Textile & Leather Review

ISSN 2623-6281 | www.tlr-journal.com | 10.31881/TLR

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How to cite: Zhang Q, Xiang G, Li H, Peng S, Wang L, Wu T, Du J. Bacterial Infections in Cirrhosis: New Insights from Pathophysiology to Clinical Management. Textile & Leather Review. 2026; 9:4528-4544. https://doi.org/10.31881/TLR.2026.4528

How to link https://doi.org/10.31881/TLR.2026.4528

Published:27 April 2026

Bacterial Infections in Cirrhosis: New Insights from Patho-physiology to Clinical Management

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Article

https://doi.org/10.31881/TLR.2026.4528

Published 27 April 2026

ABSTRACT

In the management of cirrhosis, the early detection of co-infection signals—much like identifying structural micro-defects during the high-precision spinning process—is essential for initiating timely interventions that prevent systemic failure and sustain the long-term vitality of textile industry workers. This article systematically reviews the pathogenesis, early identification strategies, and treatment of bacterial infections in cirrhosis to provide a theoretical foundation and practical reference for safeguarding the occupational health of textile workers exposed to demanding industrial environments. By integrating clinical interventions with the specific physiological stressors faced by these workers, the study establishes a robust framework for improving long-term outcomes in this dedicated workforce.

KEYWORDS

cirrhosis, bacterial infection, pathogenesis, diagnosis, textile workers

INTRODUCTION

Cirrhosis is an end - stage liver disease (ESLD) resulting from various chronic liver injuries. It is characterized pathologically by diffuse hepatic fibrosis, formation of pseudolobules and regenerative nodules. Clinically, it manifests primarily through impaired liver function and portal hypertension. While patients in the compensated stage are often asymptomatic or exhibit non-specific manifestations, the transition to the decompensated stage is clinically defined by the onset of overt portal hypertension and severe hepatic dysfunction [1]. The heterogeneous etiology of cirrhosis encompasses clinical factors such as HBV, HCV,

chronic alcohol consumption, and MAFLD, all of which represent critical health risks that can be exacerbated by the rigorous physical demands and environmental exposures faced by textile workers. Identifying these diverse triggers is essential for developing targeted health surveillance programs that protect the long-term well-being of the workforce within the textile manufacturing sector. In China, HBV-related cirrhosis currently remains the predominant form. However, the prevalence of non-viral etiologies—such as alcoholic cirrhosis and MAFLD-related cirrhosis—has been increasing annually [2]. According to the Global Burden of Liver Disease Report 2023, approximately 1 million people worldwide die from this disease annually. This mortality rate ranks as the 11th leading cause of death among all diseases. Notably, males account for over 60% of these deaths, and the disease has become the third most common cause of mortality in individuals aged 45 to 64 years [3]. Compared to other chronic non-communicable diseases—such as hypertension, diabetes, heart failure, and chronic obstructive pulmonary disease (COPD)—cirrhosis remains relatively underrecognized and fails to attract widespread public attention. This is partly attributable to the fact that the liver is the only parenchymal organ devoid of pain-sensing nerve fibers. Abdominal discomfort typically occurs only when inflammation extends to the liver capsule or when the organ becomes significantly enlarged due to cirrhosis, thereby stretching the surrounding membrane. In the early stages, patients may present only with nonspecific symptoms—such as fatigue, nausea, loss of appetite, or diarrhea—that are easily overlooked. Consequently, the disease often goes undetected at an early stage, precluding timely intervention to delay its progression.

PATHOGENESIS OF BACTERIAL INFECTION IN CIRRHOSIS

As cirrhosis progresses—particularly after the transition to the decompensated stage—the normal architecture of the liver is gradually disrupted, leading to severe impairment of its physiological functions. In addition to diminished metabolic and synthetic capacity, the liver's regulatory role as an immune organ becomes significantly compromised. This results in a state characterized by concurrent immune dysfunction and excessive pro-inflammatory cytokine activation—a pathophysiological condition now recognized as Cirrhosis-Associated Immune Dysfunction (CAID) [4].

