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Acute abdomen: postmortem analysis of 31 cases in the Emergency Department – a monocentric retrospective study

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Abstract

Acute abdominal emergencies remain a leading cause of mortality in the emergency setting, often resulting in death before surgical intervention can be performed. During the coronavirus disease 2019 (COVID-19) pandemic, these cases were further complicated by thrombotic and inflammatory mechanisms, with a notable increase in mesenteric ischemia. We conducted a retrospective monocentric study of 31 postmortem cases of acute abdomen occurring between January 1, 2019, and December 31, 2023, analyzing demographic characteristics, clinical presentation, laboratory parameters, imaging, and autopsy findings. Among 69 total deaths in the Emergency Department during this period, 31 were attributed to acute abdominal pathology, the majority involving elderly, septic patients with perforation or ischemia. Nine deaths from mesenteric ischemia occurred during the COVID-19 period, emphasizing severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2)-associated hypercoagulability. Other fatal cases included perforated ulcers, ruptured hepatic or pancreatic tumors, and advanced neoplasms with secondary peritonitis. Laboratory markers such as elevated lactate, leukocytosis, and coagulopathy corresponded with postmortem evidence of intestinal necrosis, thrombosis, or peritoneal contamination. Although urban patients predominated, rural patients frequently presented with advanced sepsis at admission. These findings highlight that acute abdominal emergencies carry high mortality and underscore the critical importance of early recognition, rapid triage, and multidisciplinary management. Postmortem analysis provides essential insights into fatal mechanisms and supports the refinement of diagnostic and treatment protocols to improve outcomes.

Keywords: acute abdomen, COVID-19, necropsy, mesenteric ischemia, peritonitis, emergency.

Introduction

Acute mesenteric ischemia (AMI) represents one of the most severe abdominal emergencies, characterized by sudden interruption of intestinal blood flow that leads to bowel necrosis and high mortality if diagnosis and treatment are delayed [1]. Despite advances in imaging and critical care, mortality rates remain between 40–80%, largely due to nonspecific clinical presentation and diagnostic delays [2, 3].

The coronavirus disease 2019 (COVID-19) pandemic introduced new challenges in emergency abdominal surgery. Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection has been associated with a hypercoagulable state, endothelial injury, and microthrombi formation, mechanisms that may precipitate AMI in predisposed patients [4–6]. Recent clinical studies reported cases of mesenteric thrombosis or non-occlusive ischemia in COVID-19 patients, with outcomes significantly worse than in non-infected individuals [7, 8].

In parallel, the pandemic context indirectly influenced the management of abdominal emergencies. Lockdowns, patient fear of hospital exposure, and overwhelmed healthcare systems led to delayed presentations and advanced disease stages at admission [9, 10]. Consequently, the proportion of patients requiring bowel resection, intensive care, or with postoperative sepsis markedly increased [11].

Beyond the pandemic impact, AMI continues to pose diagnostic and therapeutic challenges. Computed tomography angiography (CTA) remains the "gold standard" for early diagnosis, enabling differentiation between embolic, thrombotic, and non-occlusive forms [12, 13]. Nevertheless, many patients present with irreversible bowel ischemia at surgery, emphasizing the need for rapid decision-making and multidisciplinary coordination [14].

Therefore, this study analyzes the clinical and paraclinical characteristics, intraoperative findings, and postoperative outcomes of patients with AMI in a tertiary Emergency Hospital setting, emphasizing mortality predictors and

correlations between systemic inflammation markers and survival [15].

Aim

Our paper aimed to perform a postmortem clinico-pathological analysis of fatal acute abdomen cases over a five-year period (2019–2023), determining epidemiological patterns, underlying mechanisms, and the impact of COVID-19-related thrombosis on mortality.

Patients, Materials and Methods

This monocentric retrospective study was conducted at the Emergency Department of the Emergency City Hospital, Timişoara, Romania, between January 1, 2019, and December 31, 2023, including the COVID-19 pandemic period. A total of 69 patients who died in the Emergency Department during this period were reviewed. Of these, 31 cases were attributed to acute surgical abdomen, occurring before hospitalization or surgical intervention due to the severity of presentation.

