Commentary

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Cytokine Dysregulation in Viral Hepatitis: Pathogenesis and Therapeutic Targets

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Introduction

Viral hepatitis remains a major global health challenge, affecting more than 350 million individuals worldwide and contributing significantly to liver-related morbidity and mortality. Among the hepatitis viruses (A, B, C, D, and E), chronic infections with Hepatitis B Virus (HBV) and Hepatitis C Virus (HCV) are particularly associated with progressive liver fibrosis, cirrhosis, and Hepatocellular Carcinoma (HCC). A central aspect of disease progression is cytokine dysregulation, a hallmark of host immune imbalance during infection. Cytokines act as key signaling molecules that orchestrate antiviral defense, inflammation, and tissue repair. However, in viral hepatitis, persistent antigenic stimulation leads to an aberrant cytokine response that both fails to clear the virus and contributes to hepatic injury. Understanding the mechanisms underlying cytokine dysregulation is essential for developing targeted immunotherapies that restore immune homeostasis and prevent chronic liver damage [1].

Description

The gut microbiota influences immune responses to parasitic infections through multiple mechanisms, including modulation of cytokine production, regulation of mucosal barrier integrity, and induction of immune tolerance. Commensal bacteria such as Lactobacillus and Bifidobacterium can promote Th1 and Th17 responses, which are critical for controlling intracellular parasites like Toxoplasma gondii. Conversely, helminth infections, including Schistosoma and Heligmosomoides polygyrus, often induce a Th1-dominated immune profile characterized by elevated Interleukin (IL)-4, IL-5, and IL-13 production, which may suppress pro-inflammatory responses and promote chronic infection The interplay between viral replication and host immune response determines the outcome of hepatitis infections. During acute infection, cytokines such as Interferon-Gamma (IFN-γ), Tumor Necrosis Factor-Alpha (TNF-α), and interleukin-11 (IL-11) mediate the activation of Cytotoxic T Lymphocytes (CTLs) and Natural Killer (NK) cells to eliminate infected hepatocytes [2].

However, in chronic HBV and HCV infections, continuous antigenic stimulation results in immune exhaustion, characterized by elevated levels of inhibitory cytokines like interleukin-10 (IL-10) and transforming growth factor-beta (TGF-β). These cytokines suppress antiviral effector functions and promote viral persistence. Moreover, pro-inflammatory cytokines such as interleukin-6 (IL-6) and TNF-α drive fibro genic pathways by activating hepatic stellate cells, contributing to liver fibrosis Emerging research has revealed that cytokine networks in viral hepatitis are shaped by both viral and host genetic factors. HBV and HCV proteins can directly modulate cytokine production by interacting with host signaling pathways such as NF-κB and STAT3. This manipulation not only helps the virus evade immune responses but also promotes oncogenic transformation through chronic inflammation [3].

Single-cell transcriptomic analyses have demonstrated distinct cytokine profiles within liver-resident immune cells, providing insights into the spatial regulation of immune responses during infection. Notably, IL-17–producing Th17 cells and interferon-stimulated genes (ISGs) play dual roles, contributing to both antiviral defense and tissue pathology depending on their regulation. Therapeutically, cytokine modulation has emerged as a promising approach for viral hepatitis management [4,5].

Conclusion

Cytokine dysregulation plays a pivotal role in the immunopathogenesis of viral hepatitis, influencing both disease progression and treatment outcomes. While certain cytokines are crucial for viral clearance, their chronic overproduction leads to hepatocellular damage and fibrosis. A comprehensive understanding of cytokine signaling networks and their modulation by viral factors offers new therapeutic avenues to balance immunity and limit tissue injury. Future research integrating systems immunology, transcriptomics, and precision medicine will facilitate the development of personalized cytokine-targeted therapies, transforming the management of chronic hepatitis and its complications.

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Acknowledgement

None

Conflict of Interest

None

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