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# Perforated Colonic Peritonitis As A Source Of Surgical Sepsis

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**Abstract:** Background. Perforated colonic peritonitis remains one of the most severe conditions in emergency abdominal surgery and is frequently complicated by surgical sepsis. Despite radical elimination of the perforation source, systemic deterioration may develop rapidly or follow a subacute course, particularly in patients with significant comorbidity.

Methods. A retrospective cohort study included 118 patients operated on for perforated colonic peritonitis of malignant and non-malignant etiology. Surgical sepsis was defined according to Sepsis-3 criteria. Clinical course, comorbid conditions, surgical strategy, need for intensive care, progression to septic shock and multiple organ failure, and in-hospital mortality were analyzed.

Results. Surgical sepsis was identified in a substantial proportion of patients either at admission or during early postoperative period. Patients with pronounced comorbidity more often demonstrated fulminant septic progression with early development of septic shock, multiple organ failure, and high mortality, despite radical surgical source control. In patients without severe comorbidities, septic complications frequently evolved in a subacute manner, with transient postoperative stabilization that could mask ongoing systemic infection. Requirement for intensive care treatment and progression to septic shock were strongly associated with adverse outcomes.

Conclusion. Perforated colonic peritonitis should be regarded as a condition with an inherent risk of surgical sepsis regardless of the apparent adequacy of operative management. Apparent postoperative stabilization does not exclude systemic progression. Continuous assessment for septic manifestations and timely escalation of anti-septic therapy are essential to improve outcomes in this high-risk patient population.

**Keywords:** Perforated colonic peritonitis; surgical

sepsis; comorbidity; septic shock; mortality.

**1. Introduction:** Perforated colonic peritonitis remains one of the most severe conditions in emergency abdominal surgery and is associated with high rates of morbidity and mortality. The combination of fecal contamination, bacterial translocation, and systemic inflammatory response creates a clinical scenario in which rapid deterioration may occur despite prompt surgical intervention [1]. Advances in operative techniques and perioperative care have improved early survival; however, overall outcomes remain strongly dependent on systemic complications rather than on local control alone.

A major determinant of outcome in perforated colonic peritonitis is the development of surgical sepsis. According to contemporary concepts, sepsis represents a life-threatening organ dysfunction caused by a dysregulated host response to infection, shifting the clinical focus from local pathology to systemic failure [2]. In the setting of colonic perforation, septic progression may occur early, often before adequate physiological compensation can be achieved, or may evolve insidiously during the postoperative period.

Radical surgical source control is universally regarded as the cornerstone of treatment for perforated peritonitis. Procedures such as Hartmann's operation, exteriorization with stoma formation, primary anastomosis, or damage control surgery aim to eliminate the infectious focus and limit ongoing contamination [3]. Nevertheless, even technically adequate operations do not uniformly prevent septic deterioration, suggesting that factors beyond surgical radicality contribute to adverse outcomes.

Clinical experience indicates that comorbid conditions significantly influence the septic trajectory in patients with perforated colonic peritonitis. Diabetes mellitus, chronic kidney disease, cardiovascular pathology, malignancy, and states of immunosuppression are associated with impaired host response and reduced tolerance to systemic inflammation. In such patients, septic progression may be fulminant, with rapid transition to septic shock and multiple organ failure shortly after surgery [4]. Conversely, in patients without pronounced comorbidity, sepsis may develop in a subacute manner, characterized by transient postoperative stabilization that may obscure ongoing systemic infection.

This variability in septic presentation complicates postoperative assessment and may delay escalation of intensive anti-septic therapy. Apparent clinical improvement, particularly in the early postoperative period, does not necessarily reflect resolution of systemic risk. Several studies have emphasized that delayed recognition of sepsis in abdominal infections is associated with increased mortality, underscoring the need for continuous systemic evaluation alongside local surgical assessment [5,6].

The aim of the present study was to analyze the clinical course and outcomes of patients with perforated colonic peritonitis, with particular emphasis on the role of surgical sepsis, comorbidity, and postoperative systemic deterioration. By comparing patients with and without sepsis according to Sepsis-3 criteria, this study seeks to highlight patterns of septic progression that are relevant to everyday surgical practice.

## 2. Methods

Study design and patient population. A retrospective cohort study was performed including 118 consecutive patients who underwent emergency surgery for perforated colonic peritonitis. Medical records were reviewed over a defined study period. Perforation of the colon was confirmed intraoperatively in all cases. Patients with perforation of the small intestine or gastroduodenal region were excluded from the analysis.

Definition of surgical sepsis. Surgical sepsis was defined according to the Sepsis-3 criteria as infection-associated organ dysfunction, identified by an increase in the Sequential Organ Failure Assessment (SOFA) score of two points or more from baseline [2]. Septic shock was diagnosed in patients with persistent hypotension requiring vasopressor therapy to maintain a mean arterial pressure of at least 65 mmHg and serum lactate levels exceeding 2 mmol/L despite adequate fluid resuscitation.

