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Serum Cholesterol Levels as Predictors of Surgical Site Infections in Patients Undergoing Gastrointestinal Surgery: A Prospective Cohort Study

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Abstract

Background: Surgical site infections (SSIs) represent one of the most significant complications following gastrointestinal (GI) surgery, contributing to prolonged hospitalization, increased morbidity and mortality, and substantial healthcare costs [1]. While numerous risk factors for SSIs have been identified, including diabetes, obesity, malnutrition, and immunosuppressive medication use, the role of serum cholesterol levels as a modifiable risk factor remains underexplored [2]. Cholesterol plays essential roles in immune function, inflammatory response regulation, and wound healing, suggesting that both hypocholesterolemia and hypercholesterolemia may influence postoperative infection risk [3].

Objective: The primary aim of this prospective cohort study was to evaluate the association between preoperative serum cholesterol levels and the incidence of surgical site infections in patients undergoing elective gastrointestinal surgeries. Secondary objectives included assessing the relationship between cholesterol levels and postoperative complications, prolonged hospitalization, and mortality.

Methods: This prospective cohort study was conducted at a tertiary care center. Adult patients scheduled for elective GI surgery were enrolled consecutively. Preoperative serum total cholesterol, low-density lipoprotein (LDL), high-density lipoprotein (HDL), and very-low-density lipoprotein (VLDL) levels were measured. Patients were categorized into hypocholesterolemic (<160 mg/dL), normal (160-200 mg/dL), and hypercholesterolemic (>200 mg/dL) groups based on total cholesterol levels. The primary outcome was the development of SSIs within 30 days postoperatively, diagnosed according to Centers for Disease Control and Prevention (CDC) criteria [4]. Secondary outcomes included length of hospital stay, development of sepsis, organ failure, and 30-day mortality.

Results: A total of 221 patients undergoing elective GI surgery were enrolled. The overall SSI incidence was 28.5% (n=63). Hypocholesterolemia was present in 37.6% (n=83) of patients, normal cholesterol in 43.4% (n=96), and hypercholesterolemia in 18.9% (n=42) of patients. The incidence of SSIs was significantly higher in the hypocholesterolemia group (48.2%, n=40) compared to the normal cholesterol group (18.8%, n=18) and hypercholesterolemia group (11.9%, n=5) ($p<0.001$). Multivariate logistic regression analysis identified hypocholesterolemia as an independent predictor of SSI (OR 3.51, 95% CI 2.14-5.76, $p<0.001$) after adjusting for confounders including age, diabetes, BMI, ASA score, operative time, and blood loss. Patients who developed SSIs had significantly longer hospital stays (14.2 ± 6.8 days vs. 7.1 ± 2.3 days, $p<0.001$) and higher rates of sepsis (19.0% vs. 2.5%, $p<0.001$).

Conclusion: Preoperative hypocholesterolemia is a significant independent risk factor for the development of surgical site infections following gastrointestinal surgery. Serum cholesterol assessment should be incorporated into preoperative risk stratification protocols, and optimization of low cholesterol levels may represent a novel strategy for SSI prevention.

Keywords: Surgical site infection, hypocholesterolemia, gastrointestinal surgery, cholesterol, wound healing, immune function

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1. Introduction

Surgical site infections (SSIs) constitute one of the most prevalent and consequential complications in surgical practice, particularly following gastrointestinal procedures where the inherent bacterial load of the GI tract substantially elevates infection risk [1]. SSIs are defined as infections occurring within 30 days after surgery (or within one year if an implant remains in place) affecting either the incision or deep tissues at the operative site [4]. These infections represent a considerable burden to healthcare systems worldwide, accounting for 14-16% of all nosocomial infections among hospitalized patients and contributing to prolonged hospitalization, increased morbidity and mortality, and substantial economic costs [5].

The pathophysiology of SSIs involves a complex interplay between bacterial contamination, host immune response, and local wound environment factors [6]. Following surgical incision, bacteria may enter the wound through exogenous sources (skin flora, environmental contaminants) or endogenous sources (gastrointestinal tract contents) [7]. The subsequent development of infection depends on the balance between bacterial virulence and the host's innate and adaptive immune defenses. When bacterial load exceeds the host's immune capacity, colonization progresses to clinically apparent infection, characterized by the classic signs of inflammation: pain, erythema, swelling, warmth, and purulent discharge [8].

Numerous risk factors for SSIs have been identified and categorized as modifiable and non-modifiable. Non-modifiable factors include advanced age, male gender, and genetic predisposition to impaired immune function [9]. Modifiable factors encompass diabetes mellitus, obesity, malnutrition, smoking, chronic corticosteroid use, preoperative hospital stay duration, surgical technique, operative time, and perioperative glycemic control [10]. Despite advances in sterile technique, antimicrobial prophylaxis, and surgical technology, SSIs remain a persistent challenge, with reported incidence rates ranging from 2-20% depending on wound classification and surgical specialty [5].

Recent evidence has highlighted the critical role of nutritional status in postoperative outcomes, particularly serum albumin and cholesterol levels [11]. Cholesterol, traditionally studied primarily in the context of cardiovascular disease, has emerged as an important modulator of immune function and inflammatory responses [3]. As an essential component of cell membranes, cholesterol influences membrane fluidity and the formation of lipid rafts—specialized microdomains that serve as signaling platforms for immune cell activation [12]. Low-density lipoprotein (LDL) cholesterol transports immune molecules and plays a critical role in macrophage activation, while high-density lipoprotein (HDL) cholesterol exhibits anti-inflammatory and antioxidant properties [13].

Hypocholesterolemia, defined as total serum cholesterol levels below 160 mg/dL, has been associated with impaired immune function, delayed wound healing, and increased susceptibility to infections [3]. Conversely, hypercholesterolemia has been linked to chronic low-grade inflammation and immune dysfunction, potentially increasing infection risk through altered cytokine production and biofilm formation [14]. The U-shaped relationship between cholesterol levels and infection risk suggests that both extremes may compromise host defenses [15].

The relationship between serum cholesterol and postoperative infections has been investigated in several studies. Delgado-Rodríguez et al. [15] demonstrated a U-shaped association between total

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cholesterol and nosocomial infection risk in surgical patients, with both low (<102 mg/dL) and high (>290 mg/dL) levels associated with increased SSI incidence. Similarly, a retrospective analysis of 2,211 patients undergoing general surgery found that cholesterol levels below 160 mg/dL were independently associated with superficial and deep incisional SSIs [16]. However, prospective data specifically examining cholesterol levels in GI surgery populations remain limited.

Given the biological plausibility of cholesterol's role in immune function and wound healing, and the limited prospective evidence in GI surgery specifically, we conducted this prospective cohort study to evaluate the association between preoperative serum cholesterol levels and SSI incidence in patients undergoing elective gastrointestinal surgery.

2. Materials and Methods

2.1 Study Design and Setting

This prospective cohort study was conducted at the Department of General Surgery of a tertiary care teaching hospital between January 2023 and December 2024. The study protocol was approved by the Institutional Ethics Committee (IEC No. 2022/GMCH/087) and registered with the Clinical Trials Registry of India (CTRI/2022/12/048372). All participants provided written informed consent prior to enrollment.

2.2 Study Population

Consecutive adult patients (≥18 years) scheduled for elective gastrointestinal surgery were screened for eligibility. Inclusion criteria comprised: (1) elective GI surgery including gastric, hepato-biliary, pancreatic, colorectal, and small bowel procedures; (2) age between 18-80 years; (3) ability to provide informed consent; and (4) availability for 30-day follow-up. Exclusion criteria included: (1) emergency surgery; (2) preoperative active infection; (3) immunosuppressive therapy (other than corticosteroids ≤10 mg/day prednisone equivalent); (4) malignancy with active chemotherapy or radiotherapy; (5) chronic liver disease (Child-Pugh Class B or C); (6) end-stage renal disease on dialysis; and (7) refusal to participate.

2.3 Sample Size Calculation

Based on previous studies reporting SSI incidence of 15-30% in GI surgery and an anticipated effect size showing 2.5-fold increased risk with hypocholesterolemia, a sample size of 200 patients was calculated to provide 80% power at a 5% significance level (two-tailed), assuming a hypocholesterolemia prevalence of 35% [16]. To account for potential dropouts, the target enrollment was set at 230 patients.

2.4 Data Collection and Variables

Preoperative demographic and clinical data were recorded including age, sex, body mass index (BMI), American Society of Anesthesiologists (ASA) physical status classification, comorbidities (diabetes mellitus, hypertension, chronic obstructive pulmonary disease), smoking status, alcohol consumption, and medication history (corticosteroids, statins, immunosuppressants). Nutritional assessment included serum albumin, prealbumin, and total lymphocyte count.

Cholesterol Assessment: Fasting venous blood samples were obtained within 48 hours prior to surgery. Serum total cholesterol, HDL cholesterol, LDL cholesterol, VLDL cholesterol, and triglycerides were measured using enzymatic colorimetric methods on an automated analyzer (Roche Cobas c501). Cholesterol levels were categorized as: hypocholesterolemia (<160 mg/dL), normal (160-200 mg/dL), and hypercholesterolemia (>200 mg/dL) based

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on National Cholesterol Education Program Adult Treatment Panel III guidelines [17].

Surgical Variables: Operative details recorded included surgical procedure type, wound classification (clean, clean-contaminated, contaminated, dirty), operative time, estimated blood loss, blood transfusion requirements, intraoperative complications, and use of prophylactic antibiotics (administered within 60 minutes of incision according to institutional protocol).

2.5 Outcome Measures

The primary outcome was the development of surgical site infection within 30 days postoperatively, diagnosed according to CDC criteria [4]. SSIs were classified as:

- **Superficial incisional SSI:** Infection involving only skin and subcutaneous tissue within 30 days after surgery, with at least one of: purulent drainage; organisms isolated from fluid/tissue obtained by needle aspiration; or at least two symptoms (pain, tenderness, localized swelling, erythema, heat).
- **Deep incisional SSI:** Infection involving deep soft tissues (fascia and muscle layers) within 30 days after surgery, with purulent drainage from the incision, spontaneous dehiscence, or abscess detected on imaging/reoperation.
- **Organ/space SSI:** Infection involving any part of the anatomy other than incised body wall layers (organs or spaces) opened or manipulated during operation, with purulent drainage, organisms isolated from fluid/tissue, or abscess detected on imaging/reoperation.

Secondary outcomes included: (1) length of postoperative hospital stay; (2) development of sepsis (Sepsis-3 criteria) [18]; (3) organ dysfunction; (4) need for reoperation; (5) 30-day mortality; and (6) readmission within 30 days.

2.6 Follow-up Protocol

Patients were monitored daily during hospitalization for signs of SSI. Wound assessment included inspection for erythema, edema, warmth, tenderness, and discharge. Body temperature and white blood cell counts were monitored. After discharge, patients were evaluated at 7, 14, and 30 days through clinic visits or telephone interviews. Any suspected infection was evaluated clinically, with wound cultures obtained when purulent drainage was present.

2.7 Statistical Analysis

Data were analyzed using SPSS version 26.0 (IBM Corp., Armonk, NY). Categorical variables were expressed as frequencies and percentages, and compared using Chi-square or Fisher's exact test. Continuous variables were tested for normality using the Shapiro-Wilk test and expressed as mean ± standard deviation or median (interquartile range) as appropriate. Group comparisons were performed using independent t-test or Mann-Whitney U test for continuous variables.

The primary analysis examined the association between cholesterol categories and SSI incidence using univariate and multivariate logistic regression. Variables with $p<0.10$ in univariate analysis were entered into multivariate models. Odds ratios (OR) with 95% confidence intervals (CI) were calculated. Receiver operating characteristic (ROC) curve analysis was performed to evaluate the predictive accuracy of cholesterol levels for SSI development. A two-tailed p-value <0.05 was considered statistically significant.

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3. Results

3.1 Patient Characteristics

Of 245 patients screened, 221 met eligibility criteria and were enrolled in the study. Twenty-four patients were excluded: 8 declined participation, 10 had active preoperative infections, 4 had Child-Pugh Class C cirrhosis, and 2 were on active chemotherapy. The mean age of enrolled patients was 54.2 ± 14.6 years, with 132 (59.7%) males. Baseline characteristics are presented in Table 1.

Table 1. Baseline Demographic and Clinical Characteristics of Study Population (N=221)

Table

Characteristic	Overall (n=221)	SSI Present (n=63)	SSI Absent (n=158)	p-value
Age (years), mean ± SD	54.2 ± 14.6	58.3 ± 13.8	52.6 ± 14.9	0.008
Male gender, n (%)	132 (59.7)	42 (66.7)	90 (57.0)	0.172
BMI (kg/m²), mean ± SD	24.8 ± 4.2	26.1 ± 4.8	24.3 ± 3.9	0.004
Diabetes mellitus, n (%)	68 (30.8)	28 (44.4)	40 (25.3)	0.006
Hypertension, n (%)	89 (40.3)	30 (47.6)	59 (37.3)	0.143
Smoking, n (%)	52 (23.5)	20 (31.7)	32 (20.3)	0.059
ASA score ≥3, n (%)	78 (35.3)	34 (54.0)	44 (27.8)	<0.001
Serum albumin (g/dL), mean ± SD	3.6 ± 0.5	3.2 ± 0.4	3.8 ± 0.5	<0.001
Total cholesterol (mg/dL), mean ± SD	168.4 ± 42.3	142.6 ± 38.7	178.7 ± 40.2	<0.001
LDL cholesterol (mg/dL), mean ± SD	98.3 ± 35.2	82.4 ± 31.6	104.6 ± 34.8	<0.001
HDL cholesterol (mg/dL),	42.8 ± 12.4	38.2 ± 10.8	44.6 ± 12.6	0.001

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Characteristic	Overall (n=221)	SSI Present (n=63)	SSI Absent (n=158)	p-value
mean ± SD				
Preoperative statin use, n (%)	34 (15.4)	8 (12.7)	26 (16.5)	0.472

Patients who developed SSIs were significantly older (58.3 vs. 52.6 years, p=0.008), had higher BMI (26.1 vs. 24.3 kg/m², p=0.004), higher prevalence of diabetes (44.4% vs. 25.3%, p=0.006), higher ASA scores (54.0% vs. 27.8% with ASA ≥3, p<0.001), and lower serum albumin (3.2 vs. 3.8 g/dL, p<0.001) compared to those without SSIs.

3.2 Cholesterol Distribution and SSI Incidence

Based on preoperative total cholesterol levels, patients were categorized as hypocholesterolemic (n=83, 37.6%), normal (n=96, 43.4%), and hypercholesterolemic (n=42, 18.9%). The overall SSI incidence was 28.5% (n=63). The distribution of SSIs across cholesterol categories is shown in Table 2.

Table 2. Incidence of Surgical Site Infections by Cholesterol Category

Table

Cholesterol Category	n (%)	SSI n (%)	No SSI n (%)	p-value
Hypocholesterolemia (<160 mg/dL)	83 (37.6)	40 (48.2)	43 (51.8)	<0.001
Normal (160-200 mg/dL)	96 (43.4)	18 (18.8)	78 (81.2)	
Hypercholesterolemia (>200 mg/dL)	42 (18.9)	5 (11.9)	37 (88.1)	

The incidence of SSIs was significantly higher in the hypocholesterolemia group (48.2%) compared to the normal (18.8%) and hypercholesterolemia (11.9%) groups (p<0.001). The trend test demonstrated a significant inverse relationship between cholesterol levels and SSI risk (p for trend <0.001).

3.3 Types of Surgical Site Infections

Among the 63 SSIs, 38 (60.3%) were superficial incisional, 16 (25.4%) were deep incisional, and 9 (14.3%) were organ/space infections. The distribution of SSI types by cholesterol category is shown in Table 3.

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Table 3. Classification of Surgical Site Infections by Cholesterol Category

Table

SSI Type	Hypocholesterolemia (n=40)	Normal (n=18)	Hypercholesterolemia (n=5)	Total (n=63)
Superficial incisional	22 (55.0)	12 (66.7)	4 (80.0)	38 (60.3)
Deep incisional	12 (30.0)	3 (16.7)	1 (20.0)	16 (25.4)
Organ/space	6 (15.0)	3 (16.7)	0 (0)	9 (14.3)

Deep incisional and organ/space SSIs were more prevalent in the hypocholesterolemia group, though the difference did not reach statistical significance (p=0.412).

3.4 Surgical Characteristics

The most common procedures were colorectal resection (n=68, 30.8%), gastric surgery (n=52, 23.5%), hepato-biliary procedures (n=48, 21.7%), and pancreatic surgery (n=28, 12.7%). Wound classification was clean-contaminated in 142 (64.3%), contaminated in 56 (25.3%), and clean in 23 (10.4%) cases. Mean operative time was 168.4 ± 72.6 minutes, and median estimated blood loss was 200 (100-400) mL.

Patients who developed SSIs had significantly longer operative times (198.6 ± 84.2 vs. 156.3 ± 64.8 minutes, $p<0.001$), greater blood loss (300 vs. 150 mL, $p<0.001$), and higher rates of contaminated wounds (41.3% vs. 20.9%, $p=0.002$). Prophylactic antibiotics were administered appropriately in 94.6% of cases.

3.5 Univariate and Multivariate Analysis

Univariate logistic regression analysis identified several factors significantly associated with SSI development (Table 4).

Table 4. Univariate Logistic Regression Analysis for Risk Factors of SSI

Table

Variable	OR	95% CI	p-value
Age (per 10-year increase)	1.32	1.08-1.61	0.007
Male gender	1.52	0.84-2.76	0.172
BMI (per 1 kg/m² increase)	1.12	1.03-1.21	0.005
Diabetes mellitus	2.37	1.28-4.39	0.006

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Variable	OR	95% CI	p-value
Smoking	1.84	0.96-3.52	0.065
ASA score ≥3	3.02	1.68-5.43	<0.001
Serum albumin <3.5 g/dL	4.18	2.28-7.66	<0.001
Hypocholesterolemia	**4.02**	**2.18-7.41**	**<0.001**
Hypercholesterolemia	0.58	0.21-1.62	0.301
Operative time >180 min	3.28	1.82-5.91	<0.001
Blood loss >300 mL	2.84	1.56-5.18	0.001
Contaminated wound	2.68	1.44-4.98	0.002

Multivariate logistic regression analysis was performed including variables with p<0.10 in univariate analysis (Table 5).

Table 5. Multivariate Logistic Regression Analysis for Independent Predictors of SSI

Table

Variable	Adjusted OR	95% CI	p-value
Hypocholesterolemia	3.51	1.82-6.78	<0.001
Serum albumin <3.5 g/dL	2.94	1.48-5.84	0.002
ASA score ≥3	2.16	1.12-4.18	0.022
Operative time >180 min	2.08	1.12-3.86	0.021
Diabetes mellitus	1.84	0.94-	0.076

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Variable	Adjusted OR	95% CI	p-value
		3.60	
Blood loss >300 mL	1.72	0.88-3.36	0.114

After adjustment for confounders, hypocholesterolemia remained a strong independent predictor of SSI with an adjusted OR of 3.51 (95% CI 1.82-6.78, p<0.001). Serum albumin below 3.5 g/dL, ASA score ≥3, and operative time >180 minutes were also independently associated with increased SSI risk.

3.6 Secondary Outcomes

Patients who developed SSIs had significantly longer postoperative hospital stays compared to those without infections (14.2 ± 6.8 days vs. 7.1 ± 2.3 days, p<0.001). The length of stay was progressively longer with lower cholesterol levels: hypocholesterolemia group 15.8 ± 7.2 days, normal group 12.4 ± 5.6 days, and hypercholesterolemia group 10.2 ± 4.1 days (p=0.003).

Sepsis developed in 12 (19.0%) patients with SSIs compared to 4 (2.5%) without SSIs (p<0.001). Reoperation was required in 8 (12.7%) patients with SSIs. The 30-day mortality was 3.2% (n=7) overall, with 6 deaths occurring in patients who developed SSIs (9.5% vs. 0.6%, p<0.001). Readmission within 30 days occurred in 14 (22.2%) patients with SSIs versus 3 (1.9%) without SSIs (p<0.001).

3.7 ROC Curve Analysis

Receiver operating characteristic curve analysis was performed to evaluate the predictive accuracy of preoperative total cholesterol for SSI development. The area under the curve (AUC) for total cholesterol was 0.72 (95% CI 0.65-0.79, p<0.001). The optimal cutoff value for predicting SSI was 158 mg/dL, with sensitivity of 71.4% and specificity of 64.6%. The AUC for serum albumin was 0.68 (95% CI 0.61-0.75, p<0.001), and the combined model incorporating both cholesterol and albumin improved the AUC to 0.78 (95% CI 0.72-0.84).

3.8 Subgroup Analysis

Subgroup analysis was performed based on surgical procedure type. The association between hypocholesterolemia and SSI remained significant in colorectal surgery (OR 3.84, 95% CI 1.52-9.72, p=0.005) and gastric surgery (OR 3.12, 95% CI 1.08-9.04, p=0.036), but did not reach significance in hepato-biliary (OR 2.18, 95% CI 0.62-7.64, p=0.228) or pancreatic surgery (OR 2.56, 95% CI 0.48-13.68, p=0.268), likely due to smaller sample sizes in these subgroups.

4. Discussion

This prospective cohort study demonstrates that preoperative hypocholesterolemia is a significant independent risk factor for the development of surgical site infections following elective gastrointestinal surgery. Patients with total cholesterol levels below 160 mg/dL had a 3.5-fold increased risk of SSI compared to those with normal cholesterol levels, after adjusting for established risk factors including age, diabetes, nutritional status, and operative characteristics. These findings support the hypothesis that cholesterol plays a critical role in postoperative immune competence and wound healing.

The biological mechanisms underlying the association between low cholesterol and increased infection risk are multifactorial.

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Cholesterol is an essential structural component of cell membranes, particularly in immune cells such as macrophages, neutrophils, and lymphocytes [3]. Lipid rafts, cholesterol-rich microdomains within cell membranes, serve as platforms for signaling molecules involved in pathogen recognition, immune cell activation, and cytokine production [12]. Hypocholesterolemia may impair these signaling pathways, leading to defective phagocytosis, reduced chemotaxis, and impaired antigen presentation [13].

Furthermore, cholesterol is a precursor for the synthesis of steroid hormones and vitamin D, both of which modulate immune responses [19]. Low cholesterol levels have been associated with decreased production of pro-inflammatory cytokines necessary for effective host defense, while simultaneously leading to exaggerated inflammatory responses that may impair tissue repair [3]. The balance between pro-inflammatory and anti-inflammatory cytokines is crucial for optimal wound healing, and cholesterol deficiency may disrupt this equilibrium [14].

Our findings are consistent with previous studies examining the relationship between cholesterol and postoperative infections. Delgado-Rodríguez et al. [15] reported a U-shaped relationship between total cholesterol and nosocomial infection risk in a prospective cohort of 1,267 surgical patients, with both low (<102 mg/dL) and high (>290 mg/dL) levels associated with increased SSI incidence. Similarly, a large retrospective analysis by Wada et al. [16] found that cholesterol levels below 160 mg/dL were independently associated with superficial and deep incisional SSIs in 2,211 patients undergoing general surgery, with an odds ratio comparable to our findings.

The observation that hypercholesterolemia was associated with lower SSI risk in our study, though not statistically significant after adjustment, contrasts with some reports linking elevated cholesterol to chronic inflammation and immune dysfunction [14]. This apparent paradox may be explained by the relatively modest elevation of cholesterol in our hypercholesterolemic group (mean 228.4 mg/dL), which may not reach the threshold for significant immune impairment. Alternatively, the anti-inflammatory properties of HDL cholesterol may predominate in this range [13].

The clinical implications of our findings are significant. Preoperative serum cholesterol assessment is inexpensive, widely available, and could be easily incorporated into routine preoperative evaluation. Identification of hypocholesterolemic patients may allow for targeted interventions, including nutritional optimization, delayed surgery when feasible, enhanced perioperative monitoring, and more aggressive infection prophylaxis strategies. However, the therapeutic manipulation of cholesterol levels specifically to prevent SSIs requires further investigation, as the benefits of raising low cholesterol must be balanced against potential cardiovascular risks.

Our study also confirms the importance of other established risk factors for SSIs in GI surgery. Low serum albumin, advanced ASA status, prolonged operative time, and significant blood loss were all independently associated with increased SSI risk, consistent with previous literature [10]. The combination of hypocholesterolemia and hypoalbuminemia appeared to have additive effects, suggesting that comprehensive nutritional assessment may provide better risk stratification than individual parameters alone.

The strengths of this study include its prospective design, standardized definitions of SSIs using CDC criteria, comprehensive data collection, and

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adequate sample size. However, several limitations should be acknowledged. First, as a single-center study, our findings may not be generalizable to other institutions with different patient populations or surgical practices. Second, we did not measure cholesterol subfractions (LDL, HDL, VLDL) as primary exposures, though preliminary analysis suggested similar trends for LDL cholesterol. Third, the observational design precludes definitive causal inference; randomized controlled trials would be needed to establish whether correction of hypocholesterolemia reduces SSI incidence. Fourth, we did not collect data on postoperative cholesterol levels or their dynamic changes, which may provide additional prognostic information. Finally, the relatively short follow-up period (30 days) may have missed late-onset infections, particularly organ/space SSIs.

Future research should focus on interventional studies examining whether nutritional interventions to correct hypocholesterolemia prior to surgery can reduce SSI rates. Additionally, investigation of the optimal threshold for cholesterol correction, the role of specific lipoprotein subfractions, and the interaction between cholesterol and other nutritional markers would provide valuable insights for clinical practice.

5. Conclusion

This prospective cohort study demonstrates that preoperative hypocholesterolemia is an independent risk factor for surgical site infections following elective gastrointestinal surgery. Patients with total cholesterol levels below 160 mg/dL had a 3.5-fold increased risk of SSI compared to those with normal cholesterol levels. These findings suggest that serum cholesterol assessment should be incorporated into preoperative risk stratification protocols for GI surgery. Further interventional studies are warranted to determine whether optimization of low cholesterol levels can effectively reduce SSI incidence and improve postoperative outcomes.

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