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CLINICAL AND LABORATORY CHANGES IN HBV/HDV CO-INFECTION: PATHOPHYSIOLOGICAL AND CLINICAL INSIGHTS

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Abstract
Chronic hepatitis is a prolonged inflammatory liver disease, primarily caused by viral infections (HBV, HCV), autoimmune reactions, or toxic exposures. The chronic inflammatory process may result in hepatocyte injury, fibrosis, and cirrhosis, which determine the severity of the disease and the risk of complications.

Introduction

Co-infection with hepatitis B virus (HBV) and hepatitis D virus (HDV) represents a significant challenge in hepatology due to its aggressive clinical course and rapid progression to liver fibrosis and cirrhosis. HDV is a defective RNA virus that depends on HBV for replication, and its presence intensifies the immune-mediated hepatocyte injury already initiated by HBV [1,2]. Patients with HBV/HDV co-infection often present with nonspecific symptoms such as fatigue, malaise, anorexia, and right upper quadrant discomfort, which may progress to jaundice in acute exacerbations. Laboratory investigations provide critical insights into the extent of liver injury, immune activation, and disease progression in HBV/HDV co-infection. Elevation of alanine aminotransferase (ALT) and aspartate aminotransferase (AST) reflects ongoing hepatocyte necrosis, and these enzymes often show fluctuating patterns corresponding to intermittent immune-mediated attacks against infected hepatocytes [3]. Concurrently, bilirubin levels, both total and direct, may increase during flares, indicating impaired bilirubin metabolism and cholestatic features, which often correlate with clinical jaundice. Serological markers are central to both diagnosis and prognosis. Patients typically remain HBsAg positive, reflecting chronic HBV infection, while anti-HDV antibodies (IgM and IgG) and detectable HDV RNA confirm active HDV replication [4,5]. The presence of HDV RNA is particularly associated with more severe liver inflammation, accelerated fibrosis, and higher risk of cirrhosis compared to HBV monoinfection. Co-infection also impacts hematological parameters. Mild thrombocytopenia is frequently observed due to hypersplenism in advanced fibrosis, while leukopenia may occur as a consequence of chronic inflammation or immune-mediated suppression. Anemia, often mild, may reflect chronic disease or nutritional deficiencies secondary to liver dysfunction [6]. Liver synthetic function is commonly impaired in HBV/HDV co-infection. Prolonged prothrombin time (PT) and elevated INR indicate reduced hepatic production of coagulation factors, and hypoalbuminemia demonstrates diminished protein synthesis. These changes not only serve as markers of hepatic reserve but also correlate with disease severity and predict the risk of hepatic decompensation [6,7,9].

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The pathogenesis of HBV/HDV co-infection involves complex immune-mediated mechanisms. Viral infection triggers cytotoxic T lymphocyte responses and inflammatory cytokine release, leading to hepatocyte apoptosis, necrosis, and regenerative proliferation. Pro-inflammatory cytokines such as TNF-α and IL-6 amplify hepatocellular injury, while anti-inflammatory cytokines like IL-10 may modulate the immune response but potentially permit viral persistence [1,3,5]. TGF-β activation contributes to fibrogenesis, stimulating fibroblasts and extracellular matrix deposition, which manifests clinically as increased liver stiffness, detectable through imaging or elastography [7]. IL-8 and IL-17A further drive neutrophil recruitment and inflammation, linking laboratory abnormalities to histopathological damage [8].

The laboratory markers and clinical manifestations are therefore interconnected. Fluctuating transaminase levels parallel immune-mediated hepatocyte damage, bilirubin elevations correspond with cholestasis and hepatocellular dysfunction, and coagulation abnormalities reflect the decline in hepatic synthetic capacity. Hematologic changes such as thrombocytopenia indicate portal hypertension and splenomegaly secondary to progressive fibrosis. Collectively, these clinical and laboratory findings highlight the aggressive nature of HBV/HDV co-infection and underscore the need for early identification and close monitoring [2,6].

Recognizing the pattern of laboratory changes in HBV/HDV co-infection allows clinicians to assess disease activity, predict progression to cirrhosis, and identify candidates for antiviral therapy or liver-protective interventions. Monitoring ALT, AST, bilirubin, coagulation profile, and viral markers should be performed regularly. Integration of serological and biochemical data with clinical evaluation enables a more precise prognostic assessment and personalized management plan [3,5,7].

HBV/HDV co-infection induces a synergistic effect on liver injury, reflected in interconnected clinical and laboratory abnormalities. Elevations in ALT, AST, and bilirubin, coupled with changes in coagulation parameters and serological markers, provide critical information about the severity of hepatocellular damage and fibrogenesis. Understanding these interrelated parameters is essential for timely diagnosis, prognostic evaluation, and implementation of targeted therapeutic strategies to prevent progression to cirrhosis and hepatocellular carcinoma.

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