International Journal of Science and Research (IJSR)
ISSN: 2319-7064
Impact Factor 2025: 7.089

Beyond Cirrhosis: Etiology, Perioperative Determinants, and Clinical Outcomes of De Novo Postoperative Ascites Following Major Abdominal Surgery

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Abstract: ***Background**: De novo postoperative ascites is an uncommon but clinically significant complication following major abdominal surgery in patients without pre-existing cirrhosis. Evidence regarding its etiology, perioperative determinants, and clinical outcomes remains limited. **Methods**: A prospective observational study was conducted at a tertiary care center between October 2024 and September 2025. Twenty-eight adult patients who developed postoperative ascites following major abdominal surgery were enrolled. Demographic characteristics, surgical variables, ascitic fluid characteristics, management strategies, postoperative complications, and clinical outcomes were analyzed. Correlation analysis was performed to identify factors associated with ascitic fluid volume. **Results**: The mean age of the study population was 41.5 ± 15.3 years, and 64.3% were male. Hepatopancreatobiliary and colorectal procedures constituted the most common surgical categories. Exudative and transudative ascites were equally distributed (50.0% each). Large-volume ascites (>1000 mL) was observed in 50.0% of patients. Diuretic therapy was the most frequently employed management strategy. Postoperative complications occurred in 85.7% of patients, with wound infection (21.4%), sepsis (14.3%), and anastomotic leak (14.3%) being the most common. **Conclusions**: De novo postoperative ascites is a multifactorial complication associated with substantial postoperative morbidity and prolonged hospitalization. Early diagnosis, etiological evaluation, and timely multidisciplinary management are essential for improving outcomes.*

Keywords: Postoperative ascites; abdominal surgery; hepatopancreatobiliary surgery; postoperative complications; ascitic fluid; surgical outcomes

1. Introduction

Postoperative ascites is an uncommon yet clinically significant complication encountered after major abdominal surgery. Defined as the pathological accumulation of fluid within the peritoneal cavity following a surgical intervention, postoperative ascites may develop through diverse mechanisms including portal hypertension, lymphatic disruption, inflammatory exudation, hypoalbuminemia, and occult visceral leakage [1,2]. Although ascites is traditionally viewed as a manifestation of advanced chronic liver disease, its occurrence in patients without pre-existing hepatic pathology has increasingly attracted attention among surgeons because of its association with prolonged hospitalization, increased healthcare expenditure, delayed recovery, and postoperative morbidity [3,4]. With the growing complexity of hepatobiliary, pancreatic, colorectal, vascular, and oncological procedures, recognition of postoperative ascites as a distinct surgical entity has become increasingly important [5].

The global burden of ascites is predominantly attributed to liver cirrhosis, accounting for approximately 75–85% of all cases, whereas malignancy, cardiac failure, nephrotic syndrome, and peritoneal tuberculosis constitute less frequent etiologies [6,7]. In contrast, de novo postoperative ascites develops in individuals without a previous history of chronic liver disease and often arises as a direct consequence of perioperative physiological disturbances or surgical injury [8]. Despite advances in perioperative care and enhanced recovery protocols, postoperative ascites continues to represent a major challenge in selected surgical populations, particularly after extensive hepatic resections, pancreatic surgeries, retroperitoneal dissections, and complex colorectal procedures [9, 10].

A fundamental distinction exists between cirrhotic ascites and de novo postoperative ascites. Cirrhotic ascites results primarily from portal hypertension, splanchnic vasodilatation, activation of the renin–angiotensin–aldosterone system, and impaired hepatic synthetic function, leading to progressive sodium and water retention [11,12]. In contrast, postoperative ascites is frequently multifactorial and may occur in the absence of chronic portal hypertension. Surgical trauma can induce inflammatory alterations in peritoneal permeability, while disruption of lymphatic channels during extensive dissection may lead to persistent lymphatic leakage and chylous ascites [13]. Likewise, hepatic resections may produce transient portal hyperperfusion and sinusoidal congestion, whereas occult bile leaks, pancreatic fistulas, or anastomotic complications can trigger exudative fluid accumulation within the abdominal cavity [14,15]. Consequently, the diagnostic and therapeutic approach to postoperative ascites differs substantially from conventional management algorithms used in hepatology.

From a surgical perspective, postoperative ascites is associated with several adverse consequences. Persistent ascitic accumulation increases intra-abdominal pressure, compromises respiratory mechanics, impairs wound healing, and predisposes patients to surgical site infections, wound dehiscence, electrolyte disturbances, and renal dysfunction [16,17]. Furthermore, the presence of ascites may obscure early signs of serious postoperative complications such as bile leaks, pancreatic fistulas, enteric perforation, or anastomotic dehiscence, potentially delaying definitive intervention [18]. In hepatobiliary surgery, postoperative ascites has been identified as an early indicator of post-hepatectomy liver failure and inadequate future liver

remnant function [19]. Similarly, in pancreatic and retroperitoneal surgeries, chylous ascites contributes to nutritional depletion, immunosuppression, and prolonged hospitalization [20]. These observations underscore the importance of understanding the mechanisms and determinants underlying postoperative fluid accumulation.