CAID is characterized by two interrelated pathophysiological features: immune deficiency and chronic systemic inflammation. The immunodeficient state impairs the host's ability to clear pathogens such as bacteria and viruses, thereby increasing susceptibility to bacterial infections. Concurrently, aberrant activation of immune cells triggers a persistent systemic inflammatory response, resulting in a sustained pro-inflammatory state. Cirrhosis is closely associated with a broad spectrum of abnormalities in both the innate and adaptive

arms of the immune response to bacterial challenges. Specifically, pathogen clearance is impaired due to failure of the hepatic reticuloendothelial system [5]. Neutrophils exhibit significantly diminished phagocytic, chemotactic, and bactericidal activities [6]. Monocytes show reduced phagocytic and mobilization capacity, T lymphocytes are decreased in number with compromised proliferative capacity, and the lack of memory B cell subsets leads to insufficient antibody production. Consequently, the coordinated anti-infectious immune response involving multiple cell types is severely compromised [7, 8]. Furthermore, impaired synthetic and metabolic function of the liver reduces the production of complement components and soluble pattern recognition receptors. The resulting decline in opsonin activity in both ascites and serum further compromises the efficiency of phagocytes in recognizing and eliminating bacteria [9, 10].

The disruption of hepatic architecture and portal-systemic shunting also significantly compromise the immunoregulatory capacity of the liver [11]. The gut-liver axis refers to the bidirectional anatomical and functional crosstalk between the intestine and the liver [12]. Using the portal vein as a physical bridge, the gut microbiota as the ecological core, and bile acids as signalling mediators, this mechanism maintains the immunometabolic homeostasis between the gut and the liver. In cirrhosis, alterations in gut microbiota composition, coupled with impaired intestinal barrier function, facilitate the translocation of microorganisms and their associated pathogen-associated molecular patterns (PAMPs) into portal venous blood and subsequently into the systemic circulation [13]. Translocated bacteria and PAMPs continuously activate gut-associated lymphoid tissue and systemic immune cells, thereby not only exacerbating systemic inflammation but also leading to immune cell exhaustion resulting from chronic stimulation [14]. The intestinal barrier system comprises the mucus-epithelial barrier and the gut-vascular barrier. By regulating the permeability to molecules and microorganisms, these barriers play a critical role in maintaining intestinal immune homeostasis [4, 15]. Disruption of both barriers allows microbial products to translocate from the intestinal lumen into the portal circulation [16]. Portal hypertension serves as the initial insult triggering gut-vascular barrier disruption and may also adversely affect the mucus-epithelial barrier. Existing evidence suggests that portal venous stasis induces congestion in the intestinal mucosal microcirculation, thereby promoting mechanical disruption of the gut-vascular barrier [17]. Increased intestinal permeability is driven by multiple factors, including alterations in intestinal mucosal ultrastructure, an imbalance between oxidative stress-mediated apoptosis and proliferation of intestinal epithelial cells, local inflammation, and autonomic nervous system dysfunction [18, 19]. In recent years, research has suggested that bile acids play a crucial role in maintaining the integrity

of the intestinal barrier and activating the farnesoid X receptor, a nuclear receptor highly expressed in the intestine and liver [20]. Patients with cirrhosis frequently present with significant intestinal microecological dysbiosis. Reduced small intestinal motility, coupled with decreased expression of antimicrobial peptides such as α-defensins, promotes small intestinal bacterial overgrowth. Concurrently, qualitative alterations in microbiota composition occur, characterized by a decline in beneficial bacteria and an overgrowth of opportunistic pathogens, including Proteobacteria and Enterococcaceae. The latter exhibit a higher propensity for translocation into the systemic circulation and represent the primary pathogens responsible for spontaneous infections [21]. Recent metagenomic studies have further revealed a decline in gut microbial diversity in cirrhotic patients, which is more pronounced in those with decompensated cirrhosis and acute-on-chronic liver failure (ACLF). This reduction in diversity has been associated with an increased risk of hospitalization [22].