The study population included both sexes, aged 45–91 years, from urban and rural environments. Diagnoses encompassed AMI, perforated gastric and duodenal ulcers, pancreatic and hepatic tumors with perforation or rupture, colonic neoplasms, traumatic abdominal injuries, and complicated hernias. Clinical parameters collected included:

- Demographics (age, sex, urban/rural residence);
- Presenting symptoms (abdominal pain, distension, vomiting, hematemesis, melena);
- Vital signs at admission (blood pressure, heart rate, temperature);
- Laboratory investigations [complete blood count (CBC), C-reactive protein (CRP), liver and renal function, coagulation profile, lactate];
- Imaging studies [abdominal X-ray, ultrasonography (US), contrast-enhanced computed tomography (CT)];
- COVID-19 status [polymerase chain reaction (PCR) confirmed or epidemiological association].

Additionally, postmortem findings were recorded, including perforation sites, tumor invasion, hemoperitoneum, peritonitis type, and evidence of bowel ischemia or necrosis. During the autopsy, the abdominal cavity and all affected organs were examined, and representative tissue samples were obtained from ischemic or necrotic bowel segments, mesenteric vessels, perforation sites of gastric or duodenal origin, neoplastic masses showing rupture or hemorrhage, as well as hepatic abscess cavities. All specimens were fixed in 10% neutral buffered formalin and processed via standard paraffin embedding to obtain slides for microscopic evaluation.

The *inclusion criteria* comprised patients who presented with signs of acute abdomen and were subsequently confirmed postmortem to have an intra-abdominal catastrophic event as the primary cause of death. *Exclusion criteria* included patients who died of non-abdominal causes, traumatic injuries outside the abdominal cavity, or those whose records lacked sufficient clinical or postmortem data for comprehensive evaluation.

For each case, data collection was structured around three principal domains: clinical presentation, laboratory and imaging results, and autopsy findings. Clinical information included demographic characteristics (age, sex, and place of residence), onset and duration of symptoms, vital signs on admission (blood pressure, pulse, respiratory rate, temperature), and the presence of systemic inflammatory response or shock. Laboratory parameters of interest included CBC, CRP, liver and renal function markers, lactate levels, and coagulation profiles. These results were used to assess the severity of systemic involvement and to identify potential biochemical predictors of fatal outcomes.

Imaging studies were analyzed where available, primarily contrast-enhanced CT, abdominal US, and plain abdominal radiography. These were evaluated for radiological evidence of bowel ischemia, free intraperitoneal air, fluid accumulation, abscesses, or signs of vascular thrombosis. In particular, during the COVID-19 pandemic period, additional attention was given to identifying mesenteric thrombosis and its correlation with systemic coagulopathy or COVID-19-associated hyperinflammatory states.

Each case underwent a standardized autopsy protocol focused on identifying the anatomical substrate of the acute abdomen and the sequence of events leading to death. The macroscopic examination assessed the morphology of affected bowel segments, the extent of peritoneal contamination, the characteristics of peritoneal fluid, and the presence of vascular obstruction, hemorrhage, or organ rupture. Particular attention was given to determining whether the fatal event was driven primarily by ischemia, perforation, infection, neoplastic invasion, or traumatic injury.

Microscopic analysis was performed on Hematoxylin–Eosin (HE)-stained sections derived from the formalin-fixed samples collected during the autopsy. Histological assessment focused on characterizing ischemic patterns, the depth and type of necrosis, the inflammatory response, the presence of septic vasculitis or thrombosis, and the structural alterations in tumoral lesions, including necrotic foci and invasion of adjacent tissues. All findings were integrated and correlated with the clinical context to reconstruct the mechanism of death.

All cases were subsequently categorized according to the dominant pathological process – ischemic, perforative, neoplastic, or infectious. COVID-19 infection was confirmed either by PCR testing or through epidemiological context when PCR results were unavailable. The COVID-19-positive subgroup was analyzed separately to evaluate potential associations between viral infection and the occurrence of mesenteric ischemia, acknowledging the prothrombotic nature of the disease.

Statistical analysis was primarily descriptive. Quantitative variables were expressed as mean ± standard deviation, while categorical variables were expressed as absolute numbers and percentages. The relationships between clinical severity (*e.g.*, shock, sepsis), laboratory abnormalities (*e.g.*, leukocytosis, elevated lactate, coagulopathy), and postmortem findings were assessed using correlation coefficients. The analyses were performed with Statistical Package for the Social Sciences (SPSS) version 26.0 (IBM Corp., Armonk, NY, USA). Although inferential testing was not the primary focus due to the limited sample size, observed trends and patterns were interpreted in the context of pathophysiological plausibility and supported by relevant literature.