Despite its clinical significance, the available literature remains fragmented and largely procedure-specific. Most published studies have focused on postoperative ascites after hepatectomy, liver transplantation, pancreaticoduodenectomy, retroperitoneal lymphadenectomy, or vascular surgery [21–24]. Consequently, current knowledge is derived from heterogeneous populations and specialized surgical settings, limiting the generalizability of findings to broader abdominal surgical practice. Moreover, many studies concentrate on management strategies rather than investigating the underlying etiological spectrum and perioperative determinants responsible for the development of ascites [25]. There remains a paucity of prospective observational data evaluating postoperative ascites across multiple abdominal surgical procedures, particularly among patients without pre-existing cirrhosis or established medical causes of fluid accumulation.

An additional limitation of existing evidence is the lack of standardized risk stratification models capable of identifying patients at increased risk for postoperative ascites. The relative contributions of operative duration, intraoperative blood loss, surgical complexity, lymphatic disruption, and postoperative biochemical abnormalities remain incompletely understood [26]. Furthermore, few studies have examined the relationship between postoperative ascites and clinically relevant outcomes such as length of hospital stay, intensive care utilization, postoperative complications, and short-term mortality [27]. Addressing these knowledge gaps is essential for developing preventive strategies and optimizing perioperative management.

Therefore, the present study was undertaken to evaluate de novo postoperative ascites as a distinct postoperative complication among patients undergoing major abdominal surgery in a tertiary-care setting. The study aims to characterize the etiological spectrum of postoperative ascites, identify perioperative determinants associated with its development, and assess subsequent clinical outcomes and complications. By examining postoperative ascites beyond the traditional framework of cirrhosis-related fluid accumulation, this study seeks to generate clinically relevant evidence that may contribute to improved risk stratification, earlier diagnosis, and more effective management of affected patients.

We hypothesized that de novo postoperative ascites represents a multifactorial surgical complication predominantly influenced by operative complexity, intraoperative physiological disturbances, and procedure-specific factors, and that its occurrence is associated with increased postoperative morbidity, prolonged hospitalization, and adverse short-term clinical outcomes.

2. Methods

Study Design

This prospective observational cohort study was conducted to investigate the etiological spectrum, perioperative determinants, and clinical outcomes of de novo postoperative ascites following major abdominal surgery. The study was designed in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines. Given the exploratory nature of the research question and the absence of standardized risk prediction models for postoperative ascites in non-cirrhotic patients, an observational design was considered most appropriate to characterize real-world clinical presentations and outcomes.

Study Setting

The study was conducted at a tertiary-care teaching hospital with specialized hepatobiliary, gastrointestinal, colorectal, pancreatic, vascular, and emergency surgical services. Consecutive patients who developed postoperative ascites following major abdominal surgery were prospectively enrolled between October 2024 and September 2025. The institution serves as a regional referral center for complex abdominal surgical procedures, allowing the inclusion of a heterogeneous spectrum of surgical cases.

Eligibility Criteria

Inclusion Criteria

Patients were eligible for enrollment if they fulfilled all of the following criteria:

- Age ≥18 years.
- Underwent major abdominal surgery during the study period.
- Developed postoperative ascites within 30 days following surgery.
- No documented preoperative ascites.
- No established medical cause of ascites before surgery.
- Provided written informed consent for participation.

Exclusion Criteria

Patients were excluded if they met any of the following criteria:

- Age <18 years.
- Known liver cirrhosis or portal hypertension.
- Chronic liver disease with pre-existing ascites.
- Nephrotic syndrome.
- Congestive cardiac failure-associated ascites.
- Peritoneal tuberculosis.
- Peritoneal carcinomatosis diagnosed preoperatively.
- End-stage renal disease requiring dialysis.
- Patients unwilling to participate.

Definition of De Novo Postoperative Ascites

For the purposes of this study, de novo postoperative ascites was defined as the accumulation of free intraperitoneal fluid detected clinically and confirmed by ultrasonography or computed tomography within 30 days after surgery in patients without a prior history of ascites or identifiable preoperative medical causes of fluid accumulation.

Ascites was considered clinically significant when it resulted in abdominal distension, respiratory discomfort, increased drain output, requirement of diagnostic or therapeutic

paracentesis, prolonged hospitalization, or additional postoperative intervention.

Surgical Categories

To facilitate etiological analysis, surgical procedures were classified into the following categories:

1) **Hepatobiliary surgery**
- Hepatectomy
- Splenectomy
- Biliary procedures

2) **Pancreatic surgery**
- Distal pancreatectomy
- Pancreatic resections

3) **Colorectal surgery**
- Anterior resection
- Hartmann's procedure
- Bowel resection and anastomosis

4) **Upper gastrointestinal surgery**
- Gastric resections
- Exploratory laparotomy for gastric pathology

5) **Emergency abdominal surgery**
- Perforation peritonitis
- Intestinal obstruction
- Acute appendicitis

6) **Hernia and abdominal wall surgery**

7) **Vascular and miscellaneous abdominal procedures**

This classification enabled evaluation of procedure-specific patterns of postoperative ascites.

