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RESEARCH

Potential role of dyslipidemia in hemorrhoidal disease: a prospective comparative study

Hemoroidal hastalıkta dislipideminin olası rolü: prospektif karşılaştırmalı bir çalışma

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Abstract

Purpose: This study aimed to investigate the potential role of dyslipidemia in the pathogenesis of hemorrhoidal disease (HD) and to explore the underlying mechanisms.
Materials and Methods: In this prospective study, 200 patients presenting with benign anorectal complaints between February-March 2025 were enrolled. One hundred patients diagnosed with hemorrhoidal disease (HD group) and 100 patients with other benign anorectal conditions (control group) underwent fasting lipid profile assessment. The HD group was classified according to Goligher's classification, and the relationship between hemorrhoidal grade and lipid levels was analyzed.
Results: Compared with the control group, the HD group exhibited significantly higher levels of LDL (123.7 ± 41.7 vs. 103.9 ± 48.4 mg/dL), triglycerides (173.0 ± 126.1 vs. 141.8 ± 74.5 mg/dL), and total cholesterol (198.2 ± 53.9 vs. 171.7 ± 60.3 mg/dL). HDL levels correlated inversely with hemorrhoidal grade, being highest in Grade I and lowest in Grade IV disease.
Conclusion: Our findings suggest an association between hemorrhoidal disease and dyslipidemia. Because this was an observational study, causality cannot be established; further studies are needed to determine whether lipid management affects the risk or severity of hemorrhoidal disease.
Keywords:. Hemorrhoidal disease, dyslipidemia, matrix metalloproteinases

Öz

Amaç: Bu çalışmanın amacı, dislipideminin hemoroidal hastalığın (HH) etiyolojisindeki olası rolünü ve altta yatan mekanizmayı araştırmaktır.
Gereç ve Yöntem: Bu prospektif çalışmaya, Şubat–Mart 2025 tarihleri arasında benign anorektal şikâyetlerle başvuran 200 hasta dâhil edildi. Hemoroidal hastalık tanısı alan 100 hasta (HH grubu) ile diğer benign anorektal durumları bulunan 100 hasta (kontrol grubu) lipid profili açısından değerlendirildi. HH grubu Goligher sınıflamasına göre derecelendirildi ve hemoroid derecesi ile lipid düzeyleri arasındaki ilişki analiz edildi.
Bulgular: Kontrol grubuyla karşılaştırıldığında HH grubunda LDL (123.7 ± 41.7'ye karşı 103.9 ± 48.4 mg/dL), trigliserit (173.0 ± 126.1'e karşı 141.8 ± 74.5 mg/dL) ve total kolesterol (198.2 ± 53.9'a karşı 171.7 ± 60.3 mg/dL) düzeyleri anlamlı olarak daha yüksek bulundu. HDL düzeyleri hemoroid derecesiyle ters ilişki gösterdi; en yüksek değerler Evre I, en düşük değerler Evre IV hastalıkta gözlendi.
Sonuç: Bulgularımız hemoroidal hastalığın dislipidemi ile ilişkili olduğunu göstermektedir. Çalışma gözlemsel nitelikte olduğundan nedensellik kurulamamaktadır; lipid yönetiminin hemoroidal hastalık riskini veya şiddetini etkileyip etkilemediğini belirlemek için ileri çalışmalara ihtiyaç vardır.
Anahtar kelimeler: Hemoroidal hastalık, dislipidemi, matriks metalloproteinazlar

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INTRODUCTION

Hemorrhoidal cushions are specialized vascular structures within the anal canal that contribute to continence and facilitate defecation. Hemorrhoidal disease (HD) develops when these cushions enlarge and descend distally, causing symptoms such as bleeding, prolapse, pruritus, pain, swelling, and mucous discharge[1-3]. Although prevalence estimates vary by population and methodology, HD remains one of the most common benign anorectal conditions encountered in clinical practice[1].

The prevalence of hemorrhoidal disease (HD) is estimated to range from 4.4% to 38% in the general population, with a higher incidence observed in men[1]. The etiology of hemorrhoidal disease is multifactorial, involving factors such as age, sex, dietary habits, constipation, genetic predisposition, and increased intra-abdominal pressure[2].

