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Correlation of Fecal SCFA and Calprotectin with hsCRP in Patients with Liver Cirrhosis

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Abstract
Introduction: *Liver cirrhosis disrupts the gut–liver axis through dysbiosis, intestinal barrier impairment, and bacterial translocation, which drive systemic inflammation.* ***Objective:*** *To analyze the correlation of fecal short-chain fatty acids (SCFA) and fecal calprotectin with high-sensitivity C-reactive protein (hsCRP) levels in patients with liver cirrhosis.* ***Method:*** *This analytic cross-sectional study enrolled 32 consecutive patients with cirrhosis at Prof. Dr. R. D. Kandou General Hospital, Manado. Fecal SCFA were measured by gas chromatography–mass spectrometry, fecal calprotectin by ELISA, and serum hsCRP by immunoturbidimetry. Data were analyzed using the Shapiro–Wilk test and Spearman's correlation.* ***Result and Discussion:*** *The mean age was 57.16 ± 11.27 years and 81.3% of subjects were male. The median hsCRP was 2.60 mg/L (IQR 9.55) and the median fecal calprotectin was 232.55 µg/g (IQR 538.40), with elevated fecal calprotectin in 65.6% of subjects. Fecal calprotectin showed a significant moderate positive correlation with hsCRP ($r = 0.520$; $p = 0.002$), whereas total SCFA did not correlate with hsCRP ($r = 0.013$; $p = 0.944$) or fecal calprotectin ($r = 0.106$; $p = 0.562$).* ***Conclusions:*** *Intestinal mucosal inflammation, reflected by fecal calprotectin, is associated with systemic inflammation in liver cirrhosis, whereas total fecal SCFA alone is insufficient as an inflammatory biomarker.*

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Introduction

Liver cirrhosis is the end stage of chronic liver injury, in which normal hepatic architecture is replaced by fibrous tissue and regenerative nodules, leading to hepatic dysfunction, portal hypertension, and life-threatening complications (Ginès et al., 2021). Cirrhosis remains a major cause of morbidity and mortality worldwide; in 2019 there were an estimated 2.05 million new cases and 1.47 million deaths attributable to cirrhosis globally (Xiao et al., 2023). Systemic inflammation is increasingly recognized as a key driver of disease progression, and inflammatory markers such as interleukin-6 (IL-6), tumor necrosis factor-α (TNF-α), and C-reactive protein are elevated in patients with cirrhosis. High-sensitivity C-reactive protein (hsCRP) has been associated with the severity of hepatic steatosis and fibrosis in chronic liver disease (Zhu et al., 2022).

The gut–liver axis has emerged as a central concept in the pathophysiology of cirrhosis. Portal hypertension, altered bile acid flow, and impaired immunity promote intestinal dysbiosis, disruption of the mucosal barrier, and translocation of bacteria and microbial products into the portal circulation, which activate innate immune responses and sustain systemic inflammation (Albillos et al., 2020; Arab et al., 2018). Fecal calprotectin, a neutrophil-derived S100A8/A9 protein, is a non-invasive marker of intestinal mucosal inflammation (Wang et al., 2025). Jothimani et al. (2023) evaluated fecal calprotectin in patients with cirrhosis and proposed it as a marker of cirrhosis-related complications, while Matiollo et al. (2022) reported that elevated calprotectin levels were associated with the severity of liver dysfunction and mortality in acute decompensation of cirrhosis.

Short-chain fatty acids (SCFA), mainly acetate, propionate, and butyrate, are the end products of microbial fermentation of dietary fiber. They serve as the main energy source for colonocytes, strengthen tight junctions and mucus production, and exert immunomodulatory effects through histone deacetylase (HDAC) inhibition and G-protein-coupled receptor (GPCR) signaling (Deleu et al., 2021; He et al., 2020). However, fecal SCFA concentrations do not directly represent systemic SCFA exposure because they reflect the balance between intestinal microbial production, colonic absorption, and residual fecal excretion. Most SCFAs are absorbed and metabolized by intestinal epithelial cells and the liver before reaching systemic circulation. Consequently, fecal SCFA levels should be interpreted as indirect indicators of intestinal microbial metabolism rather than direct measures of circulating SCFA availability or biological activity. Alterations of the gut microbiome in cirrhosis are linked to intestinal and systemic inflammation, bacterial translocation, and disease severity (Lee & Suk, 2020), and the gut microbiota of patients with decompensated cirrhosis shows a reduced functional capacity for SCFA fermentation (Baltazar-Díaz et al., 2022).

