TYPES, PATHOGENESIS, DIAGNOSIS, AND TREATMENT METHODS OF VIRAL HEPATITIS

Nuraliyeva Madina Ergash qizi
sayoraabduraxmonova756@gmail.com
Toshkent tibbiyot akademiyasi Termiz filiali talabasi
Abdukarimov Ahmadali Sherali o‘g‘li
aliabdukarimov417@gmail.com
Toshkent tibbiyot akademiyasi Termiz filiali talabasi
Egammurodov Otabek Abduhakimovich
otabekegammurodov4@gmail.com
Toshkent tibbiyot akademiyasi Termiz filiali talabasi
Nafisa Yaranova O‘razali qizi
nafisayaranova@gmail.com
Toshkent tibbiyot akademiyasi Termiz filiali talabasi
Almardanova Kamola Tulkinovna
almardanovakomo@icloud.com
Toshkent tibbiyot akademiyasi Termiz filiali talabasi

Annotation:
Viral hepatitis is an infectious disease of viral origin characterized by general intoxication, jaundice (or non-jaundiced forms), liver damage, and metabolic disorders. The term “viral hepatitis” includes all types of viral hepatitis—A, B, C, D, and E. Viral hepatitis is an acute infectious disease that manifests with liver cell damage, general intoxication symptoms, yellowing of the skin and mucous membranes, dark-colored urine (resembling “beer”), clay-colored stools, and enlargement of the liver, sometimes accompanied by spleen enlargement.
Keywords:Viral hepatitis, general intoxication, vibrion, chemical and physical factors.

The term “viral hepatitis” unites several independent nosological forms, including hepatitis A, B, C, D, and E. Today, “non-A, non-E” viral hepatitis is also distinguished. The hepatitis A virus has a diameter of 27–32 nm, contains RNA, and lacks an envelope. It is resistant to acids and alkalis but loses activity under the influence of ether and chloroform. The virus dies after 5 minutes of boiling and can survive for a week in a dry environment at room temperature, 3–10 months in liquid environments (especially water), and up to 30 days in excreta. These properties allow the virus to

persist for long periods in water, food, stagnant water, and other external environmental objects.

The hepatitis B virus contains DNA and has a total diameter of 42–45 nm. It possesses three antigens:

1. HBsAg
2. HBcAg
3. HBeAg

The hepatitis B virus is highly resistant to low and high temperatures, as well as to chemical and physical factors. It remains viable for 3 months at room temperature, 6 months in a refrigerator, 15–20 years in a frozen state, and up to 25 years in dried plasma. The virus loses activity if boiled for more than 30 minutes. It remains active for 2 hours in a 1–2% chloramine solution and for 7 days in a 1.5% formalin solution. The virus is resistant to ether and ultraviolet radiation. In an autoclave at 120°C, the virus becomes inactive after 5 minutes, and in dry heat at 160°C, it loses viability only after 2 hours.

The hepatitis C virus is a small, spherical, enveloped virus with a diameter of 50 nm containing RNA. More than 30 genotypes of the virus have been identified.

The hepatitis D virus lacks both an outer and inner envelope and contains RNA. A key feature of this virus is its dependence on a helper virus. The hepatitis B virus acts as a helper virus, allowing the hepatitis D virus to attach to its outer envelope.

The hepatitis E virus has a diameter of 27–37 nm (most commonly 32–37 nm). It is an RNA virus without an envelope. It is less resistant to external environmental factors and rapidly degrades under laboratory conditions. In recent years, reports have emerged about “non-A, non-E” hepatitis viruses, later referred to as hepatitis F, but reliable information about this virus remains limited.

Pathogenesis of Viral Hepatitis

The pathogenesis of viral hepatitis is complex. When hepatitis B, D, or C viruses enter the body, they first cause primary viremia. Then, the viruses penetrate hepatocytes (liver cells). Hepatitis A and E viruses enter the human body through the mucous

membranes of the gastrointestinal tract, settle in the small intestine, and then spread to regional lymph nodes, where they multiply. From the lymph nodes, the viruses enter the bloodstream and spread throughout the body. However, the liver provides a favorable environment for their development. The viruses reach hepatocytes, partially enter bile, and are excreted from the body through feces.

