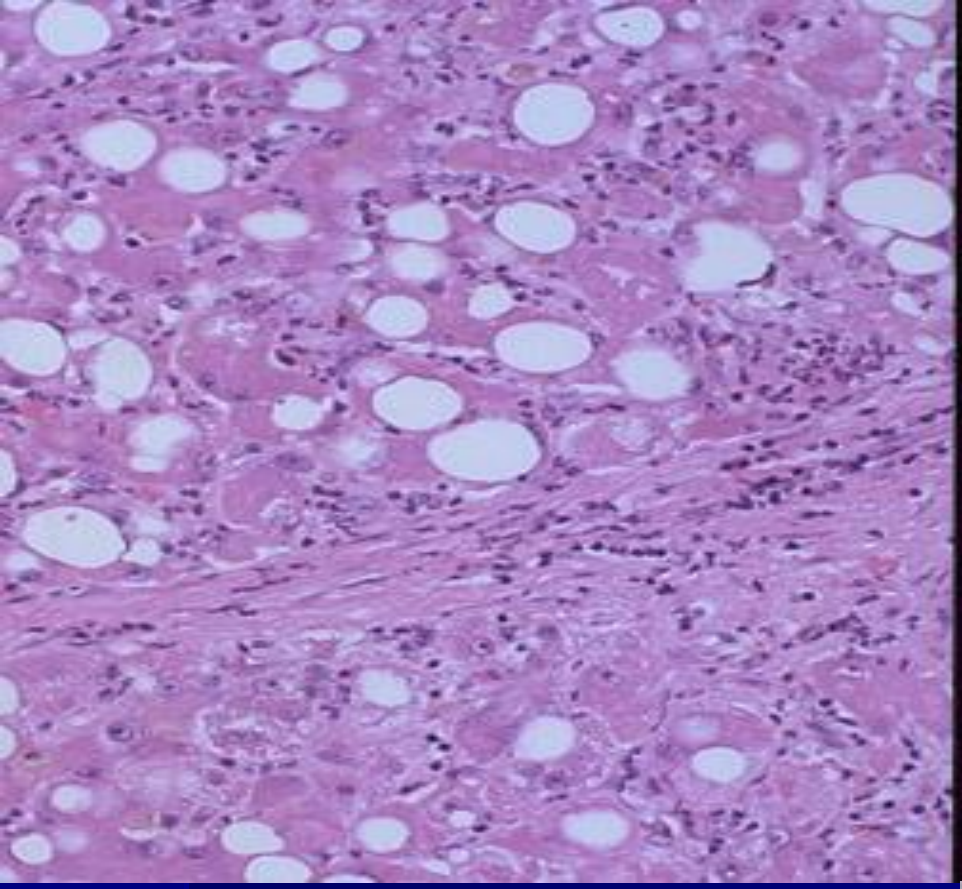
- Mallory's bodies. Individual hepatocytes accumulate cytokeratins of intermediate filaments. Mallory bodies have the appearance of eosinophilic inclusions in the cytoplasm of hepatocytes.