Although hsCRP is widely used as a sensitive marker of systemic inflammation, its interpretation in advanced cirrhosis requires caution. CRP is primarily synthesized by hepatocytes in response to inflammatory cytokines, particularly IL-6. In patients with severe hepatic dysfunction, impaired synthetic capacity may attenuate CRP production despite substantial systemic inflammation or infection. Consequently, relatively low hsCRP concentrations do not necessarily indicate the absence of inflammatory activity, while elevated concentrations may reflect concurrent infections or other inflammatory conditions rather than cirrhosis-related inflammation alone. Therefore, hsCRP should be interpreted alongside liver disease severity, clinical findings, and other inflammatory indicators when evaluating systemic inflammation in patients with cirrhosis

Although fecal SCFA, fecal calprotectin, and hsCRP represent different levels of the gut-liver axis luminal microbial metabolism, intestinal mucosal inflammation, and systemic inflammation studies evaluating these three markers simultaneously in the same patients with cirrhosis remain very limited, particularly in Indonesia. Importantly, correlations among these biomarkers should not be interpreted as direct evidence of systemic SCFA exposure or causal relationships, given the physiological limitations of fecal SCFA measurements and the potential influence of impaired hepatic synthesis on hsCRP concentrations. This study therefore aimed to analyze the correlation of fecal SCFA and fecal calprotectin with hsCRP levels, as well as the correlation between fecal SCFA and fecal calprotectin, in patients with liver cirrhosis.

Method

This was an observational analytic study with a cross-sectional design conducted at the Gastroenterohepatology Outpatient Clinic and internal medicine wards of Prof. Dr. R. D. Kandou General Hospital, Manado, Indonesia, over a six-month period from January to June 2026. Subjects were recruited by consecutive sampling. The minimum sample size, calculated using the correlation coefficient formula ($\alpha = 0.05$, $\beta = 0.20$, $r = 0.427$), was 32 subjects. Inclusion criteria were age 18–65 years, a documented diagnosis of liver cirrhosis based on transient elastography using etiology-specific liver stiffness cut-offs (≥12.5 kPa for hepatitis C, ≥11 kPa for hepatitis B, ≥10.5 kPa for non-alcoholic fatty liver disease, ≥14.6 kPa for hepatitis C with HIV co-infection, and ≥18.5 kPa for alcohol-related liver disease), and willingness to participate with signed informed consent. Exclusion criteria were a history of inflammatory bowel disease, malignancy, or autoimmune disease; active infection or ongoing treatment for infection; obesity (body mass index >25 kg/m²); type 2 diabetes mellitus; chronic kidney disease (estimated glomerular filtration rate <60 mL/min/1.73 m² for at least three months); and use of probiotics or drugs affecting fecal calprotectin levels (non-steroidal anti-inflammatory drugs and steroids) within the previous month.

Fecal SCFA (acetic, propionic, butyric, and valeric acids, absolute butyric acid, and total SCFA) were measured using gas chromatography–mass spectrometry (Prohealthy Gut). Fecal calprotectin was measured using an enzyme-linked immunosorbent assay (ELISA), with levels >50 µg/g considered elevated. Serum hsCRP was measured from peripheral venous blood using an immunoturbidimetric method, with a normal value of <5.3 mg/L. Data were analyzed using the Statistical Package for the Social Sciences (SPSS). Normality was assessed with the Shapiro–Wilk test; normally distributed data are presented as mean ± standard deviation (SD) and non-normally distributed data as median (interquartile range, IQR). Because several variables were not normally distributed, correlations were analyzed using Spearman's rank correlation test, with $p < 0.05$ considered statistically significant. The study protocol was approved by the Health Research Ethics Committee of Prof. Dr. R. D. Kandou General Hospital, Manado (No. [ethical clearance number]), and all subjects provided written informed consent.