In some cases, hepatocyte destruction (cytolysis) occurs due to the direct effect of viruses developing inside liver cells (hepatitis C, A, E), while in other cases (hepatitis B, D), it results from an immune response against infected hepatocytes. At the end of the incubation period, the permeability of cell membranes increases under the influence of viruses and immune complexes, leading to elevated levels of serum enzymes (aldolases and aminotransferases—ALT, AST). Prolonged presence of viruses in liver cells damages hepatocyte membranes and accelerates cell death (normally, hepatocytes have an average lifespan of about 150 days). During the acute phase of the disease, immune reactions against infected hepatocytes lead to their destruction (cytolysis) and bile stasis (cholestasis), ultimately causing liver disease. At this stage, symptoms of general intoxication appear. As the disease progresses, carbohydrate, protein, water-salt, and vitamin metabolism in the liver are disrupted. A large amount of bile pigments and bile acids enter the bloodstream, resulting in yellowing of the skin and mucous membranes, as well as itching. In severe cases, coagulation disorders develop.

Diagnosis

In typical cases, diagnosing the disease is not difficult. The acute onset of illness, liver changes, dark-colored urine, discolored stools, and the subsequent appearance of jaundice confirm the diagnosis. Laboratory tests play a key role in diagnosis. A general blood test reveals leukopenia (a decrease in white blood cells), while the erythrocyte sedimentation rate (ESR) is usually normal or sometimes decreased. Urinalysis shows toxic changes, including the presence of protein, casts, and leukocytes.

Treatment

In the treatment of viral hepatitis, rest and diet are crucial. From the moment the disease is diagnosed until the clinical symptoms disappear and liver function normalizes, bed rest is recommended. The diet should meet the patient’s daily nutritional needs and be enriched with sufficient proteins. Patients are prescribed a dairy-plant-based diet with

limited fats. Meals should be semi-liquid and consumed 4–5 times a day. The daily caloric intake should be around 3,000 kcal. The diet must be rich in potassium salts, iron, and vitamins. Spicy foods, fried eggs and fish, sauerkraut, pickles, and canned foods are prohibited. Kefir, yogurt, and fresh cottage cheese are beneficial. Up to 400 g of cottage cheese per day is recommended. Patients must consume enough carbohydrates, including sweets, sugar, jam, honey, and fruit juices. Sweet fruits and vegetables, as well as berry juices, are beneficial. Patients should eat boiled meat and fresh boiled fish. Drinking plenty of fluids is essential. Diet No. 5 is prescribed.

References:

1. Uchqun o'g'li, B. S. (2021). BLOOD DISEASES IN SURKHANDARYA REGION, THEIR CAUSES AND PREVENTION. International Engineering Journal For Research & Development, 6(ISPCIEI), 2-2.
2. Abdullayevich, B. E., & Uchqun o'g'li, B. S. (2021). TRANSITIONAL FEATURES OF ACUTE HERPETIC STOMATITIS IN CHILDREN AND MODERN APPROACHES TO TREATMENT. World Bulletin of Public Health, 1(1), 1-3.
3. Uchqun o'g'li, B. S. Bulvar And Psevdobulbar Syndrome. International Journal on Integrated Education, 3(11), 19-22.
4. Ugli, B. S. U., & Bekchanovich, K. Y. (2020). CLASSIFICATION OF VITAMINS AND DISEASE SYNDROME. Science and Education, 1(1).
5. Madiyev Rustam Zoirovich et al. (2021). EFFECTIVENESS OF ENDOVIDEOLAPAROSCOPIC TECHNOLOGY IN THE TREATMENT AND EARLY PREVENTION OF INTESTINAL OBSTRUCTION IN CHILDREN. European Journal of Research Development and Sustainability, 2(7), 68-73.
6. Sultonov, R. K., Sodiqova, Z. S., & o'g'li, B. S. U. (2021). Dynamics of Fat Cells of the Bronchial Tree Mucosa in Postnatal Ontogenesis. CENTRAL ASIAN JOURNAL OF MEDICAL AND NATURAL SCIENCES, 2(4), 182-184.