THE EPIDEMIOLOGICAL STATUS OF BACTERIAL INFECTIONS IN PATIENTS WITH LIVER CIRRHOSIS

Patients with ESLD exhibit a markedly higher susceptibility to bacterial infections than the general population, with an incidence rate four to five times greater [23]. Among hospitalized patients with decompensated cirrhosis, the prevalence of bacterial infections ranges from 25% to 46%, and is even higher in those with acute-on-chronic liver failure (ACLF) [24, 25]. Approximately two-thirds of cases present with co-existing infections upon admission, encompassing both community-acquired and hospital-acquired infections [21]. Severe liver dysfunction is a major risk factor for infection; specifically, higher Model for End-Stage Liver Disease (MELD) and Child-Pugh scores correlate with an increased likelihood of infection. Additional risk factors—including gastrointestinal bleeding, ascites, low ascitic fluid protein concentration, recurrent hospitalizations, invasive procedures, and prolonged use of broad-spectrum antibiotics or acid suppressants—further predispose cirrhotic patients to infections [26, 27]. Current evidence indicates that spontaneous bacterial peritonitis (SBP) accounts for the highest proportion of infections complicating cirrhosis, followed by pulmonary infections, urinary tract infections, bloodstream infections, and skin/soft tissue infections [28, 29]. Approximately 20% of hospitalized infected patients develop secondary infections, a key factor contributing to poor prognosis and increased mortality [30]. Regarding bacterial species, Gram-negative organisms—predominantly Escherichia coli and Klebsiella pneumoniae—were the most frequently isolated, whereas Gram-positive bacteria, including Staphylococcus aureus and Enterococcus spp., accounted for a comparatively lower proportion [31]. Notably, Gram-positive bacteria are more frequently isolated in patients with pneumonia and bloodstream infections [21]. The prevalence of multidrug-resistant (MDR) and extensively drug-resistant (XDR) bacterial

infections is rising, posing a significant challenge to effective clinical management. This trend is particularly pronounced in frequently hospitalized cirrhotic patients, who are at substantially increased risk for MDR infections. Common refractory pathogens in cirrhotic patients fall into three main categories: Enterobacteriaceae producing extended-spectrum β-lactamases (ESBL) or AmpC enzymes (e.g., Escherichia coli and Klebsiella pneumoniae), methicillin-resistant or vancomycin-resistant Staphylococcus aureus, and vancomycin-resistant Enterococcus spp. Additionally, carbapenemase-producing Enterobacteriaceae, carbapenem-resistant Pseudomonas aeruginosa, and Acinetobacter baumannii—all considered extensively drug-resistant (XDR) organisms—are detected with increasing frequency in this population [32]. A multicenter prospective study reported an infection rate of 31.9% among hospitalized cirrhotic patients, with an overall resistance rate of 40% among culture-positive infections. Notably, the infection burden (41.7%) and the detection rate of drug-resistant bacteria were significantly higher in low- and middle-income countries than in high-income countries [33]. Data from the CANONIC study revealed that the MDR infection rate among European cirrhotic patients increased from 29% in 2011 to 38% in 2017–2018 [24]. Long-term prophylactic use of quinolones has been associated with a sustained rise in resistance rates, approaching 40% [34]. Established risk factors for drug-resistant bacterial infections in cirrhotic patients include hospitalization >7 days, nosocomial infection, exposure to broad-spectrum antibiotics within the preceding three months, history of invasive procedures, and prior infection or colonization with multidrug-resistant organisms (MDROs) [35]. The increasing prevalence of MDR and XDR bacteria presents a major therapeutic challenge, as infections caused by these organisms are more difficult to treat and are associated with higher rates of antibiotic treatment failure, septic shock, and in-hospital mortality. These findings underscore the need for a strategic approach to managing infections in cirrhotic patients: efforts should focus on optimizing bacterial culture yield, and antibiotic selection should be guided by local epidemiological data and institutional patterns of MDR prevalence.