Clinically, internal hemorrhoids are commonly graded using Goligher's classification (I–IV) based on the degree of prolapse, which guides treatment selection and enables standardized comparisons across studies[3,4]. However, the clinical course is heterogeneous, and progression to higher grades is not fully explained by local mechanical factors alone.

Hemorrhoidal disease is currently regarded as a multifactorial disorder characterized by degeneration of the supporting connective tissue and smooth muscle framework, venous dilatation, chronic inflammation, and vascular remodeling[5]. Histopathological and immunohistochemical studies have demonstrated angiogenesis, structural alterations in the extracellular matrix (ECM), and remodeling of vascular and stromal components, supporting the 'sliding anal canal lining' concept of hemorrhoid development[5,6].

The reasons why some individuals develop clinically significant HD, or progress despite similar local exposures, remain unclear. Increasing evidence suggests that systemic metabolic and vascular risk factors—particularly adiposity, hypertension, impaired vascular homeostasis, and dyslipidemia—may influence the susceptibility of anorectal vascular cushions to injury and remodeling[7-9]. Dyslipidemia may contribute to endothelial dysfunction and chronic low-grade inflammation, potentially predisposing to vascular dilation and altered tissue perfusion within the hemorrhoidal plexus[7].

One plausible biological mechanism linking dyslipidemia to hemorrhoidal remodeling involves dysregulated extracellular matrix turnover mediated by matrix metalloproteinases (MMPs), particularly MMP-2 and MMP-9. These zinc-dependent proteases degrade collagen and elastin, weakening structural support and facilitating tissue descent and prolapse. Elevated tissue immunoreactivity of MMPs has been reported in hemorrhoidal disease and correlates with disease stage[10]. In parallel, clinical studies in metabolic and vascular disorders have linked dyslipidemia to increased circulating MMP activity, suggesting a systemic pro-remodeling milieu[11].

HDL may also be relevant to disease severity because of its vasculoprotective and anti-inflammatory properties, and reduced HDL can impair endothelial resilience and repair[12]. Despite these mechanistic links, clinical evidence directly connecting dyslipidemia to both the presence and severity of HD remains limited. Therefore, the primary objective of this study was to compare fasting lipid profiles between patients with HD and controls with other benign anorectal conditions. Secondary objectives were to assess the association between lipid parameters and Goligher grade and to identify independent predictors of HD. We hypothesized that patients with HD would have a more atherogenic lipid profile (higher LDL, triglycerides, and total cholesterol and/or lower HDL) and that adverse lipid parameters—particularly lower HDL—would be associated with higher Goligher grades.

To our knowledge, this is one of the few prospective studies to evaluate the association between dyslipidemia and hemorrhoidal disease using a clinically relevant control group of patients with other benign anorectal conditions, rather than healthy volunteers. The originality of our study lies in relating specific lipid parameters, particularly HDL, to disease severity as graded by Goligher's classification, thereby positioning dyslipidemia as a potentially modifiable systemic contributor to hemorrhoidal progression. In doing so, our findings extend current concepts of HD beyond local mechanical factors and provide a rationale for future studies on metabolic risk modification.

MATERIALS AND METHODS

This prospective study was conducted in the General Surgery Outpatient Clinic of Çukurova University

Faculty of Medicine (Adana, Türkiye), a tertiary-care university hospital. Between February and March 2025, consecutive adult patients presenting with benign anorectal complaints were prospectively evaluated. The study was conducted in accordance with the Declaration of Helsinki and was approved by the Çukurova University Faculty of Medicine Ethics Committee on February 7, 2025 (Decision No: 152/40). Written informed consent was obtained from all participants before enrollment.

Sample

A total of 200 patients were enrolled and categorized into two groups: First group was Hemorrhoidal Disease (HD) Group (n=100): Patients diagnosed with HD through physical examination and anoscopic evaluation included in this group. Control Group (n=100) included patients diagnosed with other benign anorectal conditions, such as anal fissures or perianal pruritus, following the same clinical assessment. Patients were selected using a consecutive sampling method to ensure a representative study sample.

Inclusion criteria were: age ≥18 years, presentation with benign anorectal complaints, and confirmed benign anorectal findings on clinical assessment (internal hemorrhoids on anoscopy for the HD group, or another benign anorectal condition such as anal fissure or perianal pruritus for the control group). All included patients provided written informed consent.