The study protocol was conducted in accordance with

the ethical principles of the Declaration of Helsinki (2013 revision). Given the retrospective and postmortem design, formal patient consent was waived, and approval was obtained from the institutional Ethics Committee (No. E-4676/11.11.2025).

Results

A total of 31 deaths caused by acute surgical abdomen were recorded in the Emergency Department over the five-year study period (2019–2023), representing 44.9% of all deaths (*n*=69) during the same interval. The mean age of the patients was 71.2±12.4 years, ranging from 45 to 91 years, with a slight predominance of males (17 males *vs.* 14 females) (Figures 1 and 2). Most patients (65%) originated from urban areas, while 35% were from rural communities, reflecting both demographic access patterns and healthcare disparities in emergency presentation timing.

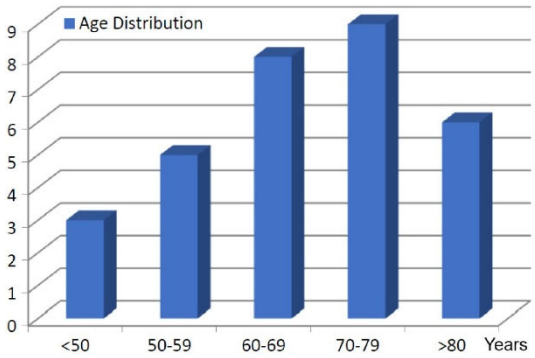

Figure 1 – ***Proportional distribution of study cases based on the age.***

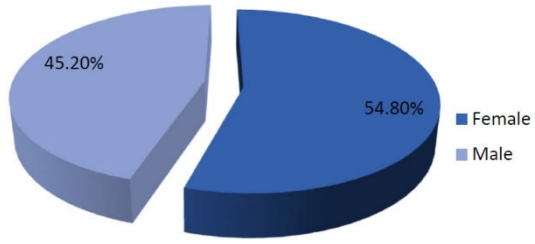

Figure 2 – ***Proportional distribution of study cases based on gender.***

Etiological spectrum includes (Figure 3):

- Mesenteric ischemia/infarction – 11 (35.48%) cases;
- Perforated peptic ulcer (gastric or duodenal) – five (16.13%) cases;
- Perforated or ruptured abdominal neoplasms – nine (29.03%) cases;
- Hepatic abscesses – two (6.45%) cases;
- Complicated hernias (strangulated) – two (6.45%) cases;
- Perforated appendicitis – one case (3.23%);
- Traumatic/posttraumatic causes (splenic rupture) – one case (3.23%).

Among the neoplastic etiologies, pancreatic head tumors (*n*=3), colonic tumors (*n*=2), and hepatocellular carcinoma (HCC) (*n*=1) predominated. These cases were characterized by direct tumor invasion of the duodenum or biliary tree, subsequent perforation, and generalized peritonitis. Autopsies revealed advanced local disease in 92% of these cases, with either vascular or peritoneal extension.

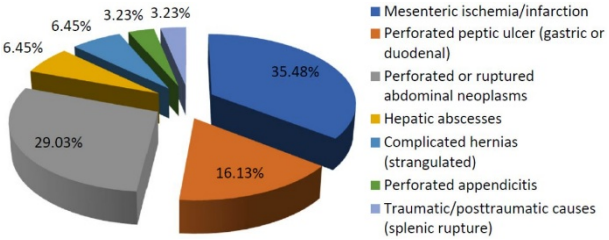

Figure 3 – ***Etiological distributions of fatal acute abdomen.***

COVID-19 period and thrombotic correlations

Between March 2020 and February 2022, corresponding to the COVID-19 pandemic, 15 fatal cases were recorded, of which nine were attributed to acute mesenteric infarction and six to other causes (perforated ulcers, hepatic and pancreatic tumors, or colonic perforations). In these COVID-19-related cases, thrombotic involvement of mesenteric vessels was a striking feature, with macroscopic evidence of superior mesenteric artery or vein thrombosis confirmed at autopsy in seven patients. Histologically, these showed widespread microthrombi and endothelial inflammation, consistent with SARS-CoV-2-associated coagulopathy (Figure 4).

