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Enteral Viral Hepatitis

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Abstract: The article presents the clinical and epidemiological features of enteral (A, E) hepatitis at the present stage, to master the criteria for early clinical and laboratory diagnosis of acute viral hepatitis, the principles of differential diagnosis in different periods of the disease, treatment methods depending on the severity of acute hepatitis, management tactics and dispensary observation of patients after discharge from the hospital, anti-epidemic measures in the outbreak, principles of specific prevention and their implementation in practice.

Keywords: Enteric viral hepatitis, hepatitis A and E, infectious disease, family Picornaviridae, genus Hepatovirus.

Relevance of the topic: Enteral viral hepatitis is a group of widespread infectious and inflammatory liver diseases, which are based on hepatocellular necrosis and inflammation with a predominance of lymphocytes and macrophages in the inflammatory infiltrate, caused by hepatotropic viruses with a fecal-oral transmission mechanism.

Enteral hepatitis with a fecal-oral transmission mechanism includes hepatitis A and E.

The purpose of the study is to study the prevalence of hepatitis A (HA) and hepatitis E (HE) based on an analysis of the frequency of detection of antibodies to hepatitis A and E viruses in the population and groups at high risk of infection.

Main part.

Hepatitis A (HA) is an acute infectious disease caused by an RNA-containing virus belonging to the *Picornaviridae* family, genus *Hepatovirus*, with a fecal-oral mechanism of infection, which is characterized by an acute onset, short-term symptoms of intoxication, rapidly transient disorders of liver function, cyclic and, as usually benign.

Etiology.

The causative agent - hepatitis A virus (HAV) - belongs to the genus *Hepatovirus*, family *Picornaviridae*, has a diameter of about 28 nm (from 28 to 30 nm). HAV RNA is packaged into a nonenveloped icosahedral nucleocapsid formed by the structural proteins VP1, VP2, VP3, VP4 (Figure 1). The HAV genome is represented by single-stranded RNA of positive polarity, about 7500 nucleotides in length, having one open reading frame encoding structural and non-structural proteins.

RNA – 7500 nucleotide bases

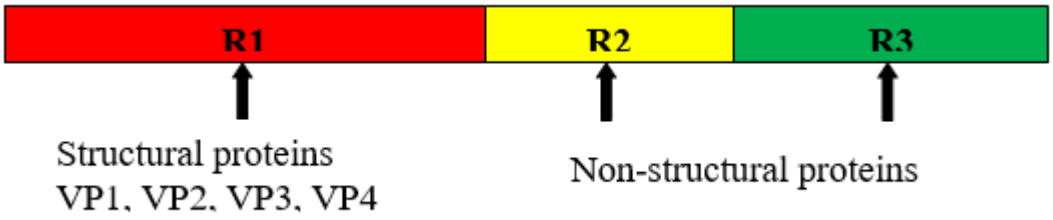

Figure 1. Structure of the hepatitis A virus genome.

The presence of at least six different HAV genotypes (I–VI) has been established, with genotypes I, II, and III occurring in humans. HAV is detected in blood serum, bile, feces and cytoplasm of hepatocytes in infected individuals at the end of incubation, the prodromal period and the initial phase of the height of the disease, and extremely rarely - at a later date. HAV is stable in the external environment: at room temperature it can persist for several weeks or months, and at 4ºC for several months or years. HAV is inactivated by boiling for 5 minutes, is sensitive to formaldehyde and ultraviolet radiation, and is relatively resistant to chlorine.

Epidemiology.

The source of infection is most often patients with atypical forms (inapparent, subclinical, anicteric and erased) or patients in the incubation, prodromal periods and the initial phase of the height of the disease, in whose feces HAV is detected.

The leading mechanism of HA infection is fecal-oral, carried out by water, food or contact-household transmission.

Susceptibility to HA is universal. The disease is most often registered in children over 1 year of age (especially aged 3–12 years and in organized groups) and in young people (20–29 years old). Children under 1 year of age are less susceptible to infection due to their preservation of passive immunity transmitted from the mother. People over 30–35 years of age develop active immunity, confirmed by the detection of antibodies to the virus (anti-HAV IgG) in the blood serum of 60–97% of donors.

HA is characterized by seasonality - an increase in incidence in the summer-autumn period. Along with the seasonal one, there is also a cyclical increase in incidence after 10–12 years, which is associated with a change in the immune structure of the population of the hosts of the virus.