Exclusion criteria, defined to minimize confounding of lipid measurements and to exclude conditions requiring alternative diagnostic pathways, were: a history of previous anorectal surgery; anorectal malignancy; current use of lipid-lowering medications (statins or fibrates); pregnancy; systemic inflammatory disease; and incomplete clinical or laboratory data.

During the study period, 262 consecutive patients with benign anorectal complaints were assessed for eligibility. Of these, 62 were excluded for the following reasons: previous anorectal surgery (n = 19), anorectal malignancy (n = 6), current lipid-lowering medication use (n = 20), pregnancy (n = 3), systemic inflammatory disease (n = 4), and incomplete data or declined consent (n = 10). The remaining 200 eligible patients were enrolled and allocated to the HD group (n = 100) or the control group (n = 100). The patient selection process is summarized in Figure 1.

Procedure

Demographic data (age, sex), clinical characteristics, and body mass index (BMI) were recorded for all participants. A standardized anorectal evaluation was performed, including perianal inspection, digital rectal examination, and anoscopy. HD was diagnosed when internal hemorrhoidal cushions were identified on anoscopy and graded according to Goligher's classification (I–IV), whereas control patients were diagnosed with other benign anorectal conditions (e.g., anal fissure, perianal pruritus) based on the same assessment. Dyslipidemia was defined and lipid values were interpreted according to the 2021 Dyslipidemia Diagnosis and Treatment Guideline of the Turkish Endocrinology and Metabolism Association[13].

Clinical and biochemical assessment

All anorectal examinations and anoscopic Goligher gradings were performed by general surgeons following a standardized institutional protocol. Venous blood samples were drawn by trained nurses after a 12-hour overnight fast and processed on the same day in the hospital's accredited central biochemistry laboratory by qualified laboratory technicians, using fully automated analyzers with routine internal quality-control procedures. This standardized, single-center workflow with a single laboratory minimized inter-operator and inter-assay variability and supports the reliability of the reported data. The presence and severity of hemorrhoidal disease (HD) were categorized according to the widely accepted Goligher classification as follows: Grade I (no prolapse), Grade II (spontaneously reducible prolapse), Grade III (prolapse requiring manual reduction), and Grade IV (irreducible prolapse).

For biochemical assessment, serum levels of low-density lipoprotein (LDL), high-density lipoprotein (HDL), triglycerides, and total cholesterol were measured. In accordance with the 2021 Dyslipidemia Diagnosis and Treatment Guideline of the Turkish Society of Endocrinology and Metabolism (TEMD), dyslipidemia was defined as the presence of one or more of the following abnormalities: total cholesterol ≥200 mg/dL, LDL cholesterol ≥130 mg/dL, triglycerides ≥150 mg/dL, or low HDL cholesterol (<40 mg/dL in men and <50 mg/dL in women).

Accordingly, values below these thresholds—total cholesterol <200 mg/dL, LDL <130 mg/dL, triglycerides <150 mg/dL, and HDL >40 mg/dL in men or >50 mg/dL in women—were regarded as within normal limits.