Ascitic Fluid Analysis

All patients underwent diagnostic evaluation to identify the probable etiology of ascites and exclude surgically correctable causes.

Fluid samples were obtained either through ultrasound-guided paracentesis or from surgically placed drains when available.

The following parameters were analyzed:
- Appearance (clear, serous, hemorrhagic, bilious, chylous, purulent)
- Total protein concentration
- Albumin concentration
- Serum-ascites albumin gradient (SAAG)
- Cell count and differential count
- Triglyceride level
- Amylase level when pancreatic leak was suspected
- Bilirubin concentration when biliary leak was suspected
- Microbiological culture and sensitivity

Ascites was categorized as:

Transudative Ascites
- SAAG ≥1.1 g/dL

Exudative Ascites
- SAAG <1.1 g/dL

Chylous Ascites
- Triglyceride concentration >110 mg/dL with characteristic milky appearance

Potential etiological mechanisms including lymphatic disruption, portal hypertension, inflammatory exudation, pancreatic leak, biliary leak, and postoperative hypoalbuminemia were documented.

Perioperative Variables

The following perioperative variables were prospectively recorded:

Demographic Variables
- Age
- Sex
- Body mass index
- Comorbidities

Operative Variables
- Primary diagnosis
- Type of surgery
- Elective or emergency surgery
- Operative duration (minutes)
- Estimated blood loss (mL)
- Requirement of blood transfusion
- Intraoperative complications
- Presence of extensive adhesions
- Extent of lymphadenectomy
- Requirement of vascular reconstruction

Postoperative Variables

a) Day of onset of ascites
b) Estimated ascitic fluid volume
c) Laboratory parameters
   - Hemoglobin
   - Serum albumin
   - Serum bilirubin
   - SGOT
   - SGPT
   - Blood urea
   - Serum creatinine
   - Lactate dehydrogenase (LDH)
d) Requirement for intensive care admission
e) Requirement for therapeutic interventions

Outcome Measures

Primary Outcome

The primary outcome was characterization of the etiological spectrum of de novo postoperative ascites following major abdominal surgery.

Secondary Outcomes

Secondary outcomes included:
- Time to ascites resolution.
- Requirement for therapeutic paracentesis.
- Requirement for albumin supplementation.
- Requirement for diuretic therapy.
- Requirement for reoperation.

- Length of hospital stay.
- Intensive care unit stay.
- Postoperative complications.
- Thirty-day readmission.
- Thirty-day mortality.

Postoperative complications were categorized as:

- Surgical site infection
- Wound dehiscence
- Sepsis
- Acute kidney injury
- Respiratory complications
- Electrolyte disturbances
- Multi-organ dysfunction

Statistical Analysis

Data were entered into Microsoft Excel and analyzed using Jamovi version 2.4.8 (The Jamovi Project, Sydney, Australia). Continuous variables were tested for normality using the Shapiro–Wilk test. Normally distributed variables were presented as mean ± standard deviation, whereas non-normally distributed variables were summarized as median and interquartile range.

Categorical variables were expressed as frequencies and percentages.

Associations between categorical variables were assessed using the Chi-square test or Fisher's exact test as appropriate.

Comparisons of continuous variables between groups were performed using the independent samples t-test or Mann–Whitney U test.

Correlation between perioperative variables and ascitic fluid volume was evaluated using Spearman's rank correlation coefficient.

Univariate logistic regression analyses were performed to identify predictors of adverse clinical outcomes. Variables with $p < 0.10$ on univariate analysis were considered for multivariable logistic regression modeling.

Statistical significance was defined as a two-tailed p-value <0.05.

Ethical Approval

The study protocol was reviewed and approved by the Institutional Ethics Committee of the participating tertiary-care center before patient enrollment. The study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki and its subsequent amendments. Written informed consent was obtained from all participants prior to inclusion in the study. Confidentiality and anonymity of patient information were maintained throughout data collection, analysis, and reporting.

3. Results

A total of 28 patients who developed de novo postoperative ascites following major abdominal surgery were included in the analysis. The mean age of the cohort was 41.5 ± 15.3 years (range 22–68 years), and males constituted 64.3% of the study population. Postoperative ascites occurred across a broad spectrum of benign and malignant surgical conditions and was observed following both elective and emergency procedures.

Table 1: Baseline Demographic, Diagnostic, and Surgical Characteristics (N=28)

Variable	n (%)
Male sex	18 (64.3)
Female sex	10 (35.7)
Age <40 years	14 (50.0)
Age 40–59 years	10 (35.7)
Age ≥60 years	4 (14.3)
Malignant pathology	11 (39.3)
Benign pathology	17 (60.7)
Elective surgery	15 (53.6)
Emergency surgery	13 (46.4)
Intraoperative complications	10 (35.7)

Table 1 summarizes the baseline characteristics of the study population. The mean age of patients was 41.5 ± 15.3 years, with half of the cohort being younger than 40 years. Male patients predominated (64.3%). Benign surgical conditions accounted for 60.7% of cases, while malignant pathologies represented 39.3%. Elective and emergency procedures were almost equally distributed. Intraoperative complications occurred in 35.7% of patients, indicating that postoperative ascites developed across a diverse range of patient demographics and operative settings.

Table 2: Distribution of Surgical Categories Associated with Postoperative Ascites

Surgical Category	n (%)
Colorectal surgery	8 (28.6)
Hepatopancreatobiliary surgery	8 (28.6)
Upper gastrointestinal surgery	3 (10.7)
Emergency exploratory procedures	3 (10.7)
Splenic surgery	2 (7.1)
Cholecystectomy procedures	3 (10.7)
Hernia surgery	2 (7.1)

Table 2 illustrates the spectrum of surgical procedures associated with de novo postoperative ascites. Colorectal and hepatopancreatobiliary surgeries each accounted for 28.6% of cases, representing the most frequently involved surgical categories. Upper gastrointestinal, cholecystectomy-related, splenic, and emergency exploratory procedures contributed smaller proportions. These findings suggest that postoperative ascites is particularly associated with complex abdominal procedures involving extensive dissection, tissue handling, and disruption of lymphatic or vascular structures.

Table 3: Ascitic Fluid Characteristics and Etiological Classification

Variable	n (%)
Exudative ascites	14 (50.0)
Transudative ascites	14 (50.0)
Small volume (<500 mL)	3 (10.7)
Moderate volume (500–1000 mL)	11 (39.3)
Large volume (>1000 mL)	14 (50.0)

Table 3 presents the biochemical and volumetric characteristics of postoperative ascites. Exudative and

transudative fluid collections were equally represented, indicating the coexistence of inflammatory and hydrostatic mechanisms in postoperative fluid accumulation. Large-volume ascites (>1000 mL) was observed in half of the study population, while only a small proportion of patients exhibited low-volume fluid collections. The predominance of moderate-to-large fluid volumes highlights the clinical significance of postoperative ascites and its potential impact on recovery.

Table 4: Management Strategies and Clinical Outcomes

Variable	n (%)
Conservative management	6 (21.4)
Diuretics alone	8 (28.6)
Albumin plus diuretics	3 (10.7)
Paracentesis plus antibiotics	3 (10.7)
Repeated paracentesis	6 (21.4)
Therapeutic paracentesis plus albumin	2 (7.1)
Hospital stay ≤10 days	1 (3.6)
Hospital stay 11–20 days	12 (42.9)
Hospital stay >20 days	15 (53.6)
Mortality	1 (3.6)

Table 4 outlines the therapeutic approaches used for the management of postoperative ascites and their associated outcomes. Diuretic therapy constituted the most commonly employed intervention, either alone or in combination with intravenous albumin. Therapeutic paracentesis was required in a substantial proportion of patients, reflecting the high prevalence of moderate-to-large volume ascites. Hospital stay was prolonged in most patients, with more than half requiring hospitalization beyond 20 days. Despite significant morbidity, overall mortality remained low at 3.6%, indicating favorable short-term survival following appropriate management.

Table 5: Postoperative Morbidity Associated with De Novo Ascites

Complication	n (%)
Wound infection	6 (21.4)
Surgical site infection	4 (14.3)
Sepsis	4 (14.3)
Anastomotic leak	4 (14.3)
Acute kidney injury	2 (7.1)
Hypoalbuminaemia	2 (7.1)
Pleural effusion	1 (3.6)
Prolonged ileus	1 (3.6)
No complication	4 (14.3)

Table 5 demonstrates the spectrum of postoperative complications observed in patients who developed ascites. Wound infection was the most common complication, followed by surgical site infection, sepsis, and anastomotic leak. Renal dysfunction, hypoalbuminaemia, pleural effusion, and prolonged ileus occurred less frequently. Only a small subset of patients experienced an uncomplicated postoperative course. These findings emphasize the strong association between postoperative ascites and increased postoperative morbidity.

Table 6: Correlation Between Ascitic Fluid Volume and Clinical-Biochemical Parameters

Variable	Spearman r	p-value
SGPT	0.468	0.012*
Blood Urea	0.427	0.023*
LDH	-0.448	0.017*
Age	0.287	0.139
Duration of surgery	0.161	0.414
Blood loss	0.045	0.821
Albumin	0.188	0.338
SAAG	-0.308	0.111
Hospital stay	0.197	0.316

Table 6 presents the correlation analysis between ascitic fluid volume and selected clinical and laboratory variables. Significant positive correlations were identified between ascitic fluid volume and serum SGPT levels as well as blood urea concentrations, suggesting that hepatic stress and impaired metabolic status may contribute to greater fluid accumulation. Conversely, LDH demonstrated a significant negative correlation with ascitic volume. No significant associations were observed between ascitic volume and age, operative duration, intraoperative blood loss, serum albumin, SAAG, or length of hospital stay. These findings indicate that biochemical markers may provide useful adjunctive information in assessing the severity of postoperative ascites.