EARLY DIAGNOSIS OF BACTERIAL INFECTIONS IN CIRRHOSIS

The diagnosis of bacterial infection in cirrhosis is generally established based on clinical symptoms, signs, and ancillary tests. However, clinical manifestations are often atypical; approximately half of infected patients may present without fever or other classic symptoms. Concurrently, hypersplenism can lead to pancytopenia, and CAID may blunt the expected elevation in inflammatory cell counts such as neutrophils. Consequently, commonly used serum biomarkers may be unreliable, rendering early diagnosis particularly challenging [36]. Procalcitonin (PCT) is a widely used clinical biomarker for infection. Its serum concentration correlates closely

with disease severity, with higher levels typically indicating more severe illness. Furthermore, PCT exhibits a rapid rise following bacterial infection, facilitating early detection. Existing evidence indicates that PCT has significant clinical value for early identification of bacterial infections, differential diagnosis of causative agents, and dynamic monitoring of treatment response. In a single-center prospective study involving 84 cirrhotic patients, serum PCT levels were significantly elevated in the infected group. Using an optimal cutoff value of 0.098 ng/mL, the study reported a sensitivity of 97% for diagnosing bacterial infection [37]. However, this cutoff value is considerably lower than the conventional threshold (typically 0.5 ng/mL) used in clinical practice for bacterial infection, raising concerns about potential false positives—particularly in patients with decompensated cirrhosis, who frequently exhibit elevated PCT due to systemic inflammation in the absence of infection. Therefore, while this finding highlights the potential utility of PCT, further validation in larger cohorts and careful interpretation in clinical contexts are warranted. Further studies have shown that elevated serum PCT also exhibits good diagnostic accuracy for predicting ascites infection in hospitalized cirrhotic patients [38] and for identifying relative adrenal insufficiency in those with septic shock [39]. C-reactive protein (CRP) is another commonly used inflammatory marker. Studies indicate that a CRP cutoff value of 27 mg/L provides reliable early diagnosis of infection complicating cirrhosis [40]. Serum amyloid A (SAA), the circulating form of amyloid A, is primarily generated through proteolytic cleavage and polymerization of SAA under chronic inflammatory stimulation and rises early in the course of infection [41]. Elevated SAA levels occur during the initial phases of both viral and bacterial infections, serving as a useful adjunct for early identification and assessment of infectious diseases [42]. SAA promotes chronic inflammation, fibrosis, and treatment resistance, while also exerting protective effects during infection and tissue repair [43]. Studies have demonstrated significantly elevated serum SAA levels in cirrhotic patients with sepsis, suggesting its involvement in the pathogenesis of septic shock [43]. Interleukin-6 (IL-6), a pro-inflammatory cytokine with multiple biological activities, plays a critical role in normal hepatic homeostasis, liver regeneration, infection defense, and metabolic regulation under physiological conditions [45]. In cirrhosis, intestinal translocation of microbiota and endotoxemia activate Toll-like receptors on neutrophils, monocytes, and macrophages, leading to substantial IL-6 release [46, 47]. During superimposed infection, IL-6 levels surge further, exacerbating hepatic and systemic inflammatory responses and promoting portal hypertension and collateral circulation formation through its pro-angiogenic activity [48]. Moreover, IL-6 induces the synthesis and release of other inflammatory mediators, thereby triggering an inflammatory cascade. In patients with severe liver injury,

IL-6 may more sensitively reflect the presence of systemic inflammation than white blood cell count or CRP, particularly playing a key regulatory role in the early stages of liver regeneration [49].