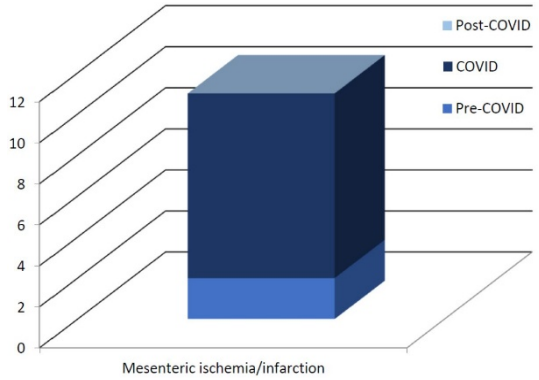

Figure 4 – ***Cases of mesenteric ischemia during, before and after coronavirus disease 2019 (COVID-19) pandemic.***

The mean D-dimer values in COVID-19-positive individuals were significantly higher (>8000 ng/mL, compared to a mean of 2400 ng/mL in non-COVID ischemic cases). CRP and ferritin levels were also markedly elevated, supporting the hyperinflammatory systemic state described in COVID-19 patients with severe coagulopathy [4, 5, 7].

Clinical and laboratory correlations

Septic presentation was observed in 26 of 31 (83.8%) cases, while shock (septic, hemorrhagic, or mixed) was documented in 14 (45.1%) cases. Of these, eight were classified as septic shock due to diffuse peritonitis, while four showed combined septic–hemorrhagic features (*e.g.*, tumor rupture with bacterial contamination). Hemorrhagic shock alone was noted in only two cases – both due to massive hemoperitoneum following hepatic or splenic rupture.

Leukocytosis (>20 000/μL) and markedly elevated CRP (>200 mg/L) were observed in 81% of patients presenting with peritonitis. Elevated lactate levels (>5 mmol/L) were found in 14 of the 16 cases where data were available, indicating severe tissue hypoxia and poor perfusion. Patients

with bowel infarction demonstrated the highest lactate concentrations (mean 8.6 mmol/L), which correlated strongly with necrotic findings at autopsy.

Postmortem findings and patterns

Autopsy examination confirmed generalized peritonitis in 21 (67.7%) cases, most commonly secondary to visceral perforation or transmural necrosis. In eight (25.8%) patients, diffuse peritonitis/hemoperitoneum exceeding 1000 mL was found, often due to rupture of vascularized tumors (especially HCC or pancreatic carcinoma). The remaining two cases exhibited localized abscess formation with secondary extension to adjacent structures such as the psoas muscle or retroperitoneum (Table 1).