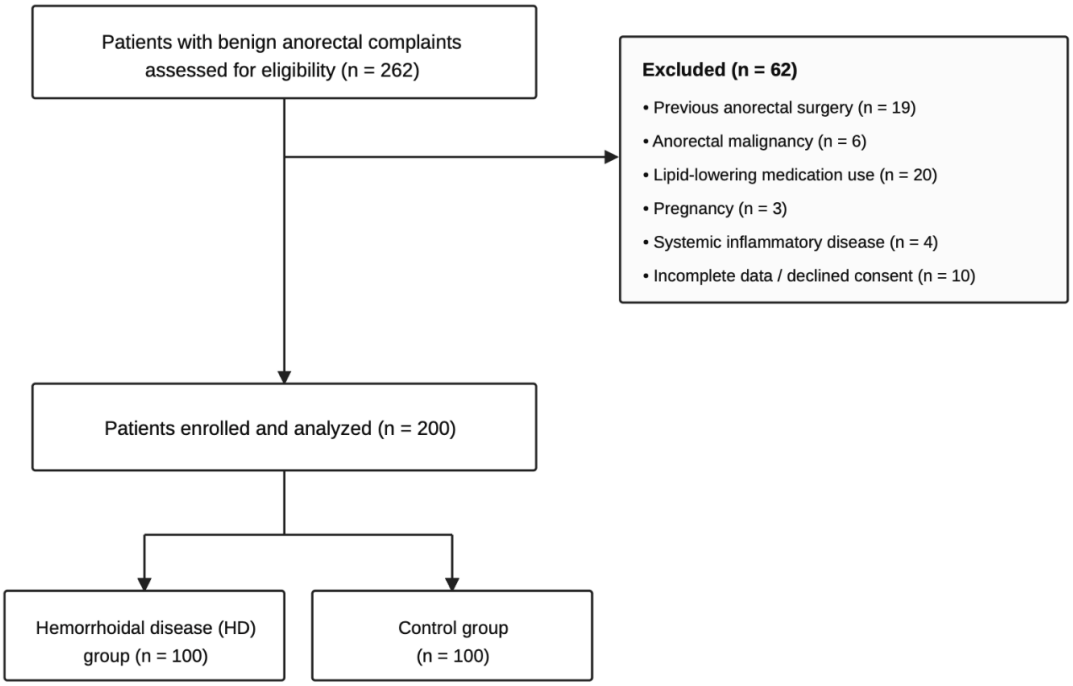


Figure 1. Study flow diagram.

Statistical analysis

Data analysis was performed using SPSS version 25. Continuous variables were presented as mean ± standard deviation (SD), and categorical variables as frequencies and percentages. Independent Student's t-tests and chi-square tests were used for group comparisons. Predictors of HD were identified via multivariate logistic regression analysis, reporting odds ratios (OR) and 95% confidence intervals (CI). The correlation between lipid levels and hemorrhoidal grade was assessed using Spearman correlation analysis. A p-value < 0.05 was considered statistically significant.

Variables included in the model were selected a priori based on prior evidence linking them to the etiology of HD, namely age, sex, and total cholesterol. The Enter method was used, whereby all independent variables were entered into the model simultaneously. Model fit was assessed using the Hosmer–Lemeshow goodness-of-fit test, and potential multicollinearity among predictors was evaluated using variance inflation factor (VIF) values. Results are reported as odds ratios (OR) with 95% confidence intervals. Total cholesterol is biologically interrelated with its subfractions (total cholesterol ≈ LDL + HDL + VLDL). To avoid multicollinearity, LDL, HDL, and triglycerides were therefore not entered together with total cholesterol, which was retained a priori as the single summary lipid measure. An alternative model substituting LDL, HDL and triglycerides for total cholesterol was additionally fitted as a sensitivity analysis.

A post hoc power analysis was based on total cholesterol levels, the primary outcome of the study. The effect size (Cohen's d) for the difference between the HD and control groups was calculated as 0.46. With a total sample size of 200 patients (100

per group), the study achieved a statistical power of 87.4% at a significance level of alpha = 0.05. This exceeds the conventionally accepted threshold of 80%, confirming that the sample size was sufficient to detect clinically significant differences between the groups.

RESULTS

The mean age of all patients was 48.6 ± 16.2 years, with a sex distribution of 120 males (60%) and 80 females (40%). The HD group was significantly younger than the control group (43.9 ± 14.9 vs. 53.3 ± 16.2 years, p = 0.001) and exhibited a higher proportion of males (69% vs. 51%, p = 0.009). There was no significant difference in BMI between the groups. (Table 1)

Lipid profiling revealed that the HD group had significantly elevated levels of LDL (123.7 ± 41.7 vs. 103.9 ± 48.4, p = 0.002), triglycerides (173.0 ± 126.1 vs. 141.8 ± 74.5, p = 0.035), and total cholesterol (198.2 ± 53.9 vs. 171.7 ± 60.3, p = 0.001) compared with the control group. Subgroup analysis based on Goligher's grading demonstrated that Grade III hemorrhoids were the most common (36%). (Table 2)

HDL levels were significantly higher in Grade I patients compared with those in higher grades (p = 0.030). Although the highest triglyceride levels were observed in Grade IV, this difference did not reach statistical significance (p = 0.584).