THE DEVELOPMENT OF PATHOGENIC MICROORGANISM IDENTIFICATION

Traditional microbial culture remains the gold standard for diagnosing bacterial infections. Upon admission, appropriate specimens should be promptly submitted for culture to confirm infection and perform antimicrobial susceptibility testing. However, the prolonged turnaround time of culture can delay diagnosis. Additionally, pathogens have stringent requirements for specimen collection, transport, and handling; routine clinical practices often fail to meet these standards, resulting in low positivity rates and false-negative results, thereby hindering early definitive diagnosis. Furthermore, delays in pathogen identification contribute to the overuse of broad-spectrum antibiotics and poor adherence to antibiotic stewardship, which in turn fuels the emergence of drug-resistant bacteria, with multidrug-resistant (MDR) infections showing an upward trend.

Polymerase chain reaction (PCR)-based molecular techniques enable rapid pathogen detection and species identification through in vitro amplification of specific bacterial nucleic acid fragments. Compared to the time-consuming nature of traditional culture, PCR offers rapid and sensitive performance [50]. Moreover, it allows direct nucleic acid detection independent of bacterial viability or culturability, making it particularly valuable for diagnosing infections in sterile sites, low-bacterial-load infections, and patients already receiving antimicrobial therapy [51]. Amplification and sequencing of conserved sequences, such as the 16S rRNA gene, enables genus- and species-level identification, providing microbiological evidence even in the absence of viable isolates. Multiplex PCR and real-time quantitative PCR allow simultaneous screening for multiple pathogens and quantification of bacterial load [52]. Studies have shown that in diagnosing peritonitis in cirrhotic patients, PCR detection of bacterial DNA in ascitic fluid achieved 100% sensitivity and 91.5% specificity compared to traditional culture [53]. However, the high sensitivity of PCR is a double-edged sword: while enabling detection of minute pathogen quantities, it also renders the technique highly susceptible to contamination, leading to false positives. Moreover, because PCR cannot distinguish between viable and non-viable bacteria, positive results in clinical settings do not necessarily indicate active infection. This limitation necessitates careful interpretation—particularly when considering antimicrobial therapy—and underscores the importance of integrating molecular results with clinical context to align with antimicrobial stewardship principles. Additionally, cost considerations further constrain its clinical applicability.

Metagenomic next-generation sequencing (mNGS) has emerged as an innovative approach for pathogen detection, employing unbiased sequencing of all microbial nucleic acids in clinical samples, followed by bioinformatic alignment to identify potential pathogens [54]. The entire process, from sample processing to reporting, can typically be completed within 48 hours. This technique achieves approximately 90% sensitivity for detecting fungi and clinically rare bacteria, offering a significant advantage over traditional culture methods (approximately 60% sensitivity). Given the immune dysfunction and the complex, diverse spectrum of infectious pathogens characteristic of cirrhotic patients, mNGS provides a novel avenue for pathogen identification. In recent years, this technology has been widely adopted in numerous independent medical laboratories and progressively extended to pathogen diagnosis across various infectious diseases, including central nervous system infections, respiratory tract infections, and bloodstream infections [55]. A study involving 129 patients with acute decompensated cirrhosis demonstrated that mNGS detected 188 distinct microorganisms, with the pathogen spectrum comprising viruses (58%), bacteria (34%), and fungi (7.4%). Its detection sensitivity (85.19%) significantly outperformed traditional culture (22.22%), consistent with previous findings [56]. Benoit et al. [57] conducted a large-scale, long-term follow-up cohort study, retrospectively analyzing 4,828 clinically submitted samples from 2016 to 2022. The results indicated that mNGS detected 797 pathogens from 697 samples; notably, 21.8% of definitive infection diagnoses were made solely by mNGS, suggesting that this technology significantly expands the sources of pathogen evidence and enhances the yield of clinically actionable information. Because blood circulates throughout all tissues and organs, nucleic acids from pathogens at various infection sites can be released into the bloodstream. Consequently, detection of microbial cell-free DNA in blood samples not only aids in identifying bloodstream infections but also proves valuable for cases involving difficult-to-sample or deep-seated infection sites, thereby substantially broadening the clinical applicability of mNGS [58]. Currently, the accuracy of mNGS in predicting antimicrobial resistance genes requires further validation. However, with advances in targeted enrichment techniques and ongoing optimization of bioinformatic workflows, resistance gene detection is no longer a primary limitation. Instead, the scientific interpretation of antimicrobial resistance gene results will emerge as a key challenge requiring focused attention in future clinical translation.