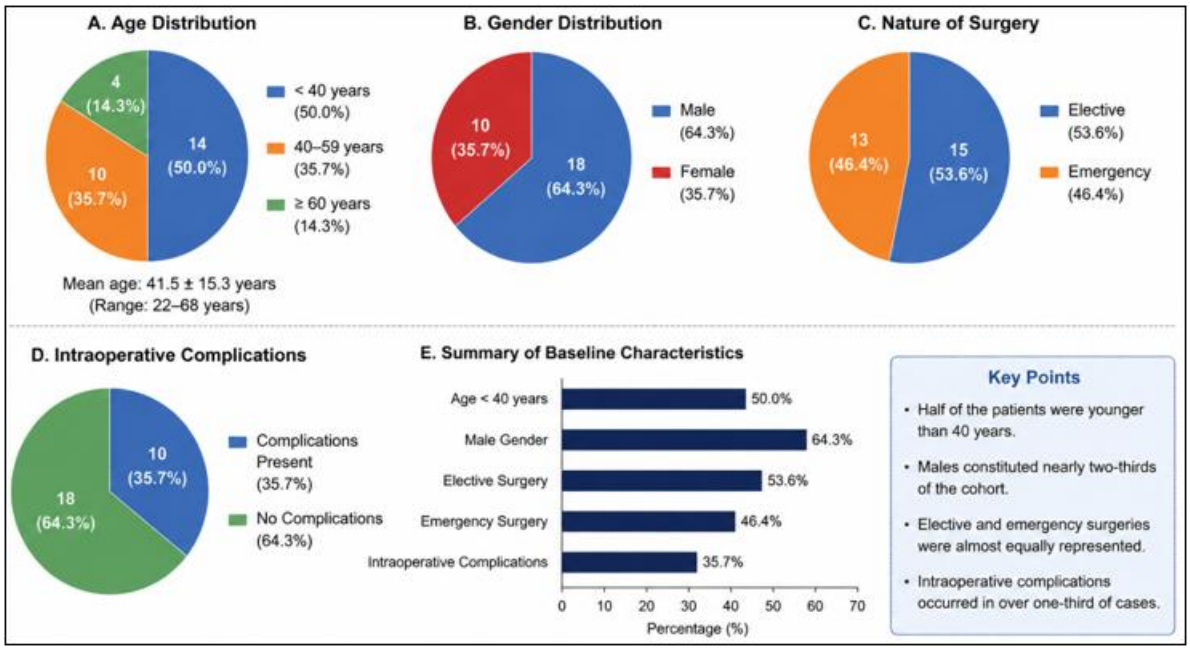

Figure 1: Demographic profile and operative characteristics of patients developing de novo postoperative ascites following major abdominal surgery

Figure 1 illustrates the baseline demographic and operative characteristics of the 28 patients who developed de novo postoperative ascites following major abdominal surgery. Half of the study population (50.0%) was younger than 40 years, while 35.7% were aged between 40 and 59 years and 14.3% were aged 60 years or older, with a mean age of 41.5 ± 15.3 years. Male patients constituted the majority of the cohort (64.3%), whereas females accounted for 35.7%. Regarding operative characteristics, elective procedures represented 53.6% of surgeries, while emergency operations accounted for 46.4%. Intraoperative complications were documented in 35.7% of patients, whereas 64.3% underwent surgery without reported intraoperative adverse events. Collectively, these findings demonstrate that postoperative ascites occurred across a broad demographic spectrum and was associated with both elective and emergency abdominal procedures, with more than one-third of cases experiencing intraoperative complications.