Multivariate logistic regression identified age (OR: 0.96, p < 0.001), male sex (OR: 2.38, p = 0.007), and total cholesterol (OR: 1.01, p < 0.001) as independent risk factors for hemorrhoidal disease. (Table 3)

When all lipid parameters were forced into a single model, total cholesterol and LDL showed variance inflation factors exceeding 10 and no lipid parameter remained statistically significant, confirming substantial multicollinearity. In the alternative model substituting LDL, HDL and triglycerides for total cholesterol, LDL was independently associated with HD (OR 1.01, 95% CI 1.00–1.02, p = 0.035), whereas HDL and triglycerides were not; age and male sex remained significant, consistent with the primary analysis (Table 5).

Spearman correlation analysis revealed a significant negative correlation between HDL levels and hemorrhoidal grade (r = -0.299, p = 0.003). (Table 4) Because HDL cut-offs are sex-specific and higher grades were male-predominant, we tested whether the HDL–grade relationship was independent of sex. Using sex-specific cut-offs (HDL <40 mg/dL in men, <50 mg/dL in women), low HDL did not differ between the HD and control groups and did not predict HD presence (adjusted OR 1.46, 95% CI 0.76–2.79, p = 0.26). Within the HD group, however, the inverse HDL–grade association persisted after adjustment for sex (−3.0 mg/dL per grade; p = 0.012). Low HDL was also independently associated with high-grade disease (Grade III–IV vs. I–II; OR, 2.48; 95% CI, 1.07–5.76; p = 0.034; Table 6). This association was significant among men (r = −0.28, p = 0.019) but not in the smaller female subgroup, in which only one patient had Grade IV disease.

Table 1. Comparison of demographic and laboratory characteristics between groups

Characteristic	Total (n=200)	Control (n=100)	Hemorrhoids (n=100)	p value
Age (years)	48.6 ± 16.2	53.3 ± 16.2	43.9 ± 14.9	< 0.001
Sex (Male/Female)	120/80	51/49	69/31	0.009
BMI (kg/m²)	26.5 ± 3.9	26.2 ± 2.6	26.7 ± 4.9	0.325
LDL (mg/dL)	113.8 ± 46.1	103.9 ± 48.4	123.7 ± 41.7	0.002
HDL (mg/dL)	42.5 ± 12.2	41.5 ± 12.9	43.4 ± 11.5	0.290
Triglycerides (mg/dL)	157.4 ± 104.5	141.8 ± 74.5	173.0 ± 126.1	0.035
Total Cholesterol (mg/dL)	184.9 ± 58.5	171.7 ± 60.3	198.2 ± 53.9	0.001

(BMI: Body Mass Index, LDL: Low-density lipoprotein, HDL: High-density lipoprotein, mg: milligram, dL: deciliter)

Table 2. Characteristics according to hemorrhoid grade (Goligher's classification)

Characteristic	Grade I (n=14)	Grade II (n=32)	Grade III (n=36)	Grade IV (n=18)	p value
Age (years)	42.8 ± 13.6	45.6 ± 15.5	43.6 ± 15.4	42.8 ± 15.3	0.899
Male/Female	10/4	16/16	26/10	17/1	0.011
BMI (kg/m²)	27.1 ± 6.5	25.7 ± 4.2	27.2 ± 3.7	27.5 ± 6.9	0.518
LDL (mg/dL)	132.0 ± 52.9	123.9 ± 33.2	122.1 ± 42.4	120.4 ± 47.0	0.954
HDL (mg/dL)	49.2 ± 11.5	45.8 ± 10.4	41.5 ± 12.5	38.6 ± 9.3	0.030*
Triglycerides (mg/dL)	136.6 ± 89.2	179.0 ± 145.0	168.2 ± 88.8	200.7 ± 173.3	0.584
Total Cholesterol (mg/dL)	198.6 ± 65.2	202.5 ± 49.8	197.4 ± 50.7	191.9 ± 61.4	0.953

(BMI: Body Mass Index, LDL: Low-density lipoprotein, HDL: High-density lipoprotein, mg: milligram, dL: deciliter)

The study sample was male-predominant overall (69% men), most markedly in Grade IV (17 men vs. 1 woman; 94% men). This may reflect the recognized male predominance of higher-grade, surgically managed hemorrhoidal disease. Because only one woman had Grade IV disease, sex-specific estimates within this subgroup are statistically unstable and should be interpreted with caution; to limit confounding by sex, the HDL–grade association was additionally assessed with adjustment for sex (see Results and Table 6).

