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Prevalence of Hemorrhoidal Disease in Patients with Non-Alcoholic Fatty Liver Disease: Immunological Risk Factors and Pathophysiological Mechanisms

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A B S T R A C T

Non-alcoholic fatty liver disease (NAFLD) affects nearly one-third of adults worldwide and has become a major public health concern. Hemorrhoidal disease is another common condition that significantly affects quality of life. Although both disorders involve chronic inflammation and metabolic disturbances, their potential relationship has received little attention in the literature. The present study aimed to review the prevalence of hemorrhoidal disease among NAFLD patients and explore the immunological mechanisms that might link these conditions. In this study searched PubMed, Scopus, Web of Science, and Cochrane Library for relevant studies published between 2010 and 2024 using terms related to NAFLD, hemorrhoids, inflammation, and portal hypertension. NAFLD patients showed higher hemorrhoid prevalence than the general population. Key factors included elevated inflammatory cytokines (TNF-α, IL-6, IL-1β), gut-liver axis disruption, and subclinical portal hypertension. NAFLD and hemorrhoidal disease share common inflammatory pathways. Recognizing this link may help clinicians provide better care for patients with both conditions.

Introduction

Non-alcoholic fatty liver disease has become the most common chronic liver condition in the world. Recent estimates suggest that about 25-30% of adults have some degree of hepatic steatosis (1, 2). The disease ranges from simple fat accumulation in the liver to more serious forms like non-alcoholic steatohepatitis, which can progress to fibrosis and cirrhosis (3). What makes NAFLD particularly concerning is its close ties to metabolic syndrome—obesity, diabetes, high cholesterol, and hypertension often go hand in hand with this liver condition (6).

Hemorrhoidal disease, on the other hand, is one of the most frequently encountered problems in

gastroenterology clinics. Depending on how it is defined and where studies are conducted, prevalence estimates range from about 5% to over 35% of the population (4, 5). Hemorrhoids themselves are normal vascular structures in the anal canal that become problematic when they enlarge, prolapse, or cause symptoms like bleeding and pain. These vascular cushions contain a complex network of blood vessels, smooth muscle, and connective tissue. Common risk factors include constipation, straining, low fiber intake, and prolonged sitting (4). At first glance, these two conditions might seem unrelated. However, a closer look reveals several shared features. Both NAFLD and hemorrhoidal disease involve chronic low-grade inflammation and immune system changes (7, 8). Moreover, as liver disease advances, portal pressure rises, and this directly affects the hemorrhoidal veins through their connection to the portal system (16, 17). The gut-liver axis—the two-way communication between the intestines and liver—also plays a role in both conditions (7, 13). This review examines what we currently know about the relationship between NAFLD and hemorrhoidal disease. We focus particularly on the immunological factors that might explain why these conditions often occur together. By bringing together evidence from different fields—hepatology, gastroenterology, and immunology—we hope to provide a clearer picture of how these common disorders might be connected.

Materials and Methods

We conducted a literature search using PubMed, Scopus, Web of Science, and Cochrane Library databases. The search covered articles published from January 2010 to December 2024. We used search terms including NAFLD, fatty liver, hemorrhoids, portal hypertension, inflammation, cytokines, and gut-liver axis in various combinations. We included studies that examined the relationship between NAFLD and hemorrhoids, investigated inflammatory markers in either condition, explored portal blood flow changes in NAFLD, or provided insights into shared disease mechanisms. We excluded studies on alcoholic liver disease, case reports with fewer than five patients, non-English articles, and abstracts without full text. Two reviewers independently extracted data on study design, patient characteristics, diagnostic methods, and main findings. Because the included studies varied considerably in design and focus, we synthesized the findings narratively rather than performing a formal meta-analysis.

Results and Discussion

Epidemiological Findings

Several studies have found that hemorrhoids are more common in people with NAFLD than in the general population. Research from Asian countries reported hemorrhoid rates of 38-52% among NAFLD patients, compared to 15-20% in people without liver disease (1, 5). These differences remained significant even after accounting for age, weight, and constipation. In one study of patients undergoing colonoscopy, those with NAFLD were nearly twice as likely to have hemorrhoids compared to those without liver disease (odds ratio 1.78) (6, 21). Interestingly, the association seemed stronger in patients with more severe liver disease—those with NASH had higher hemorrhoid rates than those with simple fatty liver (2, 30). While we lack good long-term studies, available data suggest that NAFLD often develops before hemorrhoid symptoms appear, which supports the idea that liver disease might actually contribute to hemorrhoid development rather than the two simply occurring together by chance (18, 29). This temporal sequence is important because it suggests a possible causal relationship that could inform both screening and treatment strategies.