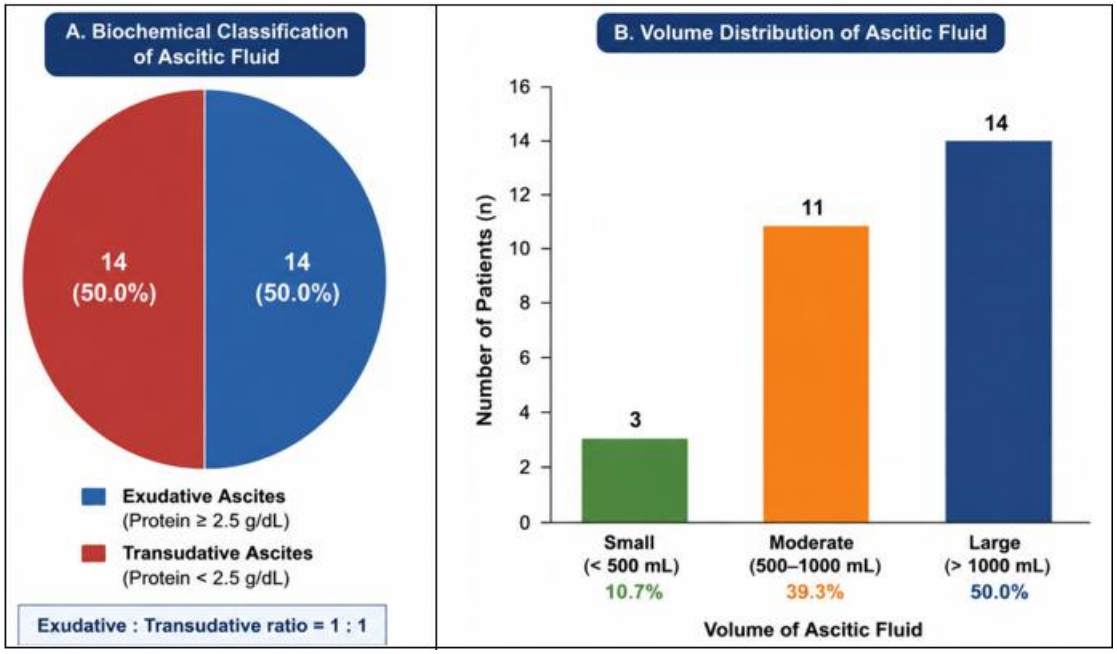

Figure 2: Biochemical Classification and Volume Distribution of Postoperative Ascitic Fluid

Figure 2. Biochemical classification and volume distribution of postoperative ascitic fluid in patients developing de novo postoperative ascites following major abdominal surgery (N = 28). Exudative and transudative ascites were equally represented (50.0% each). Large-volume ascites (>1000 mL) was the predominant presentation, followed by moderate-volume (500–1000 mL) and small-volume (<500 mL) fluid collections. Data are presented as frequencies and percentages.

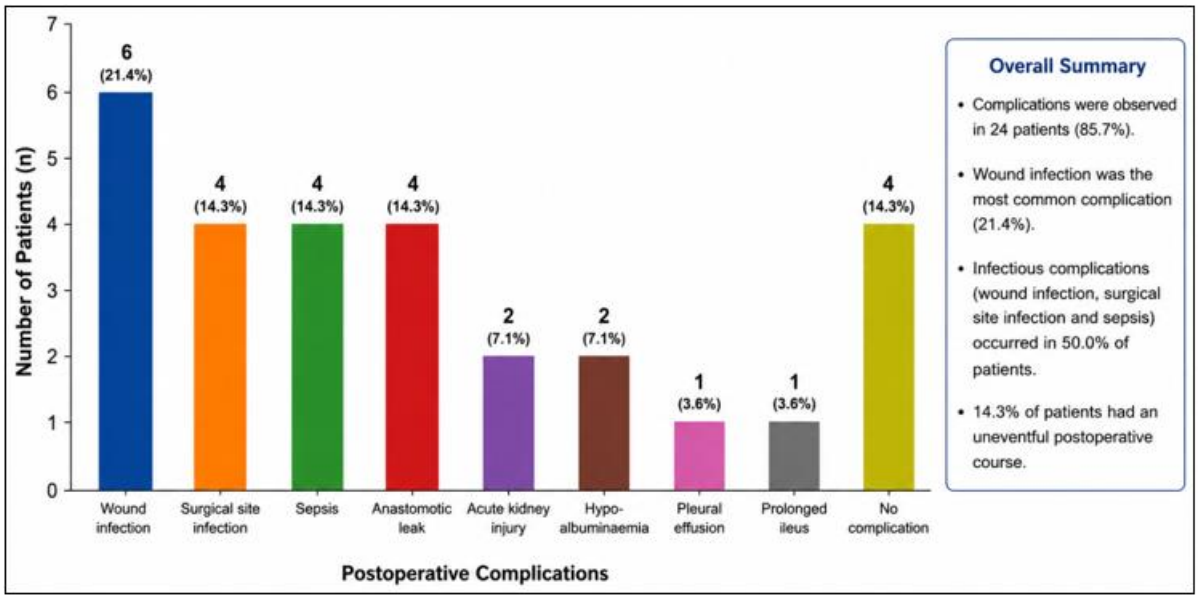

Figure 3: Frequency and Distribution of Postoperative Complications Among Patients with De Novo Postoperative Ascites

Figure 3. Frequency and distribution of postoperative complications among patients with de novo postoperative ascites following major abdominal surgery (N = 28). Wound infection was the most common complication (21.4%), followed by surgical site infection, sepsis, and anastomotic leak (14.3% each). Acute kidney injury and hypoalbuminaemia occurred in 7.1% of patients, while pleural effusion and prolonged ileus were observed in 3.6% each. Complications were present in 85.7% of patients, whereas 14.3% experienced an uncomplicated postoperative course. Data are presented as frequencies and percentages.