Table 3. Multivariate logistic regression analysis for predictors of hemorrhoidal disease

Variable	Odds Ratio (95% CI)	p value
Age (years)	0.96 (0.94–0.97)	< 0.001
Male Sex	2.38 (1.27–4.47)	0.007
Total Cholesterol	1.01 (1.00–1.01)	< 0.001

Table 4. Spearman correlation analysis of clinical and laboratory variables with hemorrhoid grade

Variable	Correlation (r)	p value
Age	-0.052	0.608
BMI	0.085	0.399
LDL	-0.047	0.642
HDL	-0.299	0.003
Triglycerides	0.090	0.375
Total Cholesterol	-0.047	0.646

(BMI: Body Mass Index, LDL: Low-density lipoprotein, HDL: High-density lipoprotein)

Table 5. Alternative multivariate logistic regression model for predictors of hemorrhoidal disease, substituting LDL, HDL, and triglycerides for total cholesterol

Variable	Odds Ratio (95% CI)	p value
Age (years)	0.96 (0.94–0.98)	< 0.001
Male sex	2.32 (1.22–4.42)	0.011
LDL (mg/dL)	1.01 (1.00–1.02)	0.035
HDL (mg/dL)	1.00 (0.98–1.04)	0.763
Triglycerides (mg/dL)	1.00 (1.00–1.01)	0.117

(LDL: Low-density lipoprotein, HDL: High-density lipoprotein)

Table 6. Multivariate logistic regression analyses for predictors of hemorrhoidal disease (Panel A) and of high-grade disease within the HD group (Panel B)

Panel A. Predictors of Hemorrhoidal Disease (HD) (vs. Control)

Variable	Odds Ratio (95% CI)	p value
Age (years)	0.96 (0.94–0.98)	< 0.001
Male sex	2.50 (1.32–4.72)	0.005
Total cholesterol (mg/dL)	1.01 (1.00–1.02)	< 0.001
Low HDL (sex-specific)*	1.46 (0.76–2.79)	0.26

Panel B. Predictors of High-Grade Disease (Grade III–IV vs. Grade I–II) Within the HD Group

Variable	Odds Ratio (95% CI)	p value
Low HDL (sex-specific)*	2.48 (1.07–5.76)	0.034
Male sex	3.30 (1.32–8.24)	0.010

*Low HDL defined as <40 mg/dL in men and <50 mg/dL in women. Panel A: n = 200. Panel B: n = 100 (54 high-grade, 46 low-grade). OR, odds ratio; CI, confidence interval.

DISCUSSION

In this prospective study of patients presenting with benign anorectal complaints, we observed a more atherogenic lipid profile in the HD group, with significantly higher LDL, triglycerides, and total cholesterol compared with controls. Importantly, total cholesterol remained independently associated with HD after multivariable adjustment, and HDL showed an inverse correlation with Goligher grade. Together, these findings support the concept that HD is not exclusively a local mechanical disorder, but may be associated with systemic metabolic factors. Our study adds to the literature by evaluating dyslipidemia in a clinically relevant setting with a control group of other benign anorectal conditions and by relating lipid parameters to disease grade.

The role of metabolic and vascular risk factors in HD has gained attention in recent years. Population-based analyses have reported associations between HD and factors such as adiposity, hypertension, constipation, and smoking, and genetic epidemiology has implicated adiposity in hemorrhoid development8,9. These observations reinforce the plausibility that systemic cardiometabolic traits can influence anorectal vascular cushions and contribute to the development of clinically significant hemorrhoids.

Our findings are consistent with emerging clinical evidence linking abnormal lipid metabolism to hemorrhoidal disease. In a recent study of patients with diabetes, Ciftel et al. reported that hemorrhoidal disease was associated with a worse lipid profile, including higher triglycerides and LDL14. While that work focused on a diabetic population, our results suggest that dyslipidemia may also be relevant in a broader outpatient population with benign anorectal presentations, supporting dyslipidemia as a potentially modifiable factor associated with hemorrhoidal disease.