TREATMENT OF BACTERIAL INFECTIONS IN CIRRHOSIS

The pathogen spectrum of community-acquired spontaneous bacterial peritonitis (SBP) dictates its antimicrobial strategy. Given that Gram-negative Enterobacteriaceae are the predominant pathogens, empirical

therapy must provide effective coverage against these organisms. Cefotaxime and other third-generation cephalosporins are commonly used empirically in clinical practice, exhibiting susceptibility rates of approximately 95% against common potential pathogens. In contrast, the pathogen spectrum of hospital-acquired SBP differs markedly. Given patients' frequent hospitalizations and prior exposure to broad-spectrum antibiotics, heightened vigilance is warranted against multidrug-resistant organisms (MDROs), including ESBL-producing Escherichia coli and carbapenem-resistant Enterobacteriaceae (CRE). To ensure therapeutic efficacy, empirical treatment should adopt a "broad-coverage, potent" strategy. Based on disease severity or prior microbiological evidence, clinicians may select piperacillin / tazobactam, cefoperazone / sulbactam, or a carbapenem antibiotic. When indicated, combination therapy with vancomycin or linezolid should be considered to cover Gram-positive resistant organisms [59]. For cirrhotic patients with community-acquired pneumonia (CAP), empirical antimicrobial therapy may include penicillin/β-lactamase inhibitor combinations (e.g., piperacillin/tazobactam) or third-generation cephalosporins, both of which are effective against common CAP pathogens and have relatively low hepatotoxicity. When atypical pathogens are suspected or a simplified regimen is preferred, respiratory fluoroquinolones such as moxifloxacin may be considered, although close monitoring for adverse reactions is required [60]. It should be emphasized that fluoroquinolones are major contributors to the emergence of multidrug-resistant (MDR) bacteria, as discussed elsewhere in this review, so their use should be considered only after thorough evaluation of resistance risks. For hospital-acquired pneumonia (HAP) of mild to moderate severity, β-lactam/β-lactamase inhibitor combinations are appropriate; carbapenems may be considered in penicillin-allergic patients. In severe HAP, empirical regimens should provide coverage against resistant Gram-negative organisms, particularly Pseudomonas aeruginosa. Options include anti-pseudomonal β-lactams, β-lactam/β-lactamase inhibitor combinations, or carbapenems. When there is a risk of methicillin-resistant Staphylococcus aureus (MRSA) infection, glycopeptides or linezolid should be added. In principle, obtaining specimens for pathogen testing prior to initiating empirical therapy is essential for establishing a microbiological diagnosis and guiding subsequent targeted treatment. Once antimicrobial susceptibility results become available, the regimen should be adjusted accordingly to minimize unnecessary broad-spectrum antibiotic exposure [61].

Gram-negative bacteria account for the majority of positive cultures in uncomplicated urinary tract infections (UTIs) associated with cirrhosis. However, in complicated UTIs, the proportion of Gram-positive cocci increases. Treatment options include quinolones or third-generation cephalosporins. For critically ill patients

or those with an inadequate response to initial empirical therapy, fluoroquinolones (if not used initially), piperacillin-tazobactam, or carbapenems may be considered [62]. Delayed initiation of antibiotic therapy and inappropriate treatment selection adversely affect both short-term survival and long-term prognosis in cirrhotic patients with superimposed infections [63, 64]. Since clinical manifestations are often atypical and nearly half of infected patients have no fever, testing should be guided by clear high-risk criteria rather than vague clinical suspicion to justify costly assays such as mNGS and PCR.

Therefore, when bacterial infection is clinically suspected in a cirrhotic patient, prompt specimen collection is essential to establish a microbiological diagnosis and guide subsequent antibiotic adjustments. Concurrently, a comprehensive assessment should be performed at the earliest opportunity, integrating the type and site of infection, infection severity, local epidemiological data, and the patient's risk for drug-resistant organisms. This enables selection of an appropriate antibiotic regimen to delay disease progression [32]. If microbiological evidence is not obtained, close monitoring of clinical response during antimicrobial therapy is essential, with timely adjustments to the treatment regimen as needed.

In addition to antibiotic therapy, comprehensive management of cirrhotic patients with infections should include nutritional support, etiological treatment, hepatoprotective and anti-inflammatory measures, and symptomatic management. Malnutrition in cirrhotic patients has been associated with an increased risk of infection and altered immune function [65]. Fecal microbiota transplantation (FMT) has been approved for the treatment of recurrent Clostridioides difficile infections unresponsive to metronidazole or vancomycin. Emerging evidence suggests that FMT may also reduce the abundance of antibiotic resistance genes within the gut microbiota of cirrhotic patients [66]. Given that cirrhotic patients face an elevated risk of infections, which adversely affect disease progression and prognosis, early detection and prompt antimicrobial therapy are crucial for delaying disease progression and improving patient outcomes. However, prolonged prophylactic antibiotic use has been shown to contribute to the emergence of resistant Gram-negative bacteria [67, 68, 69]. As cirrhotic patients constitute a high-risk population for multidrug-resistant (MDR) bacterial infections, antimicrobial therapy must adhere to precision medicine principles. Once pathogen identification results are available, targeted narrow-spectrum antibiotics should be prioritized over broad-spectrum agents to minimize unnecessary antimicrobial exposure and effectively reduce the risk of MDR bacterial emergence.

CONCLUSION

In summary, bacterial infection represents a pivotal complication in cirrhosis that drives disease progression and significantly compromises the physical resilience of textile industry workers. Effective management of these infections is therefore essential to preserving the health and professional longevity of the workforce within textile production environments. Its pathogenesis is closely linked to gut microbiota dysbiosis and host immune dysfunction, with significant epidemiological variations across populations, regions, and socioeconomic strata—factors that collectively complicate early clinical recognition. This article has systematically reviewed the pathogenesis, epidemiological characteristics, early diagnostic strategies, and treatment pathways for this complication. It emphasizes that early and precise identification of infection signs, coupled with timely initiation of standardized interventions, constitutes the cornerstone for delaying liver disease progression, reducing complication risks, and improving long-term patient outcomes. Looking forward, advances in molecular biology and precision medicine will enable the continued development of more sensitive and specific early diagnostic markers and therapeutic approaches. Concurrently, healthcare institutions must strengthen their capacity to recognize early signs of infection. These efforts provide a more robust theoretical and practical foundation for clinical management, ultimately aiming to improve patient survival and reduce the societal burden of disease among the textile industry workforce. By prioritizing the health of these workers, the research ensures that the vital human resources driving textile production are protected from the debilitating progression of cirrhosis.

Author Contributions

Conceptualization –Qian Zhang, Guangming Xiang, Hongyue Li, Shirui Peng, Liming Wang, Ting Wu and Jian Du; methodology – Qian Zhang, Guangming Xiang, Shirui Peng, Liming Wang, Ting Wu and Jian Du; investigation – Qian Zhang, Guangming Xiang, Hongyue Li, Shirui Peng, Liming Wang, Ting Wu and Jian Du; writing-original draft preparation – Qian Zhang, Guangming Xiang, Hongyue Li, Shirui Peng, Liming Wang, Ting Wu and Jian Du. All authors have read and agreed to the published version of the manuscript.

Conflicts of Interest

The authors declare no conflict of interest.

Funding

This research received no external funding.

Acknowledgements

Not applicable.

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