

A Comprehensive Analysis of Cirrhosis Microbiota: A Shift toward a More Pathogenic Propensity

Collins Meroni*

Department of Gastroenterology and Hepatology, Athens Medical, P. Faliron Hospital, 175 62 Athens, Greece

Introduction

Cirrhosis, a chronic liver disease characterized by progressive liver fibrosis, represents a major global health concern due to its association with substantial morbidity and mortality. As cirrhosis advances, it leads to irreversible damage to the liver tissue, resulting in the disruption of its normal structure and function. In recent years, there has been growing interest in the role of microbiota, particularly the gut microbiota, in the pathogenesis of cirrhosis and its complications. Traditionally, cirrhosis was thought to primarily result from a combination of genetic, lifestyle and environmental factors, with the liver playing the central role in disease progression. However, emerging evidence suggests that the gut microbiota, the complex community of microorganisms residing in the intestines, plays a significant role in cirrhosis development, progression and the onset of complications. This review aims to provide a comprehensive analysis of the microbiota in cirrhosis, with a focus on the shift toward a more pathogenic microbial profile and its implications for disease progression [1,2].

Description

The human microbiota consists of trillions of microorganisms, including bacteria, viruses, fungi and archaea, which reside in various parts of the body, including the gut, skin, mouth and respiratory tract. The gut microbiota, in particular, is composed predominantly of bacteria and plays a critical role in maintaining homeostasis by regulating immune function, nutrient absorption and the synthesis of certain vitamins and metabolites. Dysbiosis, or the imbalance in the composition of the microbiota, has been implicated in a variety of diseases, including gastrointestinal disorders, metabolic diseases and cardiovascular conditions. In the context of liver diseases such as cirrhosis, dysbiosis has been identified as a key factor contributing to the development of liver inflammation, fibrosis and other complications.

The shift toward a more pathogenic microbiota in cirrhosis has been associated with several clinical manifestations, including liver-related complications such as spontaneous bacterial peritonitis (SBP), hepatic encephalopathy (HE) and variceal bleeding. SBP is a life-threatening infection of the ascitic fluid that occurs in cirrhotic patients due to the translocation of bacteria from the gut into the peritoneal cavity. The dysbiotic gut microbiota in cirrhosis contributes to an increased risk of SBP by promoting bacterial overgrowth and translocation. In addition, the alteration in the immune response associated with dysbiosis impairs the ability of the immune system to clear bacterial infections, further exacerbating the risk of infection.

***Address for Correspondence:** Collins Meroni, Department of Gastroenterology and Hepatology, Athens Medical, P. Faliron Hospital, 175 62 Athens, Greece; E-mail: collinsmeroni89@gmail.com

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Conclusion

The shift toward a more pathogenic microbiota in cirrhosis is a key factor in the progression of liver disease and the development of associated complications. The dysbiotic gut microbiota in cirrhosis contributes to increased intestinal permeability, immune activation and the production of harmful metabolites, all of which exacerbate liver inflammation, fibrosis and other manifestations of the disease. Understanding the complex interactions between the gut microbiota, liver and immune system is crucial for developing targeted therapies to restore microbial balance and improve outcomes for patients with cirrhosis. As research in this field continues to evolve, new therapeutic strategies aimed at modifying the microbiota hold the potential to provide innovative treatments for cirrhosis and its complications.

References

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