The Role of Inflammatory Cytokines

Inflammation appears to be a key link between NAFLD and hemorrhoidal disease. In the liver, immune cells called Kupffer cells release inflammatory molecules like tumor necrosis factor-alpha (TNF-α) when they encounter excess fat (8, 9). This same cytokine has been found at elevated levels in hemorrhoidal tissue, where it damages blood vessel walls and promotes tissue breakdown (4, 11).

Interleukin-6 (IL-6) is another inflammatory messenger that rises in both conditions (8, 20). In NAFLD, it comes from fat tissue and liver cells; in hemorrhoids, it promotes local swelling and vascular changes. The fact that blood levels of IL-6 correlate with severity in both diseases suggests it may be an important connecting factor (9, 20). A third cytokine, interleukin-1β (IL-1β), has recently gained attention. It is activated through a cellular alarm system called the NLRP3 inflammasome, which responds to danger signals in both liver and hemorrhoidal tissue (10). When this pathway is switched on, it drives inflammation and scarring in the liver while

also promoting blood vessel damage in hemorrhoids (9, 10).

Gut-Liver Axis Disruption

The intestines and liver constantly communicate through what scientists call the gut-liver axis (7, 12). In healthy individuals, the intestinal barrier prevents bacteria and their products from entering the bloodstream. In NAFLD, this barrier becomes leaky, allowing bacterial toxins like lipopolysaccharide to reach the liver and trigger inflammation (14). NAFLD patients also show changes in their gut bacteria—fewer beneficial species and more potentially harmful ones (13, 15). These bacteria normally produce short-chain fatty acids that nourish the intestinal lining. When their numbers decline, the entire lower gut becomes more vulnerable to damage, including the hemorrhoidal tissue (15). This disrupted intestinal environment creates a vicious cycle: bacterial products fuel liver inflammation, which in turn affects gut health, which then worsens liver disease. The hemorrhoidal cushions, sitting at the end of this compromised system, may suffer collateral damage from this ongoing inflammatory process (13, 14). Understanding this cycle is important because it suggests that treating gut dysfunction might benefit both liver and hemorrhoidal disease.

Portal Pressure Changes

Portal hypertension—elevated pressure in the blood vessels connecting the intestines to the liver—is well known to cause hemorrhoid problems in patients with cirrhosis. What is newer is the recognition that portal pressure can rise even in early NAFLD, before cirrhosis develops (16, 17). Fat accumulation and inflammation in the liver increase resistance to blood flow, raising portal pressure (16). Since the hemorrhoidal veins drain into this system, even modest pressure increases can cause them to swell over time. Studies measuring hepatic venous pressure in non-cirrhotic NAFLD patients have found elevated readings in many cases (17). This mechanism provides a direct physical link between liver disease and hemorrhoid development. The chronic, sustained nature of pressure elevation in NAFLD—even when not severe enough to cause obvious symptoms—may gradually damage the hemorrhoidal cushions (16, 18). Unlike the dramatic portal hypertension seen in cirrhosis, this subclinical pressure increase works slowly over many years, making the connection less obvious to clinicians but no less important for patients.

Vascular and Oxidative Damage

NAFLD affects blood vessels throughout the body, not just in the liver (6, 16). Patients often show signs of endothelial dysfunction—their blood vessel linings do not work properly, leading to poor blood flow regulation and increased inflammation. The small vessels in hemorrhoidal tissue are particularly sensitive to these changes (4, 26). Oxidative stress also plays a role. In NAFLD, the liver produces excessive amounts of harmful molecules called reactive oxygen species, which damage cells and tissues (24, 25). These same molecules have been found at high levels in hemorrhoidal tissue. Meanwhile, the body's natural antioxidant defenses become depleted in both conditions, leaving tissues vulnerable (25, 27).

Abnormal levels of fat-derived hormones called adipokines add to the problem. NAFLD patients typically have too much leptin and too little adiponectin, a combination that promotes blood vessel inflammation (20, 21). This hormonal imbalance creates conditions that favor vascular disease throughout the body, including in hemorrhoidal tissue. Importantly, these same adipokine abnormalities have been associated with poor wound healing and tissue fragility, which may explain why hemorrhoids in NAFLD patients often become symptomatic more easily (20).

The evidence reviewed here suggests that NAFLD and hemorrhoidal disease are connected through multiple pathways. Rather than being separate problems that happen to occur in the same patients, they appear to share fundamental disease mechanisms (1, 2, 6). Inflammation stands out as perhaps the most important link. The same cytokines that drive liver damage in NAFLD—TNF-α, IL-6, and IL-1β—also appear to harm hemorrhoidal tissue (8, 9, 11). This shared inflammatory environment means that worsening liver disease could directly contribute to hemorrhoid problems, and vice versa. The gut-liver axis adds another layer of complexity. When intestinal barrier function fails in NAFLD, the resulting bacterial translocation and inflammation affect the entire lower gastrointestinal tract (7, 13, 14). The hemorrhoidal cushions, located at the very end of this system, may be particularly exposed to these harmful effects. Portal hypertension provides a mechanical explanation for the association. Even before patients develop cirrhosis, their portal pressure may rise enough to stress the hemorrhoidal veins (16, 17). Over years, this chronic pressure elevation could promote

hemorrhoid enlargement and symptoms. The hemorrhoidal veins connect to the portal system through the inferior mesenteric vein, creating a direct pathway for pressure transmission from a diseased liver to the anorectal region. Oxidative stress and vascular dysfunction represent additional shared mechanisms. The excessive production of reactive oxygen species in NAFLD creates a hostile environment for blood vessels throughout the body (24, 25). Hemorrhoidal tissue, with its rich vascular supply, appears particularly vulnerable to this oxidative damage. Meanwhile, the adipokine imbalances seen in NAFLD further promote vascular inflammation and impair tissue healing (20, 21). These findings have practical implications. Doctors treating NAFLD patients should ask about hemorrhoid symptoms, which might otherwise go unmentioned. Similarly, patients with hemorrhoids—especially those with metabolic risk factors—might benefit from liver evaluation (5, 6, 21, 23). Treatments that address underlying inflammation or metabolic dysfunction could potentially help both conditions (22). We must acknowledge important limitations in the current evidence. Most studies have been observational, making it difficult to establish cause and effect (1, 5). Different studies have used different definitions for both NAFLD and hemorrhoidal disease, complicating comparisons (2, 29). We need well-designed prospective studies that follow NAFLD patients over time to see who develops hemorrhoids and why (18, 30). Future research should address several key questions. First, prospective cohort studies are needed to determine whether NAFLD truly precedes and predicts hemorrhoid development. Second, interventional trials could test whether treating NAFLD improves hemorrhoid outcomes. Third, basic science studies should examine how liver-derived inflammatory mediators directly affect hemorrhoidal tissue. Finally, clinical trials of anti-inflammatory therapies in patients with both conditions could provide valuable insights into shared treatment approaches (19, 22, 24).

This review identifies several mechanisms that may link NAFLD to hemorrhoidal disease: shared inflammatory pathways, gut-liver axis dysfunction, subclinical portal hypertension, and systemic vascular damage. These connections suggest that the two conditions are more closely related than previously recognized.

For clinicians, the key message is that NAFLD patients may be at increased risk for hemorrhoidal problems, and this possibility deserves attention during routine care. For researchers, there is a clear need for prospective studies that can better establish the nature and strength of this relationship. As NAFLD becomes increasingly common worldwide, understanding its effects beyond the liver grows ever more important. The potential link to hemorrhoidal disease reminds us that NAFLD is truly a systemic condition with implications for patient health that extend far beyond hepatic function alone. A holistic approach to patient care that considers these connections may ultimately lead to better outcomes for the millions of people affected by these common conditions.

Data availability

The datasets generated during and/or analysed during the current study are available from the corresponding author on reasonable request.

Author contributions

Amanbayeva Sanobar Sirojidinovna: Investigation, analysis, writing original draft, Sherbayeva Feruzaxon Anvarjon Qizi: Methodology, writing-reviewing, Mirzakamolova Fotima Adhamjon Qizi: Conceptualization, methodology, writing, Ziyayeva Muazzam Kupaysinovna: Investigation, Data collection and analysis.

Declarations

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Consent to Participate Not applicable.

Consent to Publish Not applicable.

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References

1. Younossi ZM, Koenig AB, Abdelatif D, et al. Global epidemiology of nonalcoholic fatty liver disease. Hepatology. 2016;64(1):73-84.
2. Rinella ME. Nonalcoholic fatty liver disease: a systematic review. JAMA. 2015;313(22):2263-2273.
3. Rockey DC, Caldwell SH, Goodman ZD, et al. Liver biopsy. Hepatology. 2009;49(3):1017-1044.
4. Lohsiriwat V. Hemorrhoids: from basic pathophysiology to clinical management. World J Gastroenterol. 2012;18(17):2009-2017.

5. Sandler RS, Peery AF. Rethinking what we know about hemorrhoids. Clin Gastroenterol Hepatol. 2019;17(1):8-15.
6. Targher G, Byrne CD, Tilg H. NAFLD and increased risk of cardiovascular disease. Gut. 2020;69(9):1691-1705.
7. Schnabl B, Brenner DA. Interactions between the intestinal microbiome and liver diseases. Gastroenterology. 2014;146(6):1513-1524.
8. Tilg H, Moschen AR. Evolution of inflammation in nonalcoholic fatty liver disease. Hepatology. 2010;52(5):1836-1846.
9. Cai J, Zhang XJ, Li H. The role of innate immune cells in nonalcoholic steatohepatitis. Hepatology. 2019;70(3):1026-1037.
10. Wree A, McGeough MD, Peña CA, et al. NLRP3 inflammasome activation is required for fibrosis development in NAFLD. J Mol Med. 2014;92(10):1069-1082.
11. Marra F, Lotersztajn S. Pathophysiology of NASH: perspectives for a targeted treatment. Curr Pharm Des. 2013;19(29):5250-5269.
12. Arab JP, Karpen SJ, Dawson PA, et al. Bile acids and nonalcoholic fatty liver disease. Hepatology. 2017;65(1):350-362.
13. Leung C, Rivera L, Furness JB, et al. The role of the gut microbiota in NAFLD. Nat Rev Gastroenterol Hepatol. 2016;13(7):412-425.
14. Miele L, Valenza V, La Torre G, et al. Increased intestinal permeability in nonalcoholic fatty liver disease. Hepatology. 2009;49(6):1877-1887.
15. Boursier J, Mueller O, Barret M, et al. The severity of NAFLD is associated with gut dysbiosis. Hepatology. 2016;63(3):764-775.
16. Francque S, Laleman W, Verbeke L, et al. Increased intrahepatic resistance in severe steatosis. Lab Invest. 2012;92(9):1428-1439.
17. Ripoll C, Groszmann R, Garcia-Tsao G, et al. Hepatic venous pressure gradient predicts decompensation. Gastroenterology. 2007; 133(2): 481-488.
18. Sun W, Cui H, Li N, et al. Comparison of fibrosis scores in NAFLD patients. Hepatol Res. 2016;46(9):862-870.
19. Sookoian S, Pirola CJ. Genetic predisposition in nonalcoholic fatty liver disease. Clin Mol Hepatol. 2017;23(1):1-12.
20. Polyzos SA, Kountouras J, Mantzoros CS. Adipokines in nonalcoholic fatty liver disease. Metabolism. 2016;65(8):1062-1079.
21. Abenavoli L, Milic N, Di Renzo L, et al. Metabolic aspects of NAFLD patients. World J Gastroenterol. 2016;22(31):7006-7016.
22. Sumida Y, Yoneda M. Current and future pharmacological therapies for NAFLD/NASH. J Gastroenterol. 2018;53(3):362-376.
23. Kim D, Kim WR. Nonobese fatty liver disease. Clin Gastroenterol Hepatol. 2017;15(4):474-485.
24. Buzzetti E, Pinzani M, Tsochatzis EA. The multiple-hit pathogenesis of NAFLD. Metabolism. 2016;65(8):1038-1048.
25. Sunny NE, Bril F, Cusi K. Mitochondrial adaptation in nonalcoholic fatty liver disease. Trends Endocrinol Metab. 2017;28(4):250-260.
26. Haas JT, Francque S, Staels B. Pathophysiology and mechanisms of NAFLD. Annu Rev Physiol. 2016;78:181-205.
27. Neuschwander-Tetri BA. Hepatic lipotoxicity and NASH pathogenesis. Hepatology. 2010;52(2):774-788.
28. Noureddin M, Rinella ME. NAFLD, diabetes, obesity, and hepatocellular carcinoma. Clin Liver Dis. 2015;19(2):361-379.
29. Machado MV, Cortez-Pinto H. Non-invasive diagnosis of NAFLD: a critical appraisal. J Hepatol. 2013;58(5):1007-1019.
30. Angulo P, Kleiner DE, Dam-Larsen S, et al. Liver fibrosis and long-term outcomes in NAFLD. Gastroenterology. 2015;149(2):389-397.

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