**Etiology of perforation and comorbid conditions.**

The etiology of colonic perforation included malignant obstruction, diverticular disease, ischemic colitis, and other non-malignant causes. Comorbid conditions were identified based on documented medical history and included diabetes mellitus, chronic kidney disease, cardiovascular disease, chronic liver disease, malignancy, and states of immunosuppression. The presence of multiple comorbid conditions was specifically noted.

**Surgical management**. All patients underwent urgent surgical intervention aimed at elimination of the perforation source and control of intra-abdominal

contamination. Surgical strategies included Hartmann's procedure, exteriorization with stoma formation, primary resection with anastomosis, and damage control surgery with planned re-exploration. The choice of surgical technique was determined by intraoperative findings, patient physiological status, and surgeon judgment.

**Perioperative and intensive care management**. Empirical broad-spectrum antimicrobial therapy was initiated immediately after diagnosis and adjusted according to microbiological results when available. Postoperative management followed institutional protocols and included close monitoring of hemodynamic status, laboratory parameters, and organ function. Admission to the intensive care unit was based on the presence of organ dysfunction, hemodynamic instability, or progression to septic shock.

**Outcomes**. The primary outcome of the study was in-hospital mortality. Secondary outcomes included progression to septic shock, development of multiple organ failure, requirement for intensive care unit treatment, and the need for relaparotomy or staged surgical management.

**Statistical analysis**. Data analysis was conducted using standard statistical methods appropriate for retrospective clinical studies. Continuous variables were expressed as means with standard deviations or medians with interquartile ranges, depending on distribution. Categorical variables were reported as absolute numbers and percentages. Comparisons between patients with and without surgical sepsis were performed using appropriate comparative tests, with statistical significance defined as $p < 0.05$.

## 3. Results

**Patient characteristics and etiology of perforation**. A total of 118 patients with intraoperatively confirmed perforated colonic peritonitis were included in the analysis. Colonic perforation was most commonly associated with malignant obstruction and diverticular disease, followed by ischemic colitis and other non-malignant causes. Surgical sepsis according to Sepsis-3 criteria was identified in a substantial proportion of patients either at admission or during the early postoperative period.

Patients with surgical sepsis more frequently presented with significant comorbidity. Diabetes mellitus, cardiovascular disease, chronic kidney disease, and oncological pathology were common in this group, often occurring in combination. Patients without sepsis at presentation generally demonstrated fewer comorbid conditions, although absence of sepsis did not preclude subsequent systemic deterioration during postoperative follow-up.

**Clinical course and septic progression**. The clinical course of perforated colonic peritonitis varied markedly depending on septic involvement. In patients with pronounced comorbidity, sepsis often followed a fulminant course, characterized by rapid progression to septic shock and early development of multiple organ failure shortly after surgery. This pattern was observed despite timely operative elimination of the perforation source and initiation of intensive care measures.

In contrast, patients without severe comorbid conditions more commonly exhibited a subacute septic course. These patients experienced transient postoperative stabilization, sometimes accompanied by normalization of temperature and hemodynamic parameters. Such dynamics could obscure ongoing systemic infection and precede sudden clinical deterioration with manifestation of overt sepsis or septic shock later in the postoperative period.

**Surgical management and intensive care requirements**. All patients underwent emergency surgical intervention aimed at source control. Hartmann's procedure and resection with stoma formation were the most frequently performed operations, followed by primary resection with anastomosis in selected cases. Damage control surgery with planned re-exploration was applied in physiologically unstable patients.

Patients with surgical sepsis required a significantly higher level of postoperative support. Admission to the intensive care unit was markedly more frequent in the septic group, particularly among those who progressed to septic shock or multiple organ failure. Relaparotomy or staged surgical management was also more common in septic patients, reflecting both the severity of intra-abdominal contamination and systemic instability.

**Outcomes**. In-hospital mortality was substantially higher among patients with surgical sepsis compared with those without systemic involvement. Mortality was particularly elevated in patients who developed septic shock and multiple organ failure. Requirement for intensive care unit treatment was strongly associated with adverse outcomes.

Patients without sepsis demonstrated lower mortality and more favorable postoperative courses, although

isolated cases of late systemic deterioration and fatal outcome were observed. Comparative data on comorbidity, surgical management, septic progression, intensive care requirements, and outcomes are summarized in Table 1 and Table 2.

**Table 1.**

**Baseline characteristics, etiology of perforation, and comorbidity**

| Variable | Sepsis group (n=52) | Non-sepsis group (n=66) |
|---|---|---|
| Age, years, mean ± SD | 61.3±10.8 | 52.6±12.4 |
| Male sex, n (%) | 34 (65.4) | 41 (62.1) |
| Malignant perforation, n (%) | 27 (51.9) | 19 (28.8) |
| Diverticular disease, n (%) | 15 (28.8) | 29 (43.9) |
| Ischemic colitis, n (%) | 6 (11.5) | 9 (13.6) |
| Other causes, n (%) | 4 (7.8) | 9 (13.6) |
| Diabetes mellitus, n (%) | 26 (50.0) | 18 (27.3) |
| Cardiovascular disease, n (%) | 31 (59.6) | 26 (39.4) |
| Chronic kidney disease, n (%) | 14 (26.9) | 7 (10.6) |
| Malignancy (any), n (%) | 29 (55.8) | 22 (33.3) |
| ≥2 comorbid conditions, n (%) | 28 (53.8) | 16 (24.2) |
| Sepsis at admission, n (%) | 37 (71.2) | 0 |
| Sepsis developed postoperatively, n (%) | 15 (28.8) | 0 |

**Table 2.**

**Surgical management, intensive care requirements, and outcomes**

| Variable | Sepsis group (n=52) | Non-sepsis group (n=66) |
|---|---|---|
| Hartmann’s procedure, n (%) | 31 (59.6) | 28 (42.4) |
| Resection with stoma, n (%) | 14 (26.9) | 21 (31.8) |
| Primary anastomosis, n (%) | 4 (7.7) | 13 (19.7) |
| Damage control surgery, n (%) | 3 (5.8) | 4 (6.1) |
| Relaparotomy / staged surgery, n (%) | 21 (40.4) | 11 (16.7) |
| ICU admission, n (%) | 39 (75.0) | 18 (27.3) |
| Progression to septic shock, n (%) | 23 (44.2) | 0 |
| Multiple organ failure, n (%) | 20 (38.5) | 3 (4.5) |
| Length of hospital stay, days, median (IQR) | 19 (13–31) | 11 (8–17) |
| In-hospital mortality, n (%) | 17 (32.7) | 4 (6.1) |

**4. Discussion**

The present study demonstrates that perforated colonic peritonitis is consistently associated with a high risk of surgical sepsis, the clinical course of which remains heterogeneous and often unpredictable. Even after technically adequate and timely surgical elimination of the perforation source, systemic deterioration may develop early or evolve in a delayed and deceptive manner. These findings emphasize that,

in this setting, surgical success at the local level does not necessarily translate into systemic stabilization [1].

A key observation of this study concerns the role of comorbidity in shaping the septic trajectory. Patients with diabetes mellitus, chronic kidney disease, cardiovascular pathology, malignancy, and other conditions associated with impaired physiological reserve more frequently demonstrated a fulminant septic course. In these patients, rapid progression to septic shock and multiple organ failure occurred shortly after surgery, despite radical source control and initiation of intensive care measures. This pattern is consistent with previous reports indicating that reduced host tolerance to systemic inflammation plays a decisive role in outcomes following colonic perforation [3,4].

At the same time, the study highlights a clinically important subgroup of patients without pronounced comorbidity in whom septic complications evolved in a subacute manner. Transient postoperative stabilization, including temporary normalization of temperature and hemodynamic parameters, was observed in some cases and could create a misleading impression of recovery. Similar observations have been described in abdominal sepsis, where delayed recognition of systemic infection has been associated with increased mortality [5]. This finding underscores the limitation of relying on early postoperative dynamics alone when assessing the adequacy of treatment.

The high rate of intensive care unit admission and the strong association between septic shock, multiple organ failure, and mortality observed in this cohort further support contemporary concepts of sepsis as a syndrome of systemic organ dysfunction rather than a localized infectious process [2]. The need for relaparotomy and staged surgical management in septic patients should be interpreted not merely as a technical issue but as a marker of ongoing systemic instability and disease severity.

From a practical perspective, the results of this study reinforce the need for sustained vigilance in patients operated on for perforated colonic peritonitis. Continuous assessment for signs of organ dysfunction, early involvement of intensive care specialists, and timely escalation of anti-septic therapy should be considered integral components of management. Apparent postoperative improvement should not delay reassessment of systemic status, particularly in patients with significant comorbidity or extensive peritoneal contamination [6].

Several limitations of this study should be acknowledged. Its retrospective design limits control over confounding variables, and the single-center setting may affect generalizability. Microbiological characteristics and detailed antimicrobial regimens were not analyzed, which may influence septic progression. Nevertheless, the relatively homogeneous surgical pathology and clear stratification according to septic involvement provide clinically relevant insight into the systemic risks associated with perforated colonic peritonitis.

## 5. Conclusion

Perforated colonic peritonitis should be regarded as a surgical condition with a persistently high risk of systemic septic complications, irrespective of the apparent adequacy of operative source control. Surgical sepsis may develop rapidly with early progression to septic shock and multiple organ failure in patients with significant comorbidity, while in less burdened patients it may follow a subacute and misleading course with transient postoperative stabilization. These patterns highlight the limitation of relying solely on local or early postoperative dynamics when assessing treatment success. Continuous systemic evaluation, early recognition of organ dysfunction, and timely escalation of comprehensive anti-septic therapy are essential components of care to reduce mortality in this high-risk patient population.

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