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THE IMPORTANCE OF CYTOKINES IN LIVER DISEASES

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It is well known that liver cirrhosis is characterized by a pronounced fibrotic process, disruption of organ architecture, and the formation of regenerative nodules, leading to necrotizing inflammation. As a result, the internal veins of the liver and the portal vein become compressed, resulting in portal hypertension. In recent years, there has been an increase in research dedicated to the role of cytokines in the pathogenesis of liver cirrhosis and the development of disease complications. Under physiological conditions, cytokines regulate Ito cells, which are responsible for the main functions in organ fibrogenesis, specifically liver fibroblasts. In addition to producing profibrotic factors, Ito cells also produce antifibrotic factors, maintaining this balance. Antifibrotic factors include metalloproteinases such as collagenase, gelatinase, and stromelysin.

Recently, the role of cytokines in complications of liver cirrhosis, such as portal hypertension, hepatic encephalopathy, bleeding from varicose veins, and multiple organ failure, has been studied. Currently, researchers are extensively investigating the importance of the cytokine system in chronic liver diseases and its relationship with other regulatory systems in the body. It is known that the activity of the immune system is controlled by genetic factors, suggesting that the chronic nature of viral infections or the progression of chronic hepatitis may also be linked to immunogenetic mechanisms. Therefore, some individuals are resistant to viral infections, while others are more prone to disease.

Today, it has been proven that HCV and HBV viruses do not exert direct hepatotoxic effects but are associated with liver tissue damage resulting from immune inflammation and the progression of liver fibrosis. The increase in pro-inflammatory cytokines that enhance inflammation in patients with liver cirrhosis is attributed to the effects of endotoxins entering the systemic circulation due to increased intestinal permeability of gram-negative bacteria. Under normal physiological conditions, the endotoxins from gram-negative bacteria in the intestine are neutralized by Kupffer cells after entering the systemic circulation. Endotoxins lead to lipid peroxidation, an increase in free radicals, and an increase in TNF-α levels, resulting in enhanced apoptosis due to necrosis and cellular inflammatory infiltration. The increase in TNF-α levels causes an increase in interleukins such as IL-1, IL-6, and IL-8, which are responsible for hepatocyte necrosis, apoptosis, and fibrogenesis.

Recent studies have shown that the increase in pro-inflammatory cytokines such as TNF-α, IL-1, and IL-6 in serum is accompanied by a decrease in anti-inflammatory cytokines such as IL-4 and IL-10, leading to complications such as portal hypertension, hepatic encephalopathy, ascites, and ascitic peritonitis. Thus, in liver cirrhosis, there is a disruption in the balance between pro-inflammatory and anti-inflammatory cytokines. Pro-inflammatory cytokines play a crucial role in the pathogenesis of liver cirrhosis decompensation and the development of complications such as portal hypertension, ascites, hepatic encephalopathy, and liver failure.

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