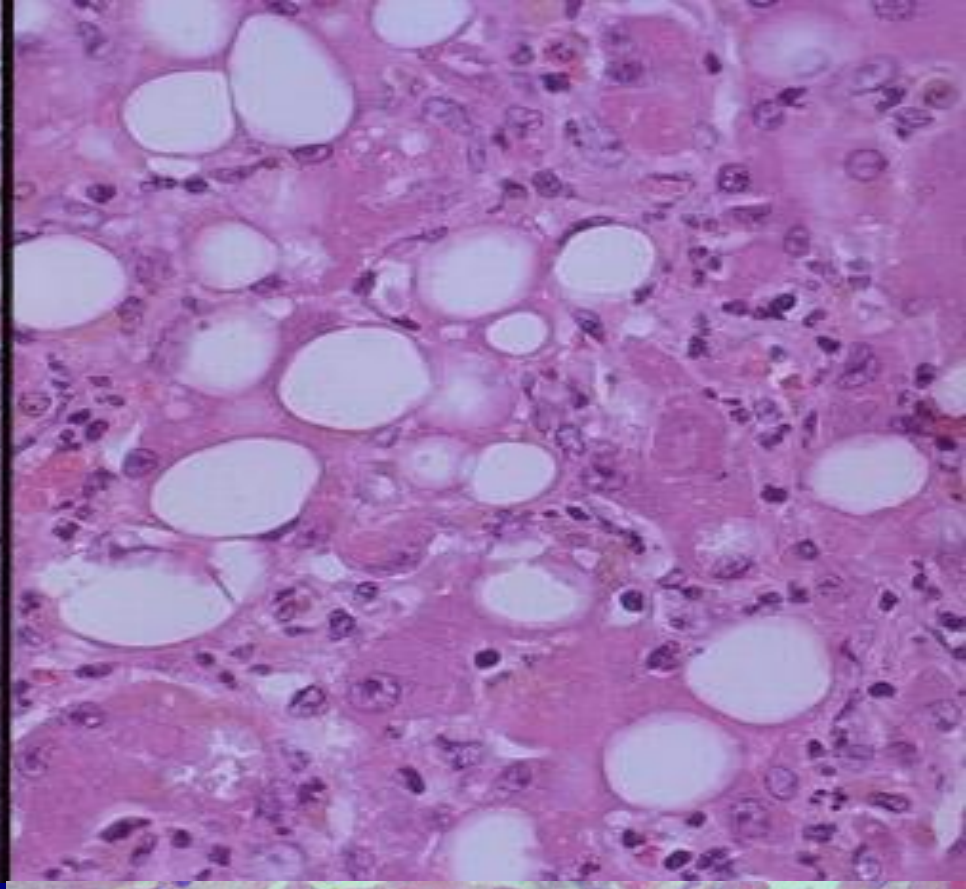
Alcoholic Liver Disease

The following symptoms are characteristic of alcoholic hepatitis (alcoholic steatohepatitis):

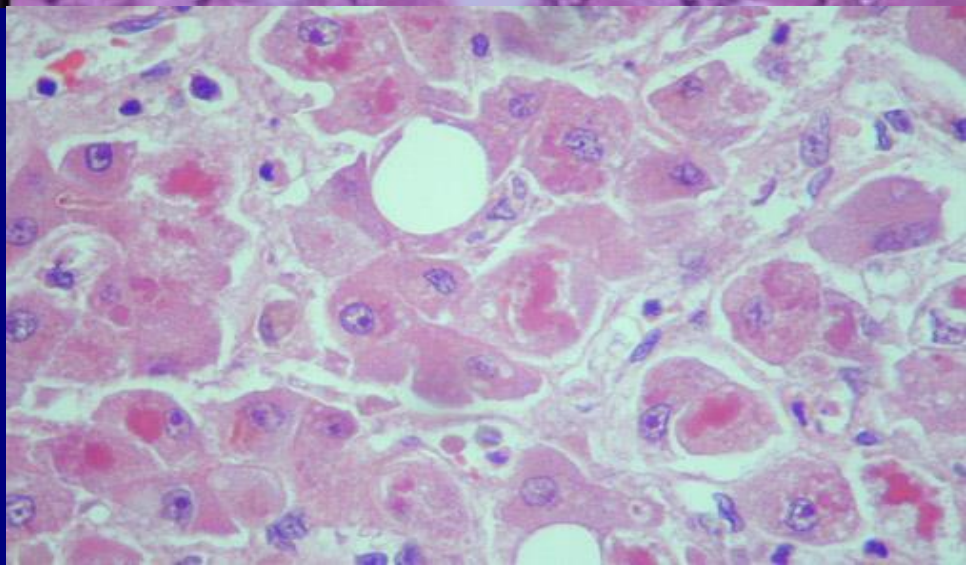
- neutrophil infiltration. Neutrophilic granulocytes penetrate the hepatic lobule and accumulate around damaged hepatocytes, especially those containing Mallory bodies.