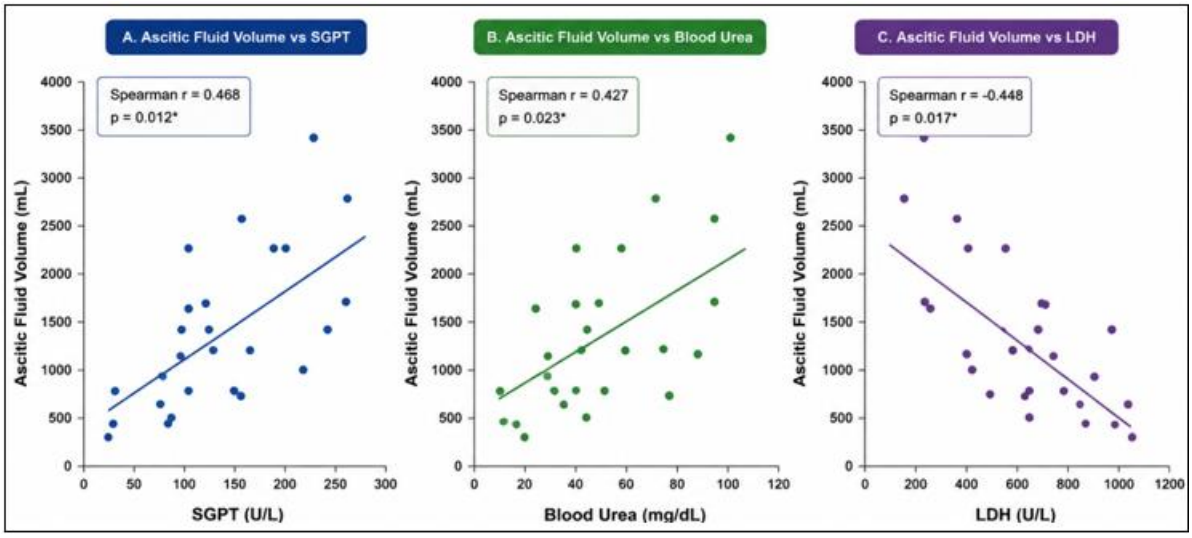

Figure 4: Correlation of Ascitic Fluid Volume with Significant Biochemical Parameters, Including SGPT, Blood Urea, and LDH

Figure 4: Correlation between ascitic fluid volume and significant biochemical parameters in patients with de novo postoperative ascites following major abdominal surgery (N = 28). Scatterplot analysis demonstrated significant positive correlations between ascitic fluid volume and serum SGPT levels ($r = 0.468$, $p = 0.012$) and blood urea concentration ($r = 0.427$, $p = 0.023$). A significant negative correlation was observed between ascitic fluid volume and serum LDH levels ($r = -0.448$, $p = 0.017$). Correlation coefficients were calculated using Spearman's rank correlation analysis. Statistical significance was defined as $p < 0.05$.

4. Discussion

This prospective observational study evaluated the etiological spectrum, perioperative determinants, and clinical outcomes of de novo postoperative ascites following major abdominal surgery in patients without pre-existing cirrhosis or chronic liver disease. Several important observations emerged. First, postoperative ascites occurred across a broad range of abdominal procedures, with hepatopancreatobiliary and colorectal surgeries accounting for the largest proportion of cases. Second, both exudative and transudative ascites were equally represented, highlighting the multifactorial nature of postoperative fluid accumulation. Third, postoperative ascites was associated

with substantial morbidity, including wound infection, sepsis, and anastomotic leak, and frequently resulted in prolonged hospitalization. Finally, ascitic fluid volume demonstrated significant associations with SGPT, blood urea, and LDH levels, suggesting that biochemical markers may reflect disease severity and postoperative physiological stress.

The development of postoperative ascites is complex and differs fundamentally from the mechanisms underlying cirrhosis-associated ascites. Rather than arising from chronic portal hypertension and hepatic insufficiency, postoperative ascites reflects an interplay of surgical trauma, lymphatic injury, inflammatory responses, transient portal hemodynamic alterations, and disturbances in vascular permeability [28,29]. The relative contribution of each mechanism varies according to the surgical procedure performed and the extent of tissue manipulation.

Our finding of an equal distribution of exudative and transudative fluid supports the concept that postoperative ascites represents a heterogeneous clinical entity rather than a single pathophysiological process. Similar observations have been reported following major hepatobiliary and pancreatic procedures where multiple mechanisms frequently coexist [30].

Lymphatic injury is one of the most recognized causes of postoperative ascites, particularly following extensive retroperitoneal dissection, lymphadenectomy, pancreatic surgery, and major oncological procedures [31]. Surgical disruption of lymphatic channels may result in persistent leakage of protein-rich lymphatic fluid into the peritoneal cavity, producing either chylous or non-chylous ascites. Experimental studies have demonstrated that postoperative lymphatic leakage may continue for several weeks before collateral lymphatic channels develop and restore normal drainage [32].

Although specific lymphangiographic evaluation was not performed in the present study, the occurrence of large-volume ascites in 50% of patients and the predominance of complex abdominal procedures suggest that lymphatic disruption likely contributed to fluid accumulation in a substantial proportion of cases.

Transient alterations in portal hemodynamics may also contribute to postoperative ascites. Following hepatic resection, portal hyperperfusion of the remnant liver can increase sinusoidal pressure and promote transudation of fluid into the peritoneal cavity [33]. Furthermore, temporary impairment of hepatic synthetic function may reduce plasma oncotic pressure, facilitating third-space fluid accumulation [34].

The significant positive correlation observed between ascitic fluid volume and SGPT levels in the present study supports a potential role for postoperative hepatocellular stress in the pathogenesis of ascites. Similar observations have been reported in patients undergoing major liver resection, where postoperative liver dysfunction frequently precedes the development of clinically significant ascites [35].

Surgical trauma initiates a cascade of inflammatory events characterized by cytokine release, increased capillary permeability, and recruitment of inflammatory cells into the peritoneal cavity [36]. These responses facilitate the formation of protein-rich exudative fluid, particularly following extensive dissection, bowel manipulation, and contamination of the operative field.

The high proportion of exudative ascites observed in our cohort may reflect these inflammatory processes. Furthermore, the relatively high incidence of postoperative infectious complications, including wound infection and sepsis, suggests that inflammatory activation may persist beyond the immediate postoperative period and contribute to ongoing fluid production.

Complex surgical procedures are associated with prolonged operative duration, greater blood loss, extensive tissue dissection, and increased physiological stress, all of which may promote postoperative fluid accumulation [37]. In the present study, intraoperative complications occurred in more than one-third of patients, supporting the hypothesis that operative complexity contributes to postoperative ascites.

Previous studies have demonstrated that major resections, vascular reconstructions, and extensive lymphadenectomy significantly increase the risk of postoperative fluid collections and ascites [38]. Although duration of surgery and blood loss were not significantly correlated with ascitic volume in our cohort, the limited sample size may have reduced the ability to detect such associations.

The findings of this study are consistent with reports from hepatobiliary surgery, where postoperative ascites is a recognized complication after hepatectomy and complex biliary procedures. Ishizawa et al. reported that postoperative ascites was associated with impaired liver function, increased postoperative morbidity, and prolonged hospitalization following liver resection [14]. Similarly, Golse and colleagues demonstrated that postoperative ascites often represents an early manifestation of post-hepatectomy physiological dysfunction [21]. Our observation that large-volume ascites was common and associated with prolonged hospitalization aligns closely with these previous reports.

The literature on postoperative ascites after colorectal surgery is comparatively limited. Nevertheless, several studies have suggested that extensive bowel manipulation, anastomotic leakage, intra-abdominal infection, and inflammatory exudation may contribute to postoperative fluid accumulation [39]. The relatively high representation of colorectal procedures in the present cohort supports the notion that postoperative ascites is not restricted to hepatobiliary surgery and may occur following major colorectal resections as well. The observed association between ascites and anastomotic leakage further reinforces the importance of considering occult surgical complications in patients presenting with postoperative fluid accumulation.

Pancreatic surgery is frequently associated with postoperative ascites because of lymphatic disruption, pancreatic fistula formation, and extensive retroperitoneal dissection [40]. Studies evaluating chylous ascites after

pancreatic resection have reported incidences ranging from 3% to 11%, particularly following radical lymphadenectomy [41]. Our findings are broadly consistent with the pancreatic surgery literature, particularly regarding the occurrence of large-volume ascites and the frequent need for repeated therapeutic interventions such as paracentesis and diuretic therapy.

The present study highlights the need for early recognition and systematic evaluation of postoperative ascites. Fluid analysis remains essential for distinguishing transudative, exudative, biliary, pancreatic, and lymphatic causes of ascites, thereby guiding appropriate management [42]. Furthermore, patients with elevated SGPT and blood urea levels may warrant closer surveillance because these markers were significantly associated with greater fluid accumulation. Given the substantial morbidity observed in this study, postoperative ascites should not be regarded as a benign postoperative finding but rather as a potential marker of underlying physiological dysfunction or occult surgical complications.

Several preventive measures may reduce the incidence and severity of postoperative ascites. These include meticulous surgical technique, careful ligation of lymphatic channels, preservation of hepatic venous outflow, optimization of perioperative fluid management, and early detection of postoperative leaks [43]. Enhanced Recovery After Surgery (ERAS) protocols may also contribute to reduced postoperative fluid overload and improved recovery [44].

5. Conclusion

De novo postoperative ascites represents a clinically important yet underrecognized complication following major abdominal surgery in patients without pre-existing cirrhosis or chronic liver disease. The present study demonstrates that postoperative ascites occurs across a broad spectrum of abdominal procedures, particularly hepatopancreatobiliary and colorectal surgeries, and is associated with significant postoperative morbidity and prolonged hospitalization. The equal distribution of exudative and transudative ascites observed in this cohort highlights the multifactorial nature of postoperative fluid accumulation, involving lymphatic disruption, transient hepatic dysfunction, inflammatory peritoneal responses, and alterations in portal hemodynamics.

Conflict of Interest

The authors declare that they have no conflicts of interest regarding the publication of this manuscript. No financial, commercial, institutional, personal relationships existed that could have influenced the workreported in this study.

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