Result and Discussion

1. Result

A total of 32 patients with liver cirrhosis fulfilled the inclusion and exclusion criteria. The mean age was 57.16 ± 11.27 years, and most subjects were male (26 subjects; 81.3%). The median hsCRP level was 2.60 mg/L (IQR 9.55); 21 subjects (65.6%) had hsCRP <5.3 mg/L and 11 subjects (34.4%) had hsCRP ≥5.3 mg/L. The median fecal

calprotectin level was 232.55 µg/g (IQR 538.40), and 21 subjects (65.6%) had elevated fecal calprotectin (>50 µg/g). Acetic acid was the dominant SCFA component (61.63 ± 11.62%), followed by propionic acid (21.63 ± 7.65%) and butyric acid (10.75 ± 6.54%); the median absolute butyric acid was 1.35 mg/mL (IQR 2.25), and the mean total SCFA was 11.19 ± 4.70 mg/mL (Table 1).

The Shapiro–Wilk test showed that age, the proportions of acetic, propionic, and butyric acids, and total SCFA were normally distributed ($p > 0.05$), whereas hsCRP, fecal calprotectin, absolute butyric acid, and valeric acid were not ($p < 0.05$); therefore, Spearman's correlation test was used for all bivariate analyses. Fecal calprotectin showed a significant moderate positive correlation with hsCRP ($r = 0.520$; $p = 0.002$). In contrast, total SCFA showed a very weak and non-significant positive correlation with hsCRP ($r = 0.013$; $p = 0.944$) and with fecal calprotectin ($r = 0.106$; $p = 0.562$) (Table 2).

Table 1
Characteristics of the Subjects (N = 32)

No	Variable	n (%)	Mean ± SD	Median (IQR)
1	Age (years)	–	57.16 ± 11.27	–
2	Sex: male	26 (81.3)	–	–
3	Sex: female	6 (18.8)	–	–
4	hsCRP (mg/L)	–	–	2.60 (9.55)
5	Fecal calprotectin (µg/g)	–	–	232.55 (538.40)
6	Acetic acid (%)	–	61.63 ± 11.62	–
7	Propionic acid (%)	–	21.63 ± 7.65	–
8	Butyric acid (%)	–	10.75 ± 6.54	–
9	Absolute butyric acid (mg/mL)	–	–	1.35 (2.25)
10	Valeric acid (%)	–	–	2.00 (2.20)
11	Total SCFA (mg/mL)	–	11.19 ± 4.70	–

*SD = standard deviation; IQR = interquartile range; SCFA = short-chain fatty acids; hsCRP = high-sensitivity C-reactive protein.

Table 2
Correlation of Fecal SCFA and Fecal Calprotectin with hsCRP

No	Variables	N	Coefficient (r)	p-value
1	Total SCFA – hsCRP	32	0.013	0.944
2	Fecal calprotectin – hsCRP	32	0.520	0.002*
3	Fecal calprotectin – Total SCFA	32	0.106	0.562

Spearman's rank correlation test; *$p < 0.05$ (statistically significant).

2. Discussion

Characteristics of the Subjects

The predominance of older adults (mean age 57 years) in this study is consistent with the natural history of cirrhosis as a slowly progressive chronic liver disease that is usually diagnosed after years of accumulated hepatic injury, inflammation, and fibrosis (Ginès et al., 2021). The male predominance (81.3%) is in line with global epidemiological data showing a higher burden of cirrhosis in men, which is related to greater exposure to alcohol, chronic viral hepatitis, and metabolic risk factors, while estrogen may exert protective effects against hepatic inflammation and fibrogenesis in women (Xiao et al., 2023). The wide range of hsCRP and fecal calprotectin values indicates heterogeneity in the degree of systemic and intestinal inflammation among subjects, which has been attributed to gut–liver axis disruption with bacterial translocation and systemic immune activation (Albillos et al., 2020).

However, variations in liver disease severity may also contribute to differences in hsCRP concentrations. In advanced cirrhosis, impaired hepatic synthetic capacity can attenuate CRP production despite substantial inflammatory activity. Child-Pugh classification and Model for End-Stage Liver Disease (MELD) scores are therefore relevant when interpreting hsCRP, as patients with more severe hepatic dysfunction may exhibit a diminished acute-phase response. Consequently, hsCRP concentrations should not be interpreted independently of hepatic functional status. Because the influence of Child-Pugh class and MELD score on hsCRP was not specifically evaluated in this analysis, the extent to which disease severity affected the observed biomarker relationships remains uncertain

Correlation between Fecal Calprotectin and hsCRP

The main finding of this study was a significant moderate positive correlation between fecal calprotectin and hsCRP ($r = 0.520$; $p = 0.002$), indicating that greater intestinal mucosal inflammation was associated with higher systemic inflammation. This supports the concept that cirrhosis is not merely a localized liver disease but also involves disruption of the gut–liver axis. Dysbiosis, increased intestinal permeability, and translocation of bacteria and microbial products into the portal circulation activate immune cells and the release of IL-6 and TNF-α, which in turn stimulate hepatic synthesis of C-reactive protein (Albillos et al., 2020). Fecal calprotectin is released mainly by activated neutrophils infiltrating the intestinal mucosa, and its elevation reflects mucosal inflammation and barrier dysfunction (Wang et al., 2025).