- fibrosis. Alcoholic hepatitis is almost always accompanied by a pronounced activation of stellate cells of sinusoids and portal fibroblasts, which leads to the development of fibrosis.





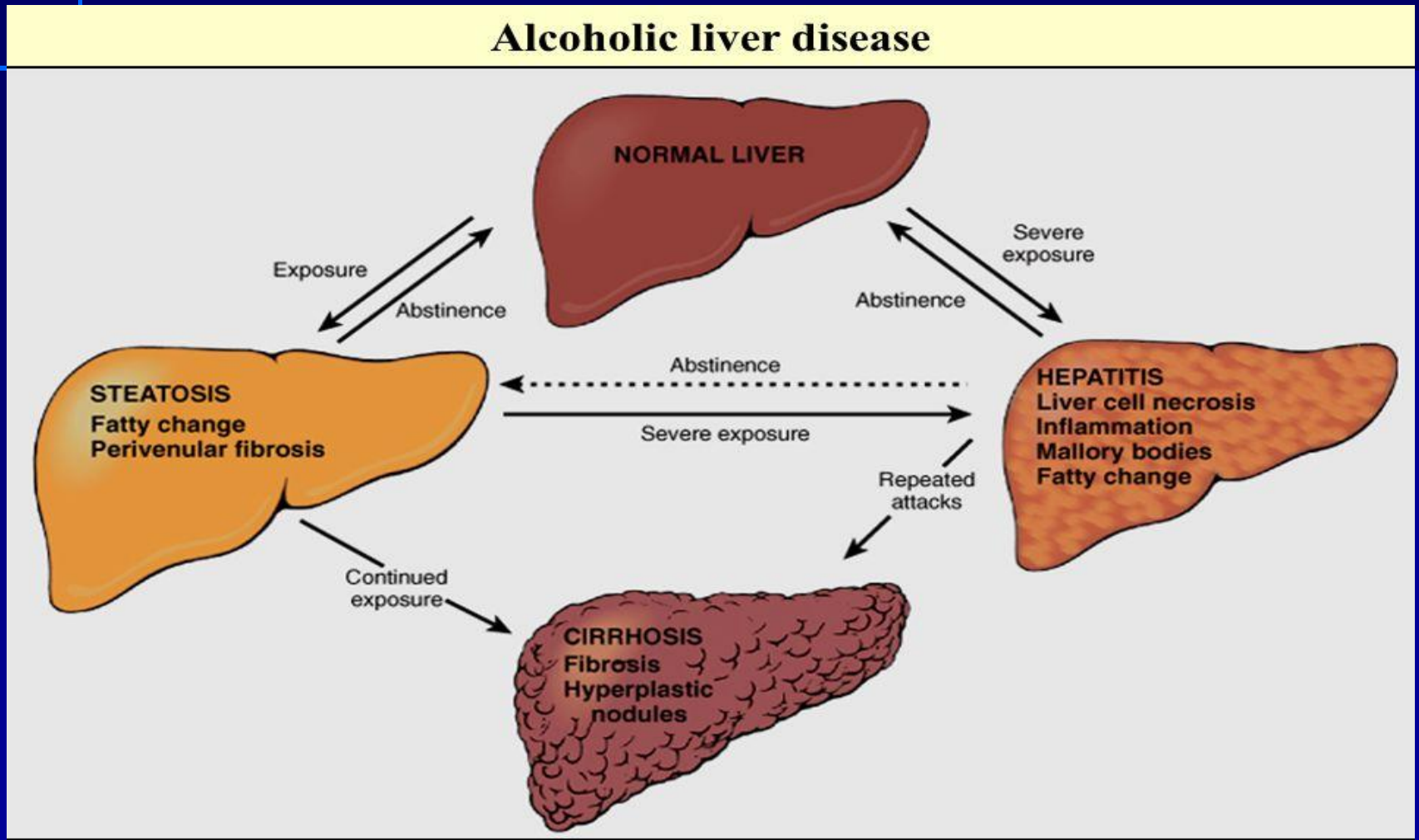
Alcoholic hepatitis
Mallory bodies and steatosis



Alcoholic Liver Disease

The final and irreversible form of alcoholic liver disease (cirrhosis) usually develops slowly and imperceptibly, however, in some cases, this period is reduced to 2 years from the start of systematic alcohol intake.

Alcoholic liver diseas



The interrelationships among hepatic steatosis, hepatitis, and cirrhosis are shown, along with a depiction of key morphologic features at the microscopic level.

Alcoholic Liver Disease

A special group is metabolic liver disease, both acquired and congenital. The most common acquired metabolic liver disease is non-alcoholic fatty liver disease. Among the hereditary metabolic diseases, the most significant are hemochromatosis, Wilson's disease and a1-antitrypsin deficiency.

Nonalcoholic Fatty Liver Disease

Non-alcoholic fatty liver disease is a group of diseases for which the presence of fatty hepatosis is common and which develop in people who do not abuse alcohol (less than 20 g of absolute alcohol per week).

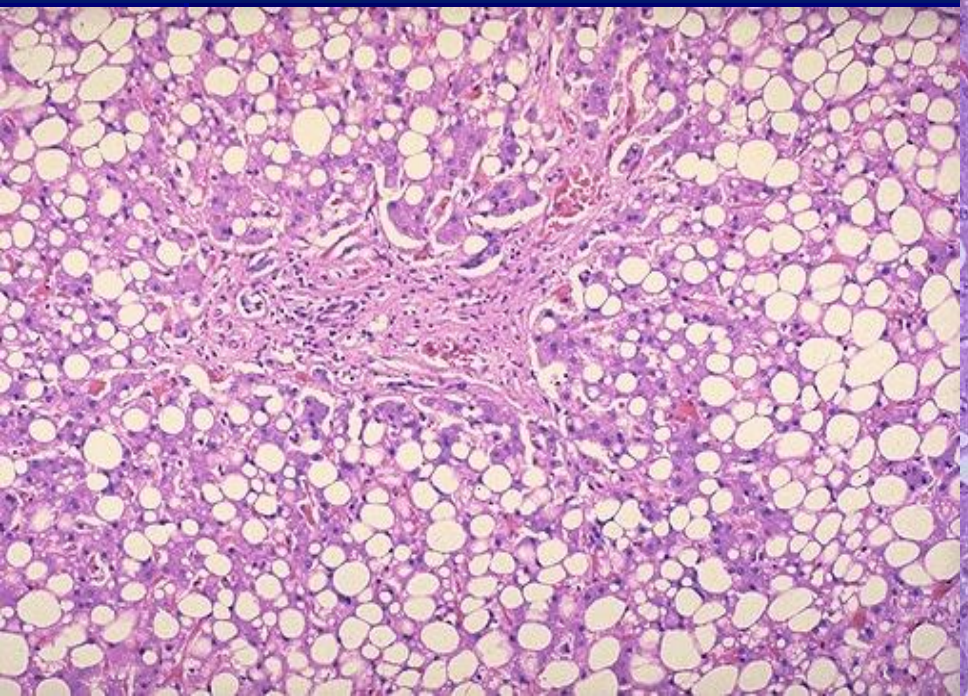
Non-alcoholic fatty liver disease includes:

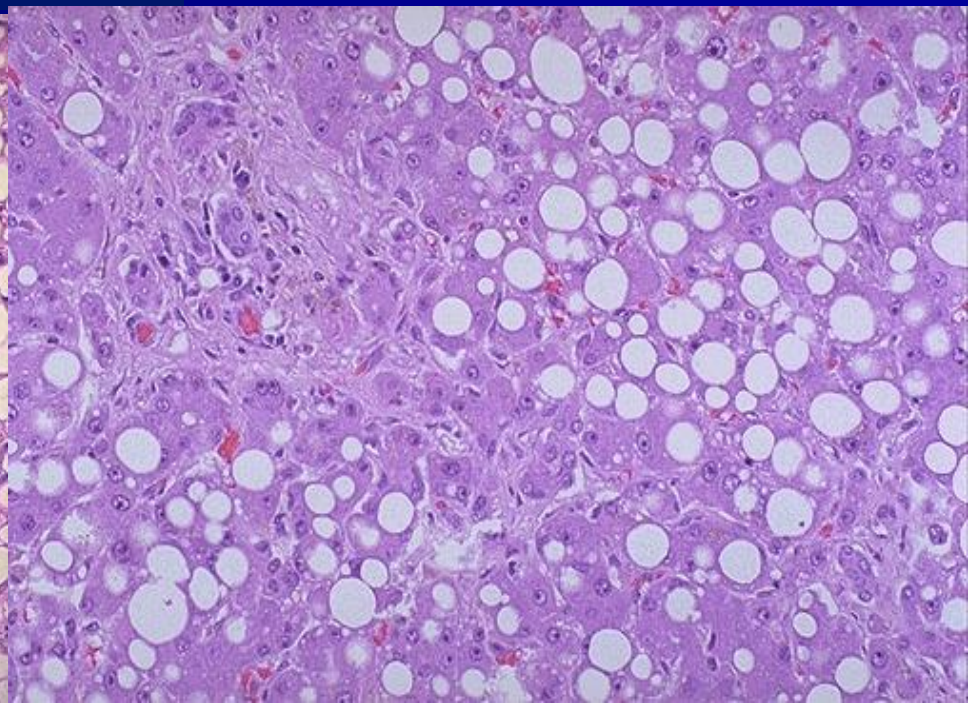
(1) simple fatty hepatosis;
(2) fatty hepatosis with non-specific inflammation;
(3) non-alcoholic steatohepatitis

Nonalcoholic Fatty Liver Disease

Morphology. In case of fatty hepatosis, the lesion, as a rule, affects at least 5% of hepatocytes (sometimes more than 90%). In the cytoplasm of hepatocytes, macrovesicular droplets of fat (large-drop fatty degeneration of hepatocytes) and microvesicular drops of fat (small-drop fatty degeneration of hepatocytes), consisting mainly of triglycerides, accumulate. In most cases, without pronounced clinical manifestations, there are no inflammatory changes, necrosis and scarring, and the only symptom is a long-term elevated level of liver enzymes in the blood serum.

Fatty hepatosis.





Nonalcoholic Fatty Liver Disease

Non-alcoholic steatohepatitis is characterized by fatty hepatosis and multifocal inflammatory parenchyma infiltration, mainly neutrophilic granulocytes, the presence of Mallory bodies, dead hepatocytes (due to balloon dystrophy and apoptosis) and sinusoid fibrosis. Fibrosis also develops in the area of the portal tracts and around the central veins. Histological changes are similar to those with alcoholic steatohepatitis.

Nonalcoholic Fatty Liver Disease

Non-alcoholic steatohepatitis with the same frequency is observed in men and women, while the disease is clearly associated with obesity and other components of the metabolic syndrome, for example, dyslipidemia, hyperinsulinemia and insulin resistance. It has been found that more than 70% of obese people have various forms of non-alcoholic fatty liver disease.

Nonalcoholic Fatty Liver Disease

As a result of prolonged subclinical progression of necrotic, inflammatory and fibrotic processes, cirrhosis of the liver may develop. It is characterized by a reduction in signs of fatty hepatosis and steatohepatitis (in some cases they cannot be detected at all).

Malignant tumors of the liver

Malignant tumors of the liver can be primary or secondary.
In most cases, primary liver carcinoma develops from hepatocytes - the so-called hepatocellular carcinoma. More rarely, carcinoma arises from the bile ducts (cholangiocarcinoma).

Malignant tumors of the liver

Hepatocellular carcinoma

More than 626 thousand new cases of primary malignant liver tumors are registered annually in the world, with almost all of them being HCC, and ~ 598 thousand patients die every year from liver cancer. HCC takes 3rd place in the structure of mortality from cancer.

Malignant tumors of the liver

Hepatocellular carcinoma

Four specific etiological factors are associated with the development of HCC:

chronic viral infection (HBV, HCV);

chronic alcoholism;

non-alcoholic steatohepatitis;

contaminated foods (primarily aflatoxin). - toxin produced by mushrooms

A. flavus infecting peanuts and grains.

Malignant tumors of the liver

Hepatocellular carcinoma

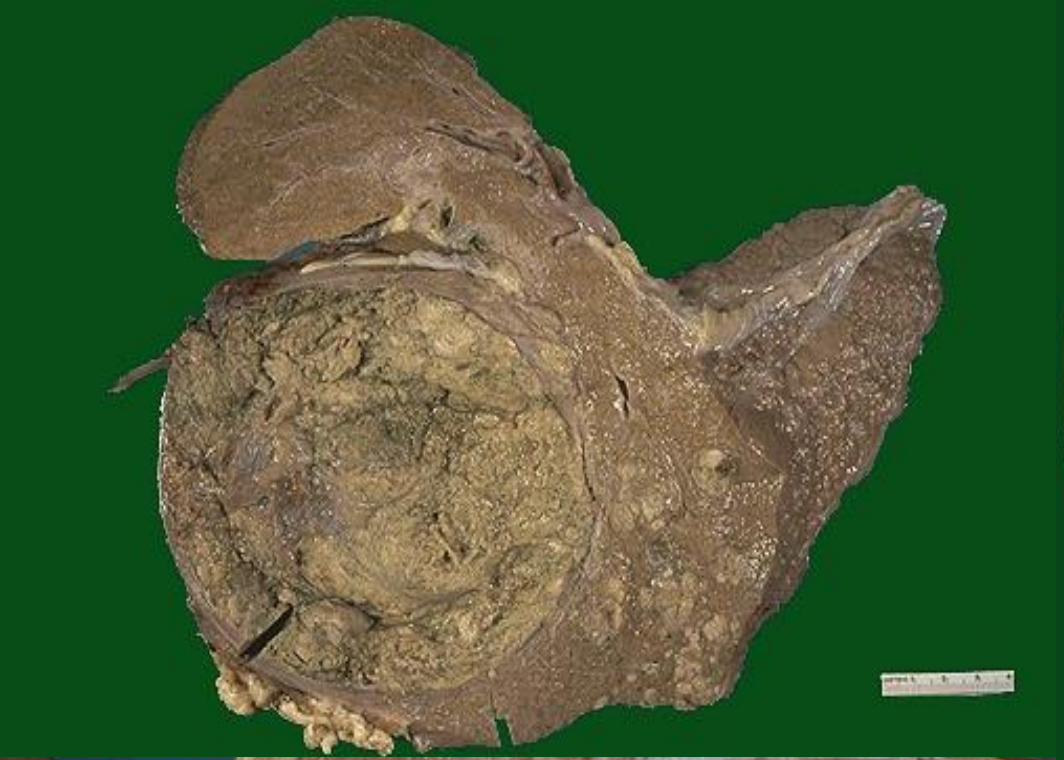
Morphology. With a macroscopic examination, the HCC may look like:

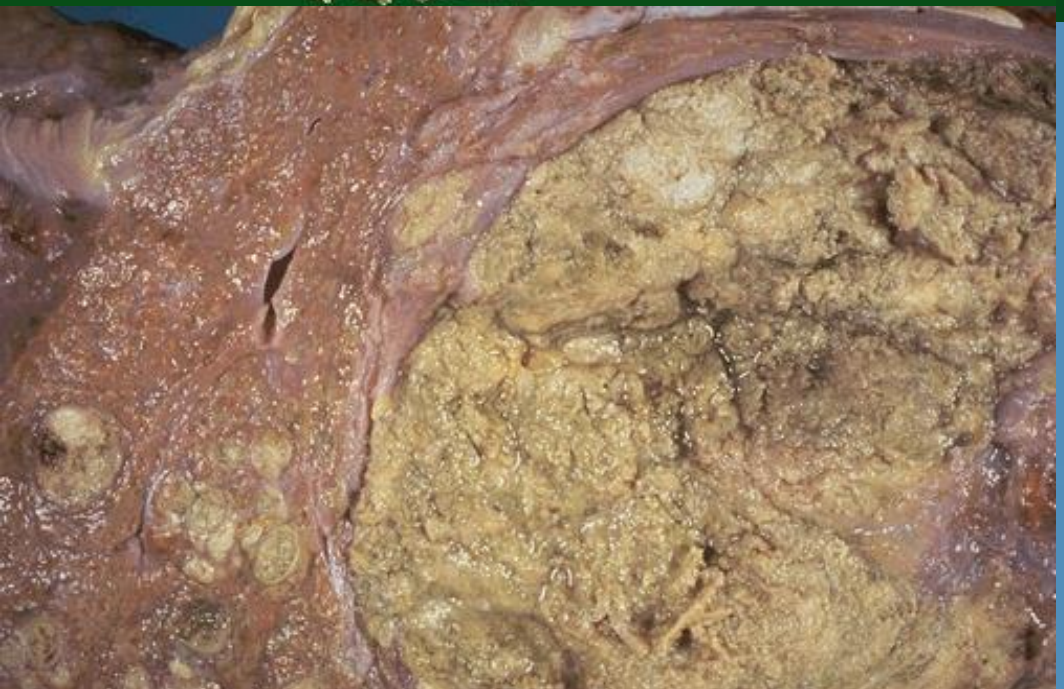
single (usually large) node

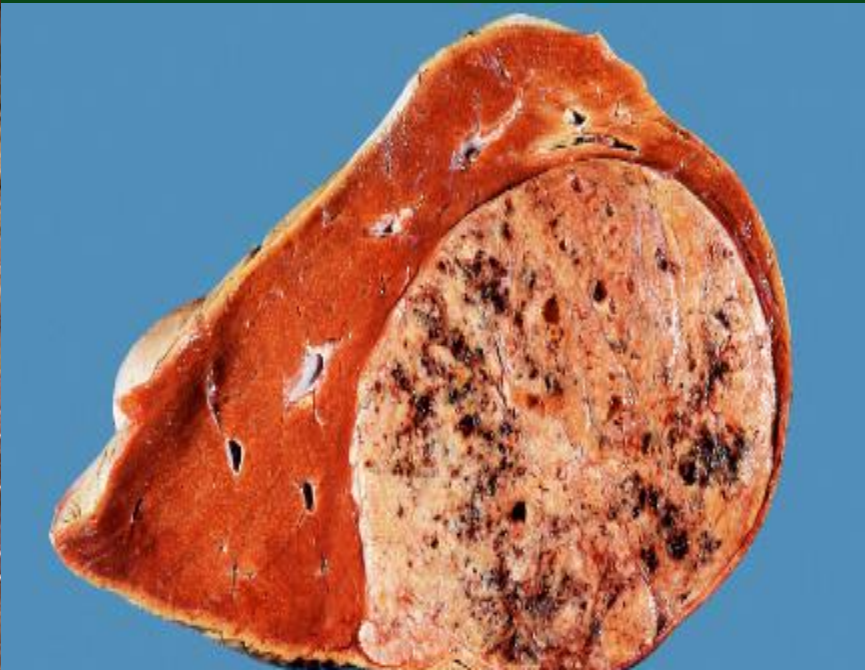
multiple scattered nodules of various sizes;

diffuse infiltrative tumor, occupying most of the liver, and sometimes replacing all of its tissue.

Diffuse infiltrative tumor can develop imperceptibly against the background of cirrhosis. All these types of fcc, but especially HCC in the form of a single node or many small nodules, can lead to hepatomegaly.







Malignant tumors of the liver

Hepatocellular carcinoma

Usually a tumor in the liver is more pale than the surrounding tissue, and sometimes the tumor has a greenish tint if it consists of highly differentiated hepatocytes that retain the ability to secrete bile. All types of HCC have a high predisposition to vascular invasion. As a result, common intrahepatic metastases appear.

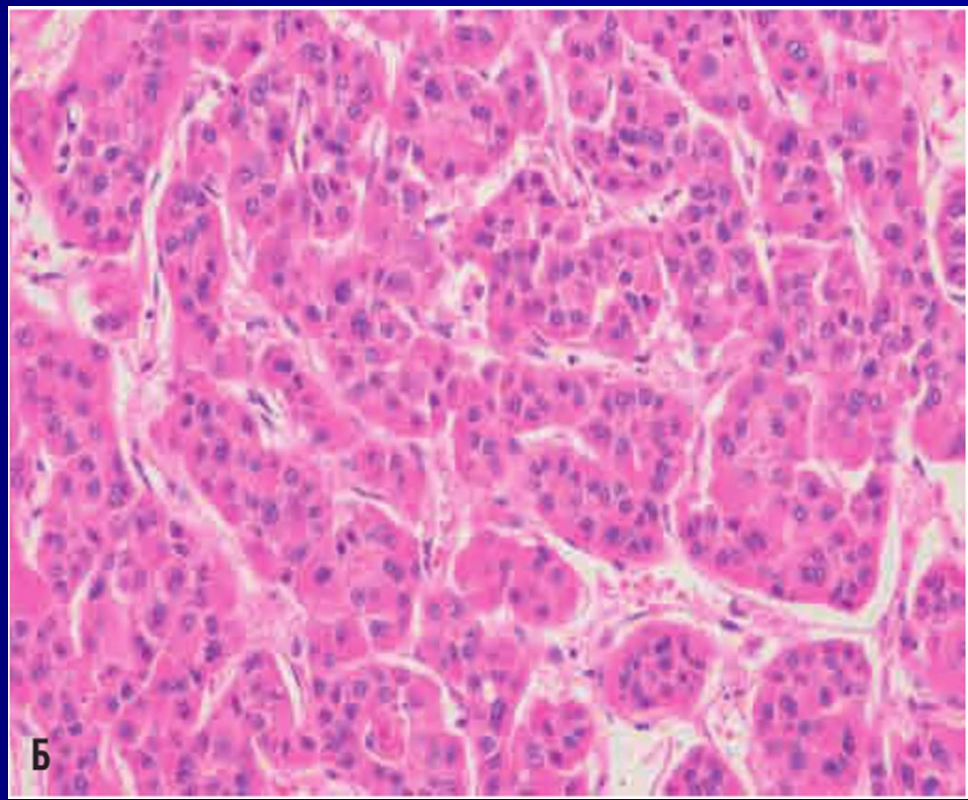
Malignant tumors of the liver

Hepatocellular carcinoma

The spread of the tumor outside the liver usually occurs by invasion of the tumor into the hepatic veins, however, at later stages of the disease hematogenous metastasis is characteristic, especially to the lungs. In less than 50% of HCC cases with spread beyond the liver, metastases are found in the lymph nodes in the area of the liver gate, peripancreatic and paraaortic lymph nodes above and below the diaphragm. If fcc with signs of venous invasion is detected in the explanted liver at the time of transplantation, there is a high probability of tumor recurrence in the donor liver.

Malignant tumors of the liver

Hepatocellular carcinoma

Histological structure of the hepatocellular carcinoma varies from highly differentiated to anaplastic undifferentiated forms. In highly differentiated and moderately differentiated tumors, cells similar to normal hepatocytes form acinar and pseudo-glandular structures, as well as trabecular structures thar are replacing the normal liver structures.

With low-grade tumor cell forms polymorphic, determined numerous anaplastic giant cells.