

# **Peculiarities of Viral Hepatitis in Young Children**

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**Abstract:** This analytical article examines the pathogenic mechanisms, immunological characteristics, age-related differences in clinical manifestations, as well as preventive and therapeutic approaches of viral hepatitis in young children. The immaturity of the immune system in children can lead to the chronicity of the disease. The mechanisms of action, morphological changes, and diagnostic approaches of hepatitis A, B, and C viruses are discussed at an academic level.

**Keywords:** Viral hepatitis, immune response, hepatocyte, hepatitis A, hepatitis B, hepatitis C, pathogenesis, child.

#### **Main Part**

Viral hepatitis ranks among the most common liver diseases in children. Their etiology is mainly associated with hepatotropic viruses (HAV, HBV, HCV). The course of these infections in children is closely related to the immaturity of the immune system, metabolic changes, and the regenerative capacity of hepatocytes.

# **Pathogenesis and Immune Mechanisms**

The virus enters the body via the enteral (A, E) or parenteral (B, C, D) route and replicates within the main liver cells — hepatocytes. The virus itself does not exert a direct cytolytic effect; instead, liver cell damage occurs due to immune cells (cytotoxic T-lymphocytes).

In young children, due to the immaturity of the immune system, both innate and adaptive components do not function fully. Low levels of NK cells and interferons hinder complete viral elimination, predisposing to chronic infection.

#### **Cytokines and Inflammatory Response**

Mediators such as interleukin-6 (IL-6), interferon-gamma (IFN- $\gamma$ ), and tumor necrosis factor (TNF- $\alpha$ ) activate inflammatory processes in viral hepatitis. In children, lower cytokine levels lead to milder clinical symptoms; however, the virus may persist in the body longer.

## **Morphological Changes**

According to biopsy data, hepatocytes in children exhibit vacuolar and granular dystrophy, lymphomononuclear infiltrations in portal tracts, and occasionally submassive necrosis. Chronic forms may involve fibrosis and bridging necrosis. Although regeneration proceeds rapidly, continuous replication intensifies fibrosis.

#### **Clinical Course**

In newborns, hepatitis often manifests asymptomatically or with mild jaundice, but HBV infection becomes chronic in about 90% of cases. In children aged 1–5 years, hepatitis A is usually moderate, while hepatitis C remains latent. In children older than five, the clinical picture resembles that of adults, and acute cases often resolve completely.

#### **Diagnosis**

Diagnosis is established using biochemical tests (ALT, AST, bilirubin), serological markers (HBsAg, Anti-HCV, Anti-HAV IgM), and molecular methods (PCR). Ultrasound may reveal hepatomegaly and a hypoechoic parenchymal structure.

#### **Treatment and Prevention**

Symptomatic therapy is used for hepatitis A, while hepatitis B and C are treated with interferon-α, pegylated interferons, and nucleoside analogs (entecavir, tenofovir). In chronic cases, immunomodulators such as thymogen and lycopid are recommended. Preventive measures include HBV vaccination and maintaining proper hygiene.

## **Significance of the Topic**

Studying viral hepatitis in young children holds great significance for the healthcare system, as early diagnosis and vaccination can prevent chronic liver diseases. This not only protects child health but also reduces the risk of severe complications such as hepatocellular carcinoma in the future.

#### Conclusion

The course of viral hepatitis in young children depends on the immune system, virus type, and mode of infection. Immune tolerance, cytokine imbalance, and hepatocyte immaturity contribute to chronic progression. Therefore, improving prevention, vaccination, and early detection strategies remains crucial.

#### References

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