

CYTOKINES IN CHRONIC HEPATITIS: PATHOGENETIC MECHANISMS AND CLINICAL SIGNIFICANCE

Tolibjonova Malika Khusanovna,
Oripov Nuriddin Najmiddinovich
Tashkent State Medical University, Uzbekistan

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

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 [1,2]. Cytokines—signaling molecules produced by immune cells—play a central role in the pathogenetic mechanisms of chronic hepatitis. They regulate viral persistence, coordinate inflammatory responses, activate immune cells, and contribute to fibrosis development [1,3]. IL-6 is a pro-inflammatory cytokine predominantly produced by macrophages and T lymphocytes. It supports lymphocyte activity, enhances inflammation, and stimulates fibroblasts to promote collagen synthesis. Elevated IL-6 levels in chronic hepatitis are associated with prolonged inflammation and liver fibrosis [3]. Clinically, IL-6 serves as a biomarker for predicting disease severity. TNF- α is a pro-inflammatory cytokine mainly secreted by macrophages. It promotes hepatocyte apoptosis, amplifies inflammatory responses, and plays a critical role in fibrosis progression [4]. Several studies have demonstrated a correlation between TNF- α levels and liver fibrosis severity, making it an important biomarker for disease assessment and therapeutic planning. IL-10 is an anti-inflammatory cytokine primarily produced by regulatory T cells. It suppresses inflammation and may influence viral persistence. High IL-10 levels in chronic hepatitis can reduce immune responses and contribute to long-term viral persistence [5]. IFN- γ , produced by Th1 lymphocytes, plays a key role in eliminating viral infections. It activates macrophages and cytotoxic T lymphocytes, inhibiting viral replication. IFN- γ levels are important for evaluating disease severity and immune cell responses [6]. TGF- β , mainly secreted by fibroblasts and macrophages, is involved in liver fibrosis. It stimulates collagen and extracellular matrix synthesis, contributing to fibrosis and cirrhosis development [7]. IL-8 acts as a chemokine, activating neutrophils and enhancing inflammation. IL-17A, produced by Th17 cells, regulates inflammatory processes. These



cytokines are used to predict disease severity and serve as biomarkers for targeted therapeutic strategies [8].

In chronic hepatitis, cytokines such as IL-6, TNF- α , IL-10, IFN- γ , and TGF- β play a central role in pathogenesis. Their levels are key biomarkers for assessing disease severity, inflammatory activity, and fibrosis progression. IL-8 and IL-17A are used to predict tissue injury and inflammatory activity. These findings are crucial for the development of personalized cytokine-targeted therapies and novel research in chronic hepatitis.

References

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