Pathogenesis.

Hepatitis A is an acute cyclic infection characterized by a clear change in periods. After infection, HAV penetrates from the intestine into the blood and further into the liver, where, after fixing to hepatocyte receptors, it penetrates intracellularly. At the stage of primary replication, no clear damage to hepatocytes is detected. New generations of viruses are released into the bile canaliculi, then enter the intestines and are excreted in feces into the external environment. Part of the viral mass penetrates into the blood, causing the development of intoxication symptoms of the prodromal period. Damage to hepatocytes that occurs during the further course of HA is not caused by viral replication, but by immune-mediated cytolysis. During the height of HA, a morphological study allows us to identify inflammatory and necrobiotic processes occurring mainly in the periportal zone of the hepatic lobules and portal tracts. These processes underlie the development of the main clinical and biochemical syndromes: disorders of pigment metabolism (bilirubin metabolism), cytolytic, mesenchymal-inflammatory and cholestatic.

In viral hepatitis, disturbances in pigment metabolism develop primarily at the stage of excretion of conjugated (bound) bilirubin by hepatocytes. The main cause of impaired bilirubin excretion should be considered damage to enzyme systems and a decrease in the energy potential of

hepatocytes. The conjugated bilirubin formed in hepatocytes ultimately does not enter the bile capillary, but directly into the blood as a result of increased membrane permeability.

Laboratory signs of cytolytic syndrome include increased activity of the enzymes ALT and AST (alanine amino- and aspartate aminotransferase). The initial stage of cytolytic syndrome is an increase in the permeability of the hepatocyte membrane. This causes the release into the blood, first of all, of ALT, an enzyme located in the cytoplasm of the liver cell (AST is contained in the cytoplasm and mitochondria; its predominance over ALT is observed in a significant necrotic process - malignant hepatitis, decompensated cirrhosis of the liver). Increased ALT activity is an early and reliable indicator of hepatocyte damage. However, it should be noted that cytolytic syndrome develops in response to any damaging influence (viral toxins, microbes, hypoxia, medications, poisons, etc.), therefore, increased ALT activity is characteristic not only of viral hepatitis.

Mesenchymal inflammatory syndrome is characterized by increased levels of alpha and gamma globulins of all classes. Cholestatic syndrome is manifested by an increase in the blood level of conjugated bilirubin, bile acids, cholesterol, copper, the activity of alkaline phosphatase, gammaglutamyl transpeptidase, as well as bilirubinuria, a decrease (disappearance) of urobilin bodies in the urine.

Thanks to the action of complex immune mechanisms (increased interferon production, activation of natural killer cells, antibody production and activity of antibody-dependent killer cells), the replication of the virus stops and it is eliminated from the human body. HA is not characterized by either the prolonged presence of the virus in the body or the development of a chronic form of the disease. However, sometimes the course of the disease can be modified in the case of coinfection or superinfection with other hepatotropic viruses. Individuals with a genetic predisposition may develop autoimmune hepatitis type 1.

Forms of hepatitis A.

1. According to the severity of clinical manifestations:

- typical (manifest, icteric);
- atypical (inapparent, subclinical, anicteric, erased).

2. By duration:

- spicy;
- protracted.

3. According to the severity of the flow, the typical form is divided into:

- light;
- medium-heavy;
- heavy.

4. By type of complications:

- relapses;
- exacerbations;
- damage to the biliary tract.

5. By outcome:

- recovery without residual effects;
- with residual effects - post-hepatitis syndrome, prolonged convalescence, damage to the biliary tract (dyskinesia, cholecystitis).

In manifest cases of the disease, the following are distinguished: incubation, pre-icteric (prodromal), icteric periods and the period of convalescence.

Clinical picture.

The incubation period of HA is on average 35 days (from 7 to 50 days). The prodromal (pre-icteric) period, lasting on average 5–7 days, is characterized by an acute onset with an increase in body temperature to 38–40ºC within 1–3 days; patients complain of headache, loss of appetite, bitter taste in the mouth and halitosis, nausea, a feeling of heaviness in the epigastric region, pain in the right hypochondrium. Characterized by irritability, increased nervousness, moodiness, loss of interest in games, and sleep disturbances. Repeated vomiting may occur, less often - multiple vomiting. Flatulence, constipation, and, less commonly, stool liquefaction occur. During this period, the liver enlarges, is of moderate density, painful on palpation; sometimes (in 10–20% of patients) the spleen enlarges. By the end of the pre-icteric period, there is a change in the color of urine, acquiring the color of dark beer or strong tea and, less often, discoloration of feces. Biochemical studies reveal an increase in the activity of ALT and AST from the first days of the disease. From the onset of the disease, the amount of urobilin in the urine increases, and at the end of the pre-icteric period bile pigments are detected.

The peak period lasts on average 2–3 weeks. The transition to this period is accompanied by a clear improvement in general condition and a decrease in complaints, which serves as an important differential diagnostic sign of HA. As a rule, the occurrence of jaundice is accompanied by stool acholia, a decrease in body temperature to normal or subfebrile levels, a decrease in headaches and other general toxic manifestations.

Hepatitis E (HE) is an anthropozoonotic viral disease with a fecal-oral mechanism of infection, prone to epidemic spread, occurring predominantly in benign cyclic forms, but with a high frequency of adverse outcomes in pregnant women.

Etiology.

The causative agent - hepatitis E virus (HEV) - belongs to the family *Hepeviridae*, genus *Orthohepevirus*. Humans are affected by *Orthohepevirus A*. Virus particles are round formations with a diameter of about 32 nm (from 27 to 34 nm) without an outer shell.

The HEV genome is a single-stranded RNA of positive polarity, approximately 7500 nucleotides long. *Orthohepevirus A* is genetically heterogeneous (Table 1).

Genotype	Occurrence	Affected organism
1	Asia, Africa	Human
2	Mexico, Africa	Human
3	Western Europe, South America, North America	Human, pig, deer, goat, rabbit, bottlenose dolphin
4	East Asia	Human, pig, deer, cattle, sheep
5	Japan	A wild boar
6	Japan	A wild boar
7	China	Camel, human
8	China	Camels

Source:https://vk.com/doc197144360_612621958?hash=vIAg2FOz6bHGk1A6M39eDkqU1z6AMbGvyI0oHHOSkN8

Epidemiological data indicate a significantly lower virulence of the pathogen compared to HAV, which explains the need for large doses of HEV for infection.

Epidemiology.

Source of infection.

- a sick person (genotype 1, 2), excreting viruses in feces several days before and 3-4 weeks after the onset of the disease;
- animals (pigs, wild boars, deer; humans; genotype 3, 4);
- camels (genotype 7).

Mechanism of infection.

- fecal-oral;
- vertical transmission from a pregnant woman to her fetus.

Transmission routes.

- water, which is of primary importance, which mainly determines the epidemic spread of infection;
- food, including meat and liver of infected animals;
- contact and household.

Susceptibility to HEV is universal. Adults are predominantly affected, especially those aged 15–29 years, who represent the most active part of the working population. HE is most dangerous for pregnant women, due to the risk of death.

HE is endemic in areas with a hot climate and extremely poor water supply to the population. The disease is widespread in Central Asia, Africa and Latin America. Endemic areas are known in Turkmenistan, Uzbekistan, Kyrgyzstan, and Tajikistan. There is a seasonality of incidence: the increase is due to the beginning or end of the rainy season in Southeast Asia, and in the countries of Central Asia the peak of infection occurs in the fall. An increase in incidence in endemic regions is recorded every 7–8 years.

The disease can occur in the form of exceptionally powerful water epidemics, covering many tens of thousands of people over a relatively short period of time (the explosive nature of the epidemic). The first suggestion of the existence of viral hepatitis with a fecal-oral transmission mechanism, etiologically different from HA, arose from a retrospective investigation of a large waterborne outbreak of hepatitis in India in 1955–1956. The outbreak affected 35 thousand residents. A distinctive feature of HE outbreaks is selective and high mortality among pregnant women in the second half of pregnancy.

After an illness, intense type-specific immunity appears to be formed. Repeated cases of HE disease have been described, which may be due to the antigenic heterogeneity of HEV.

Pathogenesis and pathological picture.

This issue has not been sufficiently studied. It is believed that liver damage is immune-mediated, but there is evidence of the cytopathogenicity of HEV, which determines the severity of necrobiotic changes in the liver tissue.

Clinical picture.

