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METABOLIC DYSFUNCTION AND ITS IMPACT ON DISEASE COURSE AND FIBROGENESIS IN CHRONIC HEPATITIS B: A REVIEW

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Background and Aims: Metabolic dysfunction-associated steatotic liver disease (MASLD) coexists with chronic hepatitis B (CHB) in 14–70% of patients across cohorts, yet its impact on disease progression remains controversial [1]. We aimed to summarize current evidence on the pathogenetic mechanisms and clinical outcomes of concurrent CHB and metabolic dysfunction, with emphasis on the gut–liver axis in liver fibrogenesis.

Methods: A literature review of PubMed- and Google Scholar-indexed studies (2011–2026) reporting quantitative risk estimates and mechanistic data in patients with CHB and comorbid MASLD was performed.

Results: Hepatic steatosis was found to suppress HBsAg secretion and HBV replication via endoplasmic reticulum stress and TLR4-mediated pathways [2,3]. In a large cohort (n = 8,052), comorbid MASLD was independently associated with a 39% lower risk of severe liver disease (aHR 0.61) and reduced risk of both cirrhosis (aHR 0.61) and hepatocellular carcinoma (aHR 0.57), most pronounced among HBsAg-positive patients with low viral load [4]. Conversely, metabolic dysfunction was shown to promote fibrogenesis through chronic inflammation, oxidative stress, and gut dysbiosis: patients with HBV-related cirrhosis exhibited depletion of short-chain fatty acid–producing genera (Alistipes, Lachnospira) and enrichment of potentially pathogenic taxa (Klebsiella, Streptococcus), correlating with Child-Turcotte-Pugh class [5]. MASLD was additionally associated with increased cardiovascular risk, particularly coronary artery disease (aHR 1.91) [4].

Conclusion: The relationship between metabolic dysfunction and CHB progression is stage-specific and context-dependent, mediated by virologic status and gut microbiota composition. The influence of metabolic dysfunction on fibrogenesis and gut microbiota in HBV/HDV coinfection remains virtually unexplored and represents a promising direction for the authors' ongoing research.

Keywords: chronic hepatitis B; metabolic dysfunction-associated steatotic liver disease; fibrogenesis; gut microbiota; gut–liver axis.

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