Mechanistically, dyslipidemia may promote endothelial dysfunction and chronic low-grade inflammation, which can impair venous tone, microvascular perfusion, and tissue repair7. At the anorectal level, these systemic vascular effects may accelerate the degenerative and remodeling processes described in HD, including angiogenesis, vascular remodeling, and connective tissue weakening5,6. Recent molecular syntheses emphasize that hemorrhoids are characterized by complex pathways involving inflammation, angiogenesis, extracellular matrix turnover, and genetic susceptibility15.

A key biological bridge between dyslipidemia and hemorrhoidal progression may be dysregulated extracellular matrix (ECM) turnover. Matrix metalloproteinases, particularly MMP-2 and MMP-9, degrade structural ECM components and have been implicated in the pathophysiology and staging of HD10. In metabolic and vascular conditions, dyslipidemia has been associated with increased MMP activity, providing a systemic pro-remodeling milieu that could plausibly weaken the supportive framework of the anal cushions and facilitate descent and prolapse11. Although we did not measure tissue

biomarkers, our clinical findings are compatible with this mechanistic framework.

The inverse association between HDL and hemorrhoid grade is clinically notable. HDL has anti-inflammatory and endothelial-protective effects and supports vascular homeostasis[12]. Lower HDL could therefore reduce resilience to venous and stromal injury, potentially contributing to more advanced Goligher grades. This relationship may help explain why some patients progress despite comparable local risk exposures and highlights HDL as a candidate marker for severity stratification.

We also observed that younger age and male sex were independently associated with HD. These findings may reflect differences in health-seeking behavior, occupational or lifestyle factors (e.g., prolonged sitting, diet), or unmeasured cardiometabolic traits that cluster in younger men. Notably, cardiometabolic risk can begin early, and integrating lifestyle counseling in this group may offer benefits beyond anorectal symptom control.

Strengths of this study include its prospective design, consecutive sampling strategy, standardized anoscopic assessment, and inclusion of a clinically relevant control group with other benign anorectal diagnoses. The sample size provided adequate power to detect between-group differences in lipid parameters.

Several limitations should be considered. First, this was a single-center study with a relatively short, consecutive enrollment window (February–March 2025). This design may limit generalizability and could introduce temporal or referral-pattern bias. Although the target sample size of 200 patients was reached and the study was adequately powered for the primary lipid comparison (post hoc power, 87.4%), larger multicenter cohorts enrolling over longer periods are needed to confirm these findings. Second, we did not directly quantify tissue biomarkers (e.g., MMP expression), inflammatory mediators, or measures of endothelial function. Third, data on several established risk factors for hemorrhoidal disease that are also associated with lipid metabolism—including constipation severity, dietary intake, physical activity, and smoking—were not collected, nor was detailed information on medication use beyond lipid-lowering therapy. Because these factors may influence both lipid levels and hemorrhoidal disease, the possibility of residual confounding cannot be excluded; this is an important limitation, and the reported associations should be interpreted accordingly. Finally, the study design does not establish causality.

Clinically, our findings suggest that lipid profiling may be informative in patients with HD both to identify cardiovascular risk and to explore a potentially modifiable contributor to hemorrhoidal progression. Taken together, this prospective study demonstrates that patients with hemorrhoidal disease have significantly higher LDL, triglyceride, and total cholesterol levels than controls with other benign anorectal conditions; after adjustment for relevant covariates, total cholesterol remained independently associated with HD, and HDL showed an inverse association with Goligher grade.

These findings support the hypothesis that systemic metabolic factors, particularly dyslipidemia, may contribute to hemorrhoidal pathogenesis and severity, potentially through endothelial dysfunction and extracellular matrix remodeling. Incorporating cardiometabolic assessment (including lipid profiling and lifestyle counseling) into the clinical evaluation of HD may provide dual benefits: identification of cardiovascular risk and a potential avenue to mitigate disease progression. Future multicenter, longitudinal, and interventional studies incorporating detailed lifestyle data and molecular endpoints (including ECM and angiogenic markers) are warranted to clarify causality and to determine whether optimizing lipid parameters can reduce symptom burden, progression, or post-treatment recurrence.

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