Table 1 – ***Acute abdomen cases involved in the study***

Case No.	Age [years]	Sex	Residence	Etiology	Shock type/necropsy findings
1.	46	Male	Urban	Pancreatic head carcinoma invading the duodenum	Septic shock; perforation with secondary peritonitis
2.	73	Female	Urban	Pancreatic tail tumor with duodenal invasion	Septic shock; perforation and secondary peritonitis
3.	78	Female	Urban	Cholangiocarcinoma invading the duodenum	Septic shock; perforation and diffuse peritonitis
4.	82	Female	Urban	Splenic flexure colon tumor with perforation	Septic shock; generalized peritonitis
5.	69	Male	Urban	Hepatic tumor with vascular invasion and rupture	Hemorrhagic shock; aseptic hemoperitoneum
6.	74	Male	Rural	Strangulated inguinal hernia with obstruction	Septic shock; intestinal perforation and obstruction
7.	86	Female	Rural	Hepatic abscess extending to right psoas muscle	Septic shock; abscess rupture
8.	45	Female	Rural	Perforated gastric ulcer	Hemorrhagic and septic shock; secondary peritonitis
9.	79	Female	Rural	Perforated gastric ulcer	Septic shock; generalized peritonitis with hemoperitoneum
10.	60	Male	Urban	Mesenteric infarction	Septic shock; entero-mesenteric necrosis
11.	58	Male	Rural	Mesenteric infarction	Septic shock; endotoxemic necrosis of small bowel
12.	64	Female	Urban	Mesenteric infarction	Hemorrhagic (aseptic) shock; necrosis of intestinal wall
13.	91	Male	Urban	Mesenteric infarction	Septic shock; perforation and peritonitis
14.	69	Male	Urban	Mesenteric infarction	Septic shock; intestinal necrosis
15.	81	Male	Urban	Mesenteric infarction	Septic shock with; necrosis of bowel
16.	77	Male	Rural	Mesenteric infarction; septic shock	Septic shock; entero-mesenteric infarction with peritonitis
17.	69	Female	Urban	Perforated gastric ulcer; secondary peritonitis	Septic shock; perforated ulcer with diffuse peritonitis
18.	79	Female	Urban	Mesenteric infarction; permeation peritonitis	Septic shock; intestinal necrosis with peritonitis
19.	53	Male	Rural	Perforated and abscessed ascending colon tumor; fecaloid peritonitis	Septic shock; generalized fecaloid peritonitis
20.	88	Male	Rural	Perforated duodenal ulcer; secondary generalized peritonitis	Septic shock; ulcer perforation with diffuse peritonitis
21.	71	Female	Rural	Mesenteric infarction	Septic shock; entero-mesenteric necrosis
22.	73	Female	Rural	Strangulated femoral hernia; secondary intestinal obstruction	Septic shock; intestinal necrosis and obstruction
23.	59	Male	Urban	Mesenteric infarction	Septic shock; intestinal necrosis
24.	80	Male	Urban	Pancreatic head tumor invading and perforating the duodenum; secondary peritonitis	Septic shock; tumor perforation with peritonitis
25.	67	Female	Urban	Perforated gangrenous appendicitis; purulent peritonitis	Septic shock; purulent peritonitis
26.	90	Male	Urban	Liver abscess involving segments 4 and 5 with extension to the gallbladder (pyocholecyst)	Septic shock; hepatic abscess rupture
27.	69	Female	Urban	Mesenteric infarction	Septic shock; intestinal necrosis
28.	53	Male	Urban	Perforated and hemorrhagic gastric ulcer; hemoperitoneum	Hemorrhagic shock; aseptic hemoperitoneum
29.	57	Female	Rural	Post-traumatic splenic rupture grade IV; massive hemoperitoneum	Hemorrhagic shock; aseptic hemoperitoneum
30.	69	Male	Urban	Pancreatic tail tumor invading the duodenum with perforation; secondary peritonitis and cachexia	Septic shock; tumor perforation with peritonitis and cachexia
31.	68	Male	Urban	Perforated gastric tumor; generalized secondary peritonitis	Septic shock; perforated gastric carcinoma with peritonitis

Macroscopic examination revealed consistent patterns for each etiological category. Mesenteric infarction typically presented as extensive transmural intestinal necrosis, with dark red to black discoloration and hemorrhagic serosa (Figure 5). In most of these cases, the peritoneal cavity contained variable amounts of turbid or purulent fluid,

indicating either secondary peritonitis or peritonitis by permeation. Perforated gastric or duodenal ulcers produced generalized peritonitis, characterized by fibrinopurulent exudate coating the abdominal organs and large volumes of purulent or mixed purulent–hemorrhagic ascites. In contrast, perforated or ruptured neoplasms displayed irregular ulcerated masses with areas of necrosis and focal perforation, frequently accompanied by fecaloid contamination (Figure 6).

Figure 5 – ***Gross appearance of mesenteric infarction.***

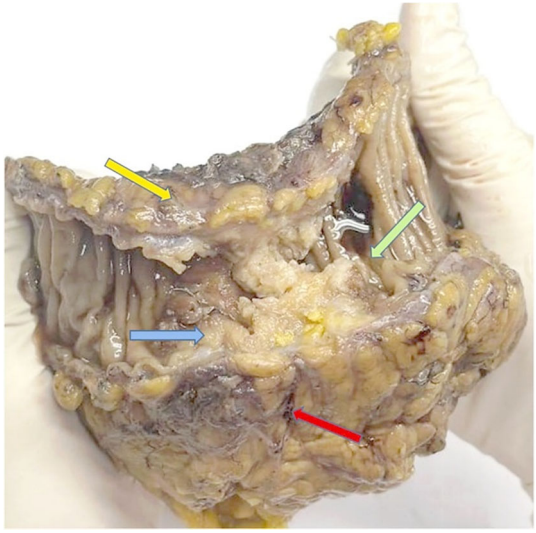

Figure 6 – ***Gross appearance of a colonic tumor – a large vegetating–ulcerated tumor causing marked wall thickening and luminal distortion: yellow arrow – exophytic, vegetating component of the tumor with irregular, friable surface, consistent with invasive colonic carcinoma; green arrow – ulcerated tumor area with loss of normal mucosal architecture and exposure of the tumor base; blue arrow – yellowish friable areas corresponding to tumor necrosis, suggestive of rapid tumor growth and insufficient vascular supply; red arrow – infiltrative tumor mass extending into the bowel wall, associated with transmural involvement and peri-colonic tissue reaction.***

