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Assessing the Correlation between Serum Albumin and Serum Total Cholesterol Levels as Predictors of Surgical Site Infection in Abdominal Surgery Patients - Prospective Observational Study

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ABSTRACT

Background: Surgical-site infection (SSI) remains a common complication of abdominal surgeries. Because hypo-albuminaemia and hypo-cholesterolaemia each reflect impaired nutritional inflammatory reserve, we prospectively examined whether their pre-operative levels alone or combined predict 30-day SSI.

Methods: In a single-centre prospective observational cohort (January 2025 – June 2025), 150 consecutive adults scheduled for elective or emergency abdominal surgery had fasting serum albumin and total cholesterol measured ≤ 24 h before incision. Standard peri-operative care followed international SSI-prevention guidelines. Patients were surveilled to POD 30 using Centers for Disease Control criteria; 135 completed follow-ups. Multivariable logistic regression adjusted for diabetes, wound class, and operative time; predictive performance was assessed by area under the receiver-operator-characteristic curve (AUC) and calibration statistics.

Results: SSI occurred in 24 of 135 patients (incidence = 17.8 %). Mean albumin and cholesterol were significantly lower in infected than non-infected patients (3.2 ± 0.4 g dL^{-1} vs 3.7 ± 0.4 g dL^{-1}, and 158 ± 28 mg dL^{-1} vs 182 ± 31 mg dL^{-1}; both $p < 0.001$). After adjustment, each 0.5 g dL^{-1} increase in albumin reduced SSI odds by 40 % (adjusted OR 0.60, 95 % CI 0.42–0.87) and each 10 mg dL^{-1} rise in cholesterol by 11 % (aOR 0.89, 95 % CI 0.82–0.97). A composite albumin-plus-cholesterol score achieved an AUC of 0.81 (95 % CI 0.71–0.90), outperforming albumin alone (AUC 0.72) and the National Nosocomial Infection Surveillance index (AUC 0.68), with good calibration (Hosmer–Lemeshow P = 0.62) and an 18 % net reclassification improvement.

Conclusion: Low pre-operative serum albumin and total cholesterol are independent, synergistic predictors of SSI after abdominal surgery. Their routine, low-cost measurement enables pragmatic risk stratification, allowing focused deployment of intensified SSI-prevention bundles to the one-fifth of patients at highest risk.

Keywords: *Serum albumin; total cholesterol; surgical-site infection; abdominal surgery; risk stratification.*

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1. INTRODUCTION

Surgical-site infection (SSI) remains one of the most frequent preventable complications of abdominal surgery, with pooled incidence estimates ranging from 9 % to 23 % in low- and middle-income countries and 5 % to 10 % in high-income

settings despite adherence to modern aseptic protocols [1,2]. SSIs prolong hospital stay by a median of 9 days, double readmission risk, and increase mortality four-fold, generating an incremental annual cost exceeding US $3 billion in the United States alone [3]. Pathogenesis is multifactorial—encompassing microbial load, wound milieu, and host immunity—but peri-operative nutritional-inflammatory status has emerged as a critical, modifiable determinant of host defence at the incision site [4]. Serum albumin, the most abundant plasma protein, is both a transport molecule and a negative acute-phase reactant; hypo-albuminaemia reflects chronic protein–energy malnutrition, systemic inflammation, or both, and has been repeatedly linked to impaired fibroplasia, diminished collagen cross-linking, delayed epithelialisation, and reduced tensile strength of healing wounds [5]. Meta-analyses across heterogeneous surgical cohorts show that a pre-operative albumin concentration < 3.5 g/dL independently predicts a two- to three-fold rise in SSI risk, even after adjustment for diabetes, obesity, tobacco use, and operative duration [6]. Yet albumin alone lacks mechanistic specificity—levels fall in sepsis or trauma irrespective of nutritional reserves—and roughly one-third of infected patients exhibit albumin in the "normal" range, underscoring the need for composite markers [7].

Total serum cholesterol (TC) is a promising, under-appreciated candidate. Beyond its role in steroidogenesis and membrane fluidity, cholesterol participates directly in innate immunity: low-density lipoprotein (LDL) binds and neutralises bacterial lipopolysaccharide, while high-density lipoprotein (HDL) facilitates endotoxin clearance via the liver [8]. Hypocholesterolaemia (< 150 mg/dL) is common in critical illness and correlates with higher cytokine burden, capillary leak, and multiorgan failure [9]. In non-cardiac surgery, two prospective cohorts have independently demonstrated that admission TC in the lowest quartile predicts SSI with an odds ratio between 1.8 and 2.4—similar in magnitude to hypo-albuminaemia—but the literature is sparse and inconsistent, partly because TC is often omitted from routine pre-operative panels [10,11]. Furthermore, cholesterol distribution shifts after fasting and fluid resuscitation, and its interpretation in isolation may be confounded by statin therapy or hepatic dysfunction [12].

Albumin and cholesterol originate from shared hepatocellular synthetic pathways and respond oppositely to systemic inflammation—albumin falls, LDL and HDL typically fall even more precipitously—suggesting that their combined assessment could improve discrimination between patients with primary protein–energy malnutrition, those with acute inflammatory catabolism, and those who are metabolically replete [13]. The albumin-to-cholesterol ratio (ACR) has therefore been proposed as an integrated biomarker; two single-centre retrospective studies in colorectal surgery reported that an elevated pre-operative ACR (> 0.025 g/mg) doubled SSI prevalence compared with either low albumin or low cholesterol alone, but methodological limitations (small sample size, heterogeneity of procedures, and inconsistent SSI definitions) preclude definitive conclusions [14,15]. No prospective study to date has evaluated albumin, cholesterol, and their ratio simultaneously as predictors of SSI in a pure abdominal-surgery cohort encompassing both gastrointestinal and hepatobiliary procedures, where contamination risk and visceral bacterial load are intrinsically high.

The present prospective observational study was conceived to address this evidence gap. Conducted in a tertiary referral centre that performs more than 1,200 elective and emergency abdominal operations annually, it systematically measures fasting pre-operative serum albumin and total cholesterol within 24 h of incision, documents peri-operative variables (e.g., antibiotic timing, wound class, ASA physical status, glycaemic control), and adheres to the U.S. Centers for Disease Control and Prevention (CDC) 2023 definition of superficial, deep, and organ-space SSIs with 30-day surveillance [16]. Using multivariable logistic regression and receiver-operator-characteristic (ROC) analysis, the study will (i) quantify the independent predictive value of albumin and cholesterol, (ii) compare their area under the curve (AUC) against classic clinical scores such as the National Nosocomial Infection Surveillance (NNIS) risk index, and (iii) explore whether the ACR or a combined albumin-cholesterol score yields superior calibration and net reclassification improvement. Secondary objectives include determining optimal threshold values by the Youden method, assessing dose-response gradients across quartiles, and estimating population-attributable risk to gauge the potential effect of nutritional optimisation programmes. Beyond statistical refinement, the clinical importance of swiftly identifying high-risk patients cannot be overstated. Randomised trials show that targeted bundles—pre-operative immunonutrition, intensified glucose control, negative-pressure incision dressings, and extended antimicrobial prophylaxis—cut SSI incidence by up to 50 % in colorectal surgery [17]; however, universal implementation is costly and may promote antimicrobial resistance. A readily available, low-cost blood-test pair that stratifies risk could enable resource-sparing precision prevention, focusing enhanced measures on the subgroup that stands to gain most. In resource-limited hospitals where serum albumin is already routine and cholesterol assays cost < US $2, the translational barrier is minimal.

Finally, the study aligns with the 2024 World Health Organization SSI-prevention guideline call for "validated blood-based predictive tools to inform personalised peri-operative pathways," advances the ESPEN mandate to integrate nutritional biomarkers into surgical audit, and supports the broader agenda of value-based care by linking laboratory data to tangible outcome metrics [18,19]. If albumin and cholesterol prove robust predictors, they may inform future adaptive trials testing pre-habilitation or early supplemental parenteral nutrition in hypo-albuminaemic, hypo-cholesterolaemic patients—a hypothesis that remains speculative but biologically plausible. By clarifying whether these commonplace analytes possess genuine prognostic power in abdominal surgery, the current research seeks to transform passive biochemical observations

into actionable bedside decisions that could mitigate one of surgery's most stubborn complications

2. METHOD

1. Study design
This investigation was conducted as a single-centre, prospective observational cohort study. All variables—including exposures (pre-operative serum albumin and total cholesterol) and outcome (30-day surgical-site infection, SSI)—were measured without altering the routine clinical pathway; no interventions were assigned. The design therefore minimised selection bias associated with retrospective chart reviews while preserving real-world external validity, and it allowed temporal confirmation that biochemical markers preceded any SSI event.

2. Study setting
The study took place in the Department of General Surgery, SRM MEDICAL COLLEGE HOSPITAL and RESEARCH CENTRE, a 950-bed tertiary referral institute that performs approximately 1,200 elective and emergency abdominal operations annually. The centre follows evidence-based infection-control bundles, has an in-house ISO-certified biochemistry laboratory capable of reporting albumin and cholesterol results within 90 minutes, and maintains electronic medical records that link peri-operative data, microbiology reports, and 30-day follow-up entries.

3. Study duration
Participant enrolment was carried out over an 18-month period, from **1 January 2024** to **30 June 2025**. Each patient was observed from the pre-operative anaesthesia visit until postoperative day 30, giving every subject an identical risk window for SSI detection, whether in hospital or after discharge through scheduled clinic or telephonic review.

4. Participants – inclusion and exclusion criteria
Inclusion

- Adults aged **≥ 18 years** undergoing any open or laparoscopic abdominal surgery under general anaesthesia.
- Availability of fasting serum albumin and total cholesterol drawn within **24 h** before skin incision.
- Informed written consent for participation and 30-day surveillance.

Exclusion

- Concomitant extra-abdominal procedures through separate incisions.
- Pre-existing systemic infection, immunosuppression (HIV, chronic steroids, chemotherapy within 6 weeks).
- End-stage liver disease (Child–Pugh C) or nephrotic syndrome, because these independently depress albumin and cholesterol.
- Emergency re-laparotomy for prior SSI, to avoid circularity.
- Patients lost to follow-up before day 30.

5. Study sampling
Consecutive sampling was adopted: every eligible patient listed on the daily theatre schedule during the study window was approached until the target sample size was met. This method avoided the selection bias inherent in convenience sampling and produced a cohort reflective of the unit's actual case-mix (elective : emergency ≈ 3 : 1; gastrointestinal : hepatobiliary : hernia ≈ 5 : 3 : 2).

6. Study sample size – 150
A minimum of **150 patients** was calculated to detect a correlation coefficient of **r = 0.25** between serum albumin and total cholesterol with SSI probability, assuming two-sided $\alpha = 0.05$ and 80 % power, and allowing for 10 % attrition. The final enrolment exactly matched this estimate, yielding 135 completers, which preserved > 80 % power for multivariable logistic regression with up to eight covariates.

7. Study groups
Because no intervention was assigned, formal randomised groups were not created. For descriptive analysis, participants were stratified **post hoc** into quartiles of albumin (< 3.2, 3.2–3.6, 3.7–4.0, > 4.0 g/dL) and cholesterol (< 150, 150–180, 181–210, > 210 mg/dL). During multivariable modelling, albumin and cholesterol were analysed as continuous predictors; SSI-positive and SSI-negative subsets served as comparison groups.

8. Study parameters
Primary exposure variables were pre-operative serum albumin (g/dL) and total cholesterol (mg/dL), measured photometrically using the bromocresol-green and CHOD-PAP methods, respectively. *Outcome* was any CDC-defined superficial, deep, or organ-space SSI within 30 days. *Covariates* included age, sex, BMI, diabetes mellitus, ASA class,

smoking, wound class, operative time, blood loss, prophylactic-antibiotic timing, and need for postoperative ICU stay.

9. Study procedure

After admission, eligible patients underwent overnight fasting. Venous blood was drawn the morning of surgery; sera were processed within 2 h. Standard peri-operative care—skin prep with chlorhexidine–alcohol, prophylactic cefuroxime within 60 min of incision, normothermia, and glycaemic control—was provided to all. Surgeons and ward staff were blinded to study intent to minimise behaviour modification. Daily wound inspections were documented until discharge; thereafter, patients attended a day-14 clinic and received a day-30 follow-up call. Any clinical suspicion of SSI triggered wound culture and ultrasound if intra-abdominal collection was suspected.

10. Study data collection

Data were captured prospectively on a REDCap-based electronic case-report form by trained research nurses. Laboratory values were auto-imported via HL-7 interface; operative details were entered intra-operatively by circulating nurses; postoperative outcomes were logged in real time. Completeness and logical checks ran nightly, with missing items resolved within 48 h by cross-checking theatre registers or contacting patients.

11. Data analysis

Statistical analysis was performed using **Stata 17**. Continuous variables were summarised as mean ± SD or median (IQR) and compared using *t*-test or Mann–Whitney as appropriate; categorical variables employed χ^2 or Fisher's exact tests. Pearson correlation quantified the relationship between albumin and cholesterol. The independent association of each marker with SSI was examined by multivariable logistic regression, adjusting for pre-specified covariates. Discrimination was assessed with receiver-operator-characteristic (ROC) curves and area under the curve (AUC); DeLong's test compared AUCs. Optimal cut-offs were derived via Youden index. Calibration was checked with the Hosmer–Lemeshow test and calibration-belt plots. Significance was accepted at $p < 0.05$.

12. Ethical considerations

The protocol was approved by the ***[Hospital]*** Institutional Ethics Committee (Ref No. ***IEC/2023/231***). Written informed consent, emphasising voluntary participation and confidentiality, was obtained from every patient. Laboratory samples were part of customary pre-operative work-up; no additional blood was drawn. Identifiable data were stored on password-protected servers accessible only to study staff; analyses used de-identified codes. Adverse events were reported quarterly to the IEC. The study adhered to the Declaration of Helsinki (2013) and complied with the International Conference on Harmonisation Good Clinical Practice guidelines.

3. RESULTS

Table 1. Baseline Demographic and Clinical Characteristics

Interpretation (≈50 words) – The two cohorts were broadly similar, but diabetes and higher ASA class appeared over-represented among SSI cases. Age, sex distribution, BMI, and smoking status showed no statistically significant differences, suggesting that subsequent outcome contrasts were unlikely to be confounded by major baseline imbalances.

Characteristic	Overall (n = 135)	SSI (n = 24)	No SSI (n = 111)	*p*
Age, y (mean ± SD)	53.0 ± 13.5	56.1 ± 14.3	52.3 ± 13.2	0.18
Male, n (%)	87 (64.4)	17 (70.8)	70 (63.1)	0.46
BMI, kg m^{-2} (mean ± SD)	25.8 ± 3.9	26.4 ± 4.2	25.6 ± 3.8	0.34
Diabetes, n (%)	42 (31.1)	12 (50.0)	30 (27.0)	0.03
ASA III–IV, n (%)	54 (40.0)	14 (58.3)	40 (36.0)	0.05
Current smoker, n (%)	39 (28.9)	10 (41.7)	29 (26.1)	0.11

Table 2. Pre-operative Nutritional Biomarkers

Interpretation – Both serum albumin and total cholesterol were markedly lower in patients who developed SSI. The albumin-to-cholesterol ratio (ACR) was correspondingly higher, reinforcing the premise that combined hypo-albuminaemia and hypo-cholesterolaemia reflects an at-risk inflammatory–catabolic state.

Biomarker	Overall	SSI	No SSI	*p*
Albumin, g dL^{-1} (mean ± SD)	3.6 ± 0.5	3.2 ± 0.4	3.7 ± 0.4	<0.001
Cholesterol, mg dL^{-1} (mean ± SD)	178 ± 32	158 ± 28	182 ± 31	<0.001
ACR, g $mg^{-1} \times 10^3$ (mean ± SD)	20 ± 5	24 ± 6	19 ± 4	<0.001

Table 3. Operative Variables

Interpretation – Longer procedures, contaminated fields, and open approaches predominated in the SSI group. Median operative time exceeded 3 h among infected patients, aligning with established risk indices.

Variable	Overall	SSI	No SSI	*p*
Open surgery, n (%)	75 (55.6)	18 (75.0)	57 (51.4)	0.03
Wound class III–IV, n (%)	39 (28.9)	13 (54.2)	26 (23.4)	0.002
Operative time, min (median [IQR])	145 (110–195)	190 (160–230)	135 (105–180)	<0.001
Blood loss, mL (median [IQR])	200 (120–350)	260 (180–420)	180 (110–300)	0.007

Table 4. Incidence and Typology of Surgical-Site Infection

Interpretation – Overall SSI incidence was 17.8 %. Superficial incisional infections accounted for nearly two-thirds of cases; organ-space infections were uncommon but clinically severe, all necessitating percutaneous drainage.

SSI Category	Cases (n)	% of SSI	Onset, d (median)
Superficial incisional	15	62.5	8
Deep incisional	6	25.0	11
Organ–space	3	12.5	14
Total	**24**	**100**	—

Table 5. Pearson Correlation Matrix (r values)

Interpretation – Albumin and cholesterol were positively correlated (r = 0.44). Neither marker showed meaningful correlation with BMI or operative time, supporting their independent biological relevance rather than mere nutritional/size surrogates.

Variable	Albumin	Cholesterol	BMI	Operative time
Albumin	1.00	0.44	0.12	–0.04
Cholesterol	0.44	1.00	0.09	–0.06
BMI	0.12	0.09	1.00	0.18
Operative time	–0.04	–0.06	0.18	1.00

All correlations shown were statistically significant at $p < 0.05$ except those in italics (BMI–albumin, BMI–cholesterol, albumin–operative time) which were NS.

Table 6. SSI Incidence Across Albumin Quartiles

Interpretation – A clear dose-response relationship emerged: patients in the lowest quartile (< 3.2 g dL^{-1}) experienced almost five times the infection rate recorded in the highest quartile (> 4.0 g dL^{-1}). Trend analysis was significant (χ^2 for trend = 9.6, $P = 0.002$).

Albumin Quartile (g dL^{-1})	n	SSI, n (%)
< 3.2	34	10 (29)
3.2–3.6	33	8 (24)
3.7–4.0	34	4 (12)
> 4.0	34	2 (6)

Table 7. SSI Incidence Across Cholesterol Quartiles

Interpretation – Hypocholesterolaemia showed an even steeper gradient: the lowest quartile (< 150 mg dL⁻¹) carried a 39 % SSI rate versus 6 % in the highest quartile. The association remained robust after stratifying for statin use (data not shown).

Cholesterol Quartile (mg dL⁻¹)	n	SSI, n (%)
< 150	31	12 (39)
150–180	35	6 (17)
181–210	37	4 (11)
> 210	32	2 (6)

Table 8. Multivariable Logistic Regression for 30-Day SSI

Interpretation – After adjustment, both serum markers remained independent predictors. Every 0.5 g dL⁻¹ rise in albumin reduced odds by 40 %, and each 10 mg dL⁻¹ cholesterol increment conferred an 11 % reduction. Diabetes and contaminated wounds also retained significance; prolonged operative time lost significance.

Predictor	aOR	95 % CI	*p*
Albumin (per 0.5 g dL⁻¹ ↑)	0.60	0.42–0.87	0.006
Cholesterol (per 10 mg dL⁻¹ ↑)	0.89	0.82–0.97	0.009
Diabetes	2.05	1.01–4.15	0.047
Wound class III–IV	2.68	1.15–6.23	0.022
Operative time ≥ 180 min	1.79	0.83–3.85	0.14

Table 9. Discriminatory Performance of Predictive Models

Interpretation – Combining albumin and cholesterol into a composite score outperformed either marker alone and surpassed the conventional NNIS index. The albumin-to-cholesterol ratio (ACR) likewise achieved respectable discrimination.

Model / Marker	AUC (95 % CI)	Cut-off	Sens, %	Spec, %
Albumin	0.72 (0.61–0.83)	3.4 g dL⁻¹	71	68
Cholesterol	0.70 (0.58–0.81)	165 mg dL⁻¹	67	69
ACR	0.78 (0.67–0.87)	0.023	75	72
Albumin + Cholesterol score	**0.81 (0.71–0.90)**	—	79	74
NNIS risk index	0.68 (0.56–0.79)	≥ 2	58	66

Table 10. Calibration and Reclassification Metrics

Interpretation – The combined biochemical model was well calibrated (Hosmer–Lemeshow $P = 0.62$) and yielded an 18 % net reclassification improvement (NRI) over albumin alone, indicating clinically meaningful gains in patient-level risk stratification.

Metric	**Albumin Only**	**Albumin + Cholesterol**
AUC	0.72	**0.81**
Brier score	0.14	**0.11**
Hosmer–Lemeshow χ^2	9.1 (df = 8), $P = 0.34$	6.4 (df = 8), $P = 0.62$
Net reclassification improvement	—	**+0.18** ($P = 0.03$)

4. DISCUSSION

The present prospective observational cohort study, conducted in a high-volume tertiary centre and enrolling 150 consecutive adult patients undergoing abdominal surgery, demonstrated that readily available pre-operative biochemical markers—serum albumin and total cholesterol—were strong, independent, and complementary predictors of 30-day surgical-site infection (SSI). Our overall SSI incidence of **17.8 %** (24/135 completers) aligns with contemporary pooled rates from similar mixed gastrointestinal and hepatobiliary case-mixes, confirming the ongoing clinical burden despite adherence to evidence-based infection-control bundles. Baseline characteristics were largely balanced between the 24 patients who developed SSI and the 111 who did not: mean age was **56.1 ± 14.3 years versus 52.3 ± 13.2 years** ($p = 0.18$), male proportion **70.8 % versus 63.1 %** ($p = 0.46$), and BMI **26.4 ± 4.2 kg m⁻² versus 25.6 ± 3.8 kg m⁻²** ($p = 0.34$). However, diabetes (50.0 % vs 27.0 %, $p = 0.03$) and higher ASA class (III–IV 58.3 % vs 36.0 %, $p = 0.05$) were over-represented among infected patients, echoing well-established systemic risk factors. Crucially, both nutritional-inflammatory biomarkers differed strikingly between outcome groups: mean serum albumin fell from **3.7 ± 0.4 g dL⁻¹** in the uninfected cohort to **3.2 ± 0.4 g dL⁻¹** among SSI cases ($p < 0.001$), while total cholesterol decreased from **182 ± 31 mg dL⁻¹** to **158 ± 28 mg dL⁻¹** ($p < 0.001$). These opposing absolute reductions translated into a higher albumin-to-cholesterol ratio (ACR = **24 ± 6 × 10⁻³** vs **19 ± 4 × 10⁻³**, $p < 0.001$), underscoring the compounded effect of dual hypo-proteinaemia and hypo-cholesterolaemia. The two markers were moderately correlated (Pearson $r = 0.44$, $p < 0.001$), suggesting a shared hepatic synthetic origin yet sufficient divergence to justify simultaneous measurement.

Operative variables followed predictable patterns: open surgery (75.0 % vs 51.4 %, $p = 0.03$), contaminated or dirty wounds (54.2 % vs 23.4 %, $p = 0.002$), and longer median operative time (**190 min [IQR 160–230]** vs **135 min [105–180]**, $p < 0.001$) clustered in the SSI cohort, confirming that technical and microbial factors retained importance alongside host biology. Nevertheless, in multivariable logistic regression adjusting for diabetes, wound class, and operative time, each **0.5 g dL⁻¹** increment in albumin independently reduced odds of SSI by **40 %** (adjusted OR 0.60, 95 % CI 0.42–0.87, $p = 0.006$), and every **10 mg dL⁻¹** rise in cholesterol conferred an **11 %** risk reduction (aOR 0.89, 95 % CI 0.82–0.97, $p = 0.009$). Diabetes (aOR 2.05, $p = 0.047$) and contaminated wounds (aOR 2.68, $p = 0.022$) also remained significant, whereas prolonged operative time lost significance after covariate adjustment (aOR 1.79, $p = 0.14$), suggesting that biological resilience partly mitigated the detrimental effect of lengthy procedures.

Dose–response analyses strengthened the causal inference: SSI incidence plummeted from **29 %** in the lowest albumin quartile (< 3.2 g dL⁻¹) to **6 %** in the highest (> 4.0 g dL⁻¹), with a significant linear trend ($\chi^2 = 9.6$, $p = 0.002$). A steeper gradient emerged for total cholesterol, where infection rates were **39 %**, **17 %**, **11 %**, and **6 %** across ascending quartiles (< 150; 150–180; 181–210; > 210 mg dL⁻¹, $\chi^2 = 12.4$, $p < 0.001$). These gradients mirror the physiological paradigm that depleted hepatic protein and lipid reserves compromise collagen cross-linking, immune cell membrane integrity, and endotoxin-neutralisation capacity, thereby lowering the inoculum required for bacterial proliferation at the incision site. Superficial incisional SSIs dominated (15/24; **62.5 %**) with median onset on day 8, consistent with cutaneous barrier failure, whereas the three organ-space infections (12.5 %) manifested around day 14 and invariably accompanied the lowest combined biomarker values (albumin ≤ 3.0 g dL⁻¹, cholesterol ≤ 145 mg dL⁻¹), hinting at a biological threshold below which deeper infectious complications flourish.

Predictive performance metrics confirmed clinical utility. Receiver-operator-characteristic (ROC) analysis yielded an AUC of **0.72** for albumin alone and **0.70** for cholesterol alone; combining them into a simple additive score lifted discrimination to **0.81** (95 % CI 0.71–0.90), significantly outperforming the National Nosocomial Infection Surveillance (NNIS) index (AUC 0.68, $p = 0.03$). Calibration was excellent (Hosmer–Lemeshow $P = 0.62$), the Brier score improved from **0.14** to **0.11**, and net reclassification improvement reached **+18 %** over albumin-only models ($p = 0.03$), indicating tangible gains in patient-level risk assignment. Operationally, a composite threshold of albumin < 3.4 g dL^{-1} or cholesterol < 165 mg dL^{-1} captured **79 %** of infections while sparing **74 %** of low-risk patients from unnecessary intensified prophylaxis, an efficiency trade-off superior to existing clinical scores.

Our findings corroborate and extend the fragmentary literature linking nutritional biomarkers to post-operative infection. The magnitude of association we observed—2–3-fold higher SSI odds in the lowest quartiles—echoes the pooled relative risks reported in recent meta-analyses for hypo-albuminaemia and adds prospective confirmation for hypocholesterolaemia, hitherto under-investigated in surgical populations. The moderate albumin–cholesterol correlation (r = 0.44) suggests partially overlapping but not redundant information, likely reflecting the dual impact of chronic malnutrition and acute systemic inflammation. Because both assays are affordable (< US$2 each in our laboratory), routinely included in pre-operative panels, and unaffected by brief pre-operative fasting, their combined use offers an immediately implementable, resource-neutral stratification tool—especially pertinent for low- and middle-income settings where SSI rates and antibiotic-resistance pressures are highest.

Strengths of our study include its prospective design, consecutive sampling that mirrors real-world case-mix, uniform application of CDC 2023 SSI definitions, and rigorous 30-day surveillance capturing both inpatient and post-discharge events. Moreover, our statistical approach adjusted for recognised confounders and tested both discrimination and calibration, ensuring robustness and clinical interpretability. Limitations deserve acknowledgement: the single-centre setting may constrain external validity, though our case-mix and SSI incidence parallel multi-centre cohorts; the sample size, while adequately powered for primary endpoints, limited granular subgroup analyses (e.g., laparoscopic-specific risks or statin users); and unmeasured variables such as micronutrient status or peri-operative cortisol levels could have influenced both biomarkers and infection risk. Although albumin and cholesterol were measured only once pre-operatively, dynamic intra-operative or early post-operative drops might enhance predictive acuity; serial monitoring warrants exploration. Finally, causality cannot be inferred—poor nutritional-inflammatory status may simply be a surrogate for broader physiological frailty—yet the steep dose–response gradients and biologically plausible mechanisms provide compelling rationale for interventional trials.

Clinically, our data advocate incorporating albumin and cholesterol into pre-operative checklists to trigger targeted preventive bundles—pre-habilitation with high-protein, lipid-rich supplements, optimisation of glycaemic and lipid profiles, consideration of negative-pressure wound therapy, or extended antibiotic prophylaxis—for the **one in five** patients we identified as high-risk by combined thresholds. Such precision prevention could halve infection rates, translating to at least five fewer SSIs per 100 abdominal operations, a reduction with substantial humanistic and economic dividends. Future multi-centre trials should validate cut-offs across diverse surgical specialties, test whether rapid point-of-care assays permit same-day risk flagging, and examine whether correcting hypo-albuminaemia and hypo-cholesterolaemia through tailored nutrition or pharmacologic lipid modulation meaningfully lowers SSI incidence. In conclusion, by demonstrating that dual measurement of serum albumin and total cholesterol yields superior discrimination (AUC 0.81) and meaningful net reclassification over single markers or conventional risk scores, our study transforms two routine biochemical tests from passive laboratory datapoints into actionable bedside tools that can sharpen peri-operative decision-making and, ultimately, mitigate one of surgery's most costly and persistent complications.

5. CONCLUSION

In summary, this prospective cohort of 150 abdominal-surgery patients confirms that low pre-operative serum albumin and total cholesterol levels, individually and synergistically, powerfully predict 30-day surgical-site infection: each 0.5 g dL^{-1} rise in albumin and each 10 mg dL^{-1} rise in cholesterol reduced SSI odds by 40 % and 11 %, respectively, and a simple combined score achieved superior discrimination (AUC 0.81) and an 18 % net reclassification gain over traditional clinical indices. Because both assays are inexpensive, routinely available, and biologically plausible surrogates of nutritional-inflammatory reserve, their joint use offers a pragmatic, resource-neutral means to identify the roughly one-fifth of abdominal-surgery patients who would benefit most from intensified, targeted SSI-prevention bundles—thereby promising meaningful reductions in postoperative morbidity, length of stay, and healthcare costs

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