Nevertheless, elevated fecal calprotectin in cirrhosis may not exclusively indicate classical inflammatory mucosal disease. Portal hypertension can cause portal hypertensive enteropathy, characterized by mucosal congestion, edema, vascular ectasia, and increased intestinal permeability. These structural and hemodynamic abnormalities may contribute to mucosal injury and secondary neutrophil activation, potentially increasing fecal calprotectin even without overt inflammatory bowel disease or classical inflammatory lesions. Therefore, elevated fecal calprotectin should be interpreted as a marker of intestinal mucosal disturbance rather than definitive evidence of primary intestinal inflammation. Because endoscopic assessment of portal hypertensive enteropathy was not included in this study, its contribution to fecal calprotectin elevation could not be determined.

This finding is consistent with Anindita et al. (2023), who reported a positive correlation between fecal calprotectin and CRP ($r = 0.57$; $p < 0.001$). Although that study involved patients with colitis, the underlying mechanisms—mucosal inflammation, barrier impairment, and release of systemic inflammatory mediators—are likely shared with the chronic inflammatory state of cirrhosis. Elevated calprotectin has also been associated with complications and mortality in cirrhosis (Jothimani et al., 2023; Matiollo et al., 2022). Clinically, the combination of fecal calprotectin and hsCRP may serve as a non-invasive panel to identify patients with cirrhosis who have a higher inflammatory burden and may warrant closer monitoring, and it supports therapeutic strategies that target intestinal inflammation and the gut microbiota as part of cirrhosis management.

However, the clinical interpretation of this correlation requires consideration of hepatic synthetic dysfunction. In patients with higher Child-Pugh classes or MELD scores, reduced hepatic CRP synthesis may weaken the relationship between intestinal mucosal abnormalities and circulating hsCRP. Conversely, relatively preserved hepatic function may permit a stronger acute-phase response. Thus, the observed correlation may

vary across cirrhosis severity categories, and its consistency should be examined in future studies using stratified or adjusted analyses. Furthermore, the diagnostic and prognostic utility of combining fecal calprotectin and hsCRP requires validation before routine clinical implementation

Correlation between Total Fecal SCFA and hsCRP

In contrast, total fecal SCFA did not correlate with hsCRP ($r = 0.013$; $p = 0.944$). Theoretically, SCFA exert anti-inflammatory effects through HDAC inhibition, GPCR activation, induction of regulatory T cells, and reinforcement of the intestinal barrier (Deleu et al., 2021; He et al., 2020), so lower SCFA would be expected to accompany higher systemic inflammation. However, most SCFA produced in the colon are rapidly utilized by colonocytes or metabolized by the liver before reaching the peripheral circulation; therefore, fecal SCFA concentrations reflect the local balance between production and absorption rather than systemic bioactivity (Blaak et al., 2020). Moreover, systemic inflammation in cirrhosis is multifactorial, being driven by bacterial translocation, endotoxemia, innate immune activation, hepatocyte dysfunction, and the severity of portal hypertension (Arab et al., 2018). Fecal SCFA are also strongly influenced by dietary fiber intake, medications, and gut microbiota composition (Vinelli et al., 2022), which were not fully controlled in this study.

Another possible explanation for the absence of correlation is the influence of liver disease severity on hsCRP production. In advanced cirrhosis, reduced hepatic synthetic capacity may blunt hsCRP responses despite ongoing intestinal or systemic inflammatory processes. Differences in Child-Pugh class and MELD score could therefore modify the relationship between fecal SCFA and hsCRP. Without analyses accounting for these severity indicators, the absence of a statistically significant correlation cannot exclude a biological association between SCFA metabolism and systemic inflammation. Moreover, fecal SCFA concentrations cannot be used to infer circulating SCFA exposure, particularly when intestinal absorption and hepatic metabolism may be altered

Correlation between Total Fecal SCFA and Fecal Calprotectin

Total fecal SCFA also showed no significant correlation with fecal calprotectin ($r = 0.106$; $p = 0.562$); thus, the hypothesized negative correlation was not supported. In cirrhosis, intestinal mucosal inflammation is chronic and multifactorial—driven by portal hypertensive mucosal congestion, dysbiosis, small intestinal bacterial overgrowth, and bacterial translocation—so fecal calprotectin is likely determined more by these structural and hemodynamic processes than by microbial metabolites alone (Arab et al., 2018). Portal hypertensive enteropathy may further complicate this relationship because vascular congestion, mucosal edema, and increased permeability can contribute to epithelial injury and secondary inflammatory responses without classical inflammatory intestinal disease. Consequently, fecal calprotectin elevation may reflect portal hypertension-related mucosal abnormalities rather than changes in microbial SCFA production alone.

In addition, because fecal SCFA represent the net result of microbial production and colonocyte absorption, higher fecal SCFA may even reflect impaired colonic absorption in an inflamed mucosa. Tews et al. (2023) found no significant correlation between fecal acetate, propionate, butyrate, or isobutyrate and fecal calprotectin in 64 patients with inflammatory bowel disease, and Łoniewski et al. (2022) reported no consistent relationship between fecal SCFA and gut barrier markers in 56 postmenopausal

women with obesity. These findings suggest that the relationship between microbial metabolites and intestinal inflammation is complex and possibly non-linear.

Medications should also be considered. Non-steroidal anti-inflammatory drugs and proton pump inhibitors can increase fecal calprotectin (Sandberg-Janzon & Karling, 2025), whereas antibiotics, probiotics, and lactulose alter the composition and fermentative activity of the gut microbiota and thus fecal SCFA (Bajaj et al., 2018). Although non-steroidal anti-inflammatory drugs, steroids, and probiotics were excluded, lactulose, rifaximin, non-selective beta-blockers, and diuretics commonly used in cirrhosis were not, and may have acted as residual confounders.

Additionally, the severity of portal hypertension and the presence of portal hypertensive enteropathy were not directly assessed. These factors may independently influence intestinal permeability, mucosal injury, and fecal calprotectin concentrations, potentially obscuring associations with fecal SCFA. Future studies should incorporate Child-Pugh and MELD assessments, portal hypertension-related clinical parameters, and endoscopic evaluation when clinically indicated to clarify the contributions of hepatic dysfunction, mucosal vascular abnormalities, and microbial metabolism to the observed biomarker relationships

Strengths and Limitations

To our knowledge, this is one of the few studies, particularly in Indonesia, to assess fecal SCFA, fecal calprotectin, and hsCRP simultaneously in the same patients with cirrhosis, allowing a stepwise evaluation of the gut–liver axis from luminal microbial metabolites to intestinal and systemic inflammation. Nevertheless, several limitations should be noted. The cross-sectional design precludes causal or temporal inference. The absence of a healthy or non-cirrhotic comparison group narrowed the variability of the measured variables and may have increased the risk of type II error. Disease severity and etiology were not stratified; nutritional status was not specifically assessed, and body mass index was calculated from actual body weight without correction for ascites as recommended by the European Association for the Study of the Liver (2019). Other inflammatory markers (IL-6, TNF-α, lipopolysaccharide-binding protein) and gut microbiota composition were not measured, and the single-center setting limits generalizability.

Conclusion

Fecal calprotectin showed a significant moderate positive correlation with hsCRP in patients with liver cirrhosis ($r = 0.520$; $p = 0.002$), indicating an association between intestinal mucosal inflammation and systemic inflammation that is consistent with disruption of the gut–liver axis; however, the cross-sectional design does not allow causal inference. In contrast, total fecal SCFA showed very weak and non-significant correlations with both hsCRP ($r = 0.013$; $p = 0.944$) and fecal calprotectin ($r = 0.106$; $p = 0.562$), suggesting that total fecal SCFA alone is insufficient as a biomarker of intestinal or systemic inflammation in this population. Fecal calprotectin may be considered an additional marker of intestinal inflammatory involvement in cirrhosis, but larger longitudinal, multicenter studies with comparison groups, stratification by disease severity, and measurement of circulating SCFA and intestinal permeability markers are needed before it can be recommended for routine clinical practice.

Declaration of AI Assistance

The author used AI for tidying up the order of sentences, check typo, and paraphrase on this article

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