The incubation period lasts an average of 40 days (from 15 to 60 days). The disease begins gradually. The prodromal period lasts from 1 to 10 days with the development of asthenovegetative and dyspeptic symptoms, less often in the form of a short fever. During the peak period with the appearance of jaundice, the patients' well-being usually does not improve, in contrast to that with HA. Yellowness of the skin, as a rule, increases in the first 2–3 days, sometimes within 10 days and persists for 1–3 weeks. After 2–4 weeks, a reversal of symptoms and recovery is observed. HE is not characterized by a chronic course and viral carriage.

With HE, moderate and severe forms of the disease are recorded more often than with HA.

Verification of the diagnosis of HE is carried out on the basis of clinical and epidemiological data and detection in blood serum of:

- Anti-HEV IgM и Anti-HEV IgG;
- РНК HEV методом ПЦР.

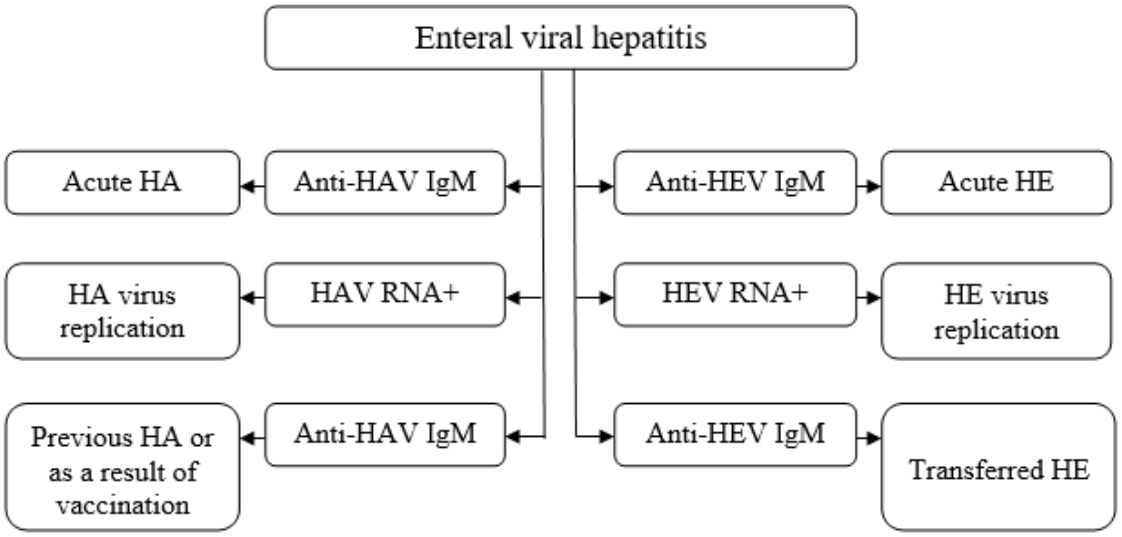

Figure 2. Enteral viral hepatitis.

Conclusion:

The treatment of patients with enteric viral hepatitis is based on pathogenetic and syndromic therapy (includes basic - drug-free and nonspecific drug therapy).

Mild and moderate forms of HA and HE do not require extensive drug therapy. The basis of their treatment is basic therapy, which includes a therapeutic and protective regimen (in the acute period - bed; subsequently - half-bed and general) and diet therapy. Plenty of fluids are administered orally, a 5% glucose solution is allowed (taking into account anamnestic data on the risk of diabetes mellitus) and vitamin therapy (C, P, B1, B2, B6, PP, in the period of late convalescence - tocopherol).

The diet for acute HA and HE should be complete, high-calorie, easily digestible and, if possible, physiological (according to the type of liver table No. 5, for children according to Pevzner). The ratio of proteins, fats and carbohydrates is 1:1:4.5. Proteins are introduced into the diet in the form of cottage cheese, children's forms of kefir (preferably with bifidobacteria), lean meats (beef, veal), and lean fish. Fats in the acute period are given in limited quantities with an increase in the period of convalescence in the form of butter and vegetable oil (olive, sunflower). Carbohydrates - in the form of oatmeal and buckwheat porridge, bread, pasta, boiled vegetables (cabbage, potatoes, etc.). Meals should be fractional.

The daily diet of a patient with enteric viral hepatitis must include a sufficient amount of boiled or stewed vegetables (cabbage, zucchini), fruits (pears, bananas, apples), and juices. Honey, jam, pastille or boiled apples, watermelon, raisins, dried apricots, prunes, fruit or oatmeal jelly are allowed.

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