Microscopically, mesenteric infarction cases demonstrated full-thickness ischemic necrosis with preserved crypt shadows, vascular thrombosis, bacterial translocation, and in septic patients, disseminated neutrophilic infiltration of the serosa. Perforated ulcers showed sharply demarcated loss of mucosa, necrosis extending into the muscularis propria or through the entire wall, fibrinopurulent serositis, and abundant bacterial colonies. Neoplastic perforations were associated with tumoral invasion of the bowel wall, extensive necrotic tumor areas, vascular erosion, and transmural inflammation (Figure 7). Hepatic abscesses showed central liquefactive necrosis surrounded by acute inflammatory infiltrate, whereas the traumatic splenic rupture presented with parenchymal lacerations, massive hemoperitoneum, and absence of septic features.

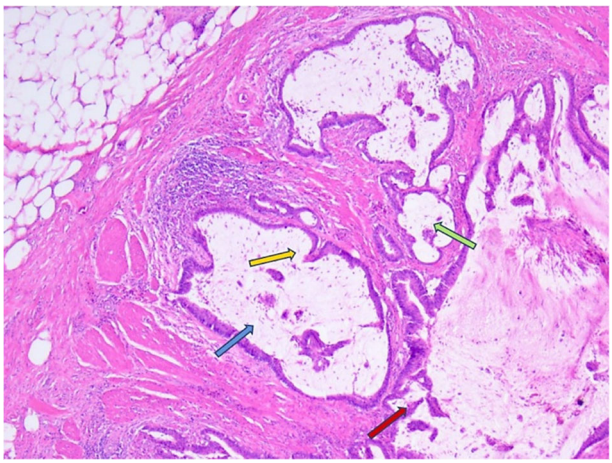

Figure 7 – ***Invasive mucinous adenocarcinoma: yellow arrow – irregular infiltrative mucin-filled neoplastic glands; green arrow – atypical columnar epithelium with nuclear pseudostratification; red arrow – marked desmoplastic stromal reaction indicating stromal invasion; blue arrow – extracellular mucin pools. Hematoxylin–Eosin (HE) staining, ×200.***

When stratifying the cases according to the COVID-19 pandemic interval, a distinct distribution was observed. Between March 2020 and February 2022, 15 fatal cases were recorded, of which nine (60%) resulted from mesenteric infarction and six (40%) from other causes (perforated ulcers, ruptured pancreatic or hepatic tumors, and colonic perforations). Outside the pandemic interval (pre- and post-COVID-19), the distribution was more heterogeneous, with a proportionally higher incidence of neoplastic perforations and septic complications unrelated to thrombotic mechanisms.

Histopathological analysis confirmed ischemic necrosis in 12 cases, and septic vasculitis in three cases. Microthrombi were noted in mesenteric and subserosal vessels in nine cases, seven of which corresponded to COVID-19-positive patients. These findings underline the thromboinflammatory interplay in abdominal catastrophes during the pandemic period.

Statistical associations

A positive correlation (r=0.73, p<0.01) was observed between serum lactate levels and the degree of bowel necrosis confirmed postmortem. Similarly, CRP and leukocytosis showed strong associations (r=0.64, p<0.05) with diffuse peritonitis severity. Age correlated moderately with both comorbidity burden and presence of malignancy (r=0.58).

When stratified by residence, urban patients presented significantly more neoplastic perforations, while rural patients showed more ulcerative or hernia-related complications, reflecting differences in healthcare accessibility and early intervention.

Mortality context

Compared to non-abdominal emergency deaths in the

same department, patients with acute abdomen exhibited faster progression to circulatory collapse, with an average time from presentation to death of 2.4±1.1 hours. This finding emphasizes the fulminant nature of these conditions and highlights the challenges of stabilization and transfer to surgical care.

Discussions

The morphopathological examination of fatal acute abdomen cases reveals a coherent progression from the initial organ insult to systemic collapse. Both macroscopic and microscopic findings illustrate how ischemic injury, perforation, or tumoral disruption rapidly overwhelm compensatory mechanisms, culminating in multiorgan failure. AMI and visceral perforation are consistently recognized as among the most lethal abdominal emergencies, with persistently high mortality despite modern diagnostic and surgical advances [1, 16].

Visceral perforation – whether peptic, neoplastic, or traumatic – results in abrupt contamination of the peritoneal cavity. Gross examination typically reveals diffuse fibrino-purulent or fecaloid peritonitis, while histological analysis shows intense neutrophilic infiltration, fibrin deposition, and necrosis at the perforation margins. The resulting polymicrobial peritonitis rapidly progresses to sepsis and multiorgan dysfunction if not promptly controlled. Perforations associated with gastrointestinal or pancreatic malignancies carry a particularly unfavorable prognosis. Tumoral infiltration disrupts normal tissue architecture, compromises vascular supply, and predisposes to ischemic necrosis and wall rupture. Macroscopically, perforation is often associated with hemorrhagic effusion and friable tumor tissue, while microscopically extensive tumoral necrosis, desmoplastic reaction, and vascular invasion are common findings. These pathological features explain the rapid progression to diffuse peritonitis and sepsis described in oncological emergency literature [17].

AMI remains one of the most challenging and lethal abdominal emergencies, characterized by rapid progression from ischemia to transmural intestinal necrosis if not promptly recognized and treated. Mortality rates continue to range from 40% to over 60%, particularly when diagnosis and intervention are delayed [1, 11, 12]. The emergence of the COVID-19 pandemic has further complicated this scenario, introducing a new thromboinflammatory dimension to mesenteric ischemia. SARS-CoV-2 infection is now known to trigger endothelial injury, complement activation, and widespread microvascular thrombosis, mechanisms that have been implicated in intestinal ischemic injury even in patients without traditional vascular occlusion (Figure 8) [2–5].

The pathophysiology of AMI in COVID-19 involves a combination of macrovascular occlusion, microthrombotic phenomena, and a hypercoagulable state driven by cytokine storm and endotheliitis. Histopathological evidence demonstrates fibrinous thrombi in mesenteric arterioles, mucosal necrosis, and endothelial swelling, consistent with the hypothesis of thrombotic vasculopathy [4, 5]. These findings highlight the multifactorial nature of ischemia in COVID-19, where systemic inflammation and vascular injury act synergistically to compromise mesenteric perfusion. Also, these alterations contributed to both occlusive and non-occlusive mesenteric ischemia, explaining the increased incidence and severity of intestinal infarction observed during the pandemic. Moreover, COVID-19-associated systemic hyperinflammation amplified the host response to abdominal emergencies, favoring rapid progression to septic shock in cases of ischemia or perforation [18].

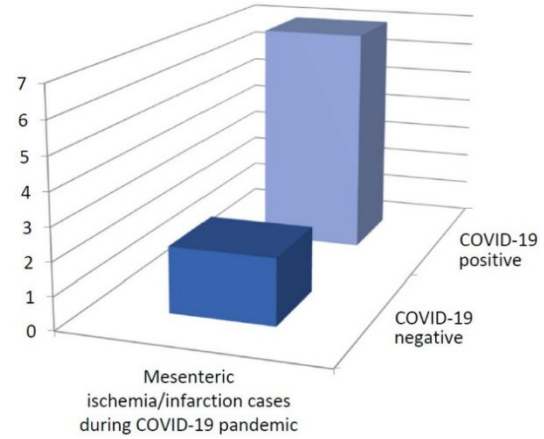

Figure 8 – ***Negative/positive COVID-19 testing of the cases with mesenteric ischemia during COVID-19 pandemic.***

Macroscopically, the affected bowel typically appears congested, violaceous, or frankly necrotic, while microscopically coagulative necrosis, mucosal denudation, hemorrhage, and thrombotic occlusion of mesenteric vessels are observed. Once transmural necrosis develops, loss of the intestinal barrier facilitates bacterial translocation and endotoxemia, rapidly triggering systemic inflammatory response and septic shock. Extensive intestinal infarction is associated with mