Hepatitis

Hepatitis is inflammation and damage of the liver tissue by various etiological agents, including **both infectious and non-infectious causes**. The most common infectious agents include **viruses** – viral hepatitis, less often bacteria (e.g., Leptospira). Non-infectious causes include **toxins**, **drug abuse**, or for example **autoimmune diseases**. The cell damage is a result of either **a direct cytotoxic effect** (toxin-mediated necrosis) or **the body's response** to the harmful agents (viral hepatitis).The clinical picture is very diverse and includes an asymptomatic course, nausea, abdominal pain, jaundice, and liver failure, depending on the extent of the ongoing necrosis and the liver tissue infiltration by inflammatory cells.

Types

Hepatitis can be classified according to several different criteria.

- According to the clinical course, we can classify it as:
  - **acute** (if it lasts less than 6 months)
  - **chronic** (if it does not subside after 6 months)
- according to histological criteria (the extent of the ongoing fibrotic changes and the reconstruction of the lobar architecture of the liver tissue)
- according to the cause:
  - infectious
  - toxic
  - autoimmune
  - hereditary diseases associated with hepatitis

Infectious hepatitis

Infectious hepatitis is caused by either viruses, which can be further divided into hepatotropic viruses (hepatitis A-E virus) and nonhepatototropic viruses, or into RNA and DNA viruses, or by bacteria and fungi.

- Viral agents include:
  - RNA viruses, the most common responsible hepatotropic viruses are HAV, HCV, HDV, HEV (these viruses replicate mainly in the liver and cause inflammatory degenerative changes) and nonhepatotropic include for example the yellow fever virus and the coxsackie virus
  - The most common respomsible DNA viruses are: HBV, EBV, CMV and HSV.
- Bacterial agents include Leptospira (Weil´s disease), Brucella, Coxiella burnetii (Q fever), Salmonella (abdominal typhus)

Non-infectious hepatitis

Non-infectious hepatitis is caused by liver damage from external influences or as a result of an already existing autoimmune disease. The causes include:

- alcohol abuse
- drug abuse – paracetamol, halothane
- poisoning – fungal toxins and other toxins
- autoimmune causes (for autoimmune hepatitis, we determine the following antibodies: antinuclear (ANA), antimitochondrial (AMA), liver membrane antibodies (LMA) and others
- metabolic diseases (e.g., Wilson's disease)

Acute hepatitis

The most common cause of acute hepatitis is **alcohol abuse** and **viral hepatitis**. Toxic damage, paracetamol and halothane and many others are also common causes of acute hepatitis.

Chronic hepatitis

Chronic hepatitis has manifestations that **do not subside after 6 months** (laboratory and histopathological changes persist for more than 6 months - e.g., elevated liver enzymes, especially ALT, AST). Of the viral hepatitis, the ones that turn chronic most often are:

- hepatitis C (85-90% of the total number of symptomatic courses)
- hepatitis D (which only occurs with HBV as a coinfection or superinfection)
- hepatitis B, which becomes chronic in 10-15% of cases.

Besides viral hepatitis, these can also cause chronic hepatitis:

- **autoimmune causes**
  - autoimmune hepatitis
  - primary sclerosing cholangitis

    - primary biliary cirrhosis
- **alcoholic hepatitis**,
- **drug abuse**
- **metabolic diseases**. The most common are:
    - α1-antitrypsin deficiency
    - Wilson's disease
    - Hemochromatosis

Unlike in acute hepatitis, the cause is more difficult to diagnose in chronic hepatitis. One can only speculate whether it is an autoimmune cause, drug damage, antibody-negative hepatitis, etc... For this reason, a biopsy is very often indicated.

Clinical examination

Patients usually complain of **decreased activity, fatigue, loss of appetite, arthralgia, and intermittent diarrhea**. The liver is palpably sensitive. Patients with low inflammatory activity in the liver tissue may also have an **asymptomatic course**. There is a temporary acute **flare-up of the chronic course** – during this inflammatory flare-up there is **jaundice**, the liver is enlarged and palpable. The spleen may also be enlarged. Typically, in the biopsy we find inflammatory activity in the liver tissue, as well as impaired metabolic function of the liver - menstrual disorders and amenorrhea in women, testicular atrophy, gynecomastia, and lack of body hair in men. Immunopathological reactions may occur – especially type III. immunopathological reaction, when antibody complexes with viral components are formed, in which case the hepatitis is accompanied by extrahepatic diseases such as cryoglobulinemia , Panarteritis nodosa, glomerulonephritis, polyarteritis nodosa, and others.

Metabolic liver diseases

There are many metabolic liver diseases due to the important role of the liver in metabolism. It can be a disorder of the metabolism of lipids, carbohydrates, amino acids, urea, metals, porphyrins, or a disorder of the transport of bile pigments. Hereditary and congenital metabolic disorders:

Hereditary disorders of lipid metabolism: Wolman's disease and cholesterol storage disease, Niemann-Pick complex, Gaucher's disease, abetalipoproteinemia, β-oxidation disorders

Hereditary disorders of carbohydrate metabolism: glycogenosis, galactosemia, hereditary fructose intolerance

Hereditary disorders of amino acid metabolism: tyrosinemia and defects in branched-chain amino acid metabolism

Congenital disorders of urea metabolism

Congenital disorders of metal metabolism: hemochromatosis, Wilson's disease

Congenital disorders of porphyrin metabolism: hepatic porphyria

Hereditary disorders of bile pigments transport [1]

Clinical picture

Hepatitis occurs with varying intensity of clinical manifestations.

Inapparent form
This form is completely asymptomatic
Abortive form
Short-term illness with flulike symptoms or dyspepsia. It is often accompanied by a slight increase of aminotransferase
Icteric form

Anicteric form
It is the most common form in all types of hepatitis.
Cholestatic form
Reminiscent of obstructive jaundice.
Malignant or fulminant form
Liver failure, development of a hepatic coma with high mortality.[2]

The course

The course of hepatitis is divided into 3 stages.

Prodromal stage

Stage of liver failure

This stage usually lasts 2-8 weeks (if it is longer, it is chronic hepatitis).

Stage of convalescence

We most often encounter functional disorders of various parts of the GIT. For example, anorexia, a feeling of fullness after a meal, constipation or diarrhea, and pressure under the ribs are common. Quite often there can be pain in the area above the liver. This is posthepatic heparalgia (due to adhesions between the capsule and the peritoneum). They usually disappear within a few months.[2]

Laboratory tests

The first biochemical change that we notice in the laboratory examination is an increase in aminotransferases. This change is already visible in the prodromal stage.

In acute hepatitis, ALT rises more than AST. The changes in ALT and AST values help assess the course of the disease and the prognosis.

Persistent levels after jaundice may indicate possible chronicity. Rise after recovery is indicative of relapse. In prolonged and chronic hepatitis, we observe **changes in the serum protein electrophoresis**. A decrease of albumin and an increase of gamma globulins are observed. There are also **viral markers**.[2]

Differential diagnosis

=== Anamnesis===*family history – liver disease, bile duct disease, familial hyperbilirubinemia, hemolytic conditions

- personal history – neonatal jaundice...
- Reasons for accidentally detected high aminotransferases in a healthy child:
  - **laboratory mistake** – old or hemolyzed blood (new sample is necessary)
  - **incipient acute hepatitis**
  - **liver damage from another infection**
  - **drug damage to the liver**
  - **diseases of the bile ducts or the pancreas** – ALP, GMT, amylase
  - **diseases of other organs with AST or ALT elevation – myocardium, muscles**
  - **hepatic steatosis** – diabetes, obesity, disorders of lipid metabolism, thyropathy.[2]

Acute hepatitis therapy

Hospitalization in the infectious ward – isolation, for the purpose of a quick and comprehensive diagnosis and for the observance of the rest regime. Antiviral therapy is not performed and treatment is mainly supportive: rest and dietary changes (an exception is HCV, where early administration of interferon α reduces the likelihood of transition to chronicity).

Diet

Physical activity is not recommended in the acute phase, it is harmful. When it comes to diet, excessive protein intake or fat reduction is unnecessary. Substances formed from the burning of fats and alcohol are harmful, which is why we avoid them. We make sure that the patient does not take hepatotoxic drugs. **Hepatoprotective drugs** (Silymarin – Flavobion) do not significantly affect the course.

The patient is discharged when the problems disappear (jaundice) and after the enzymes fall within the physiologic range. We monitor the patient even after discharging them. The first check-up is 2-3 weeks after. Palpation and liver tests are appropriate during the first check-up. **The condition for returning to school** is the normalization of aminotransferases and the regression of jaundice.[2]

After curing Hepatitis A, we observe the disappearance of IgM antiVHA.

After curing Hepatitis B, we observe the disappearance of HBsAg.

In Hepatitis C it is useful to ensure that the viral DNA disappeared from the serum by PCR.

References

Related articles

- Viral hepatitis
- Viral hepatitis / case study
- Chronic non-infectious liver diseases
- Liver cirrhosis • Portal hypertension • Liver failure (pediatrics) • Liver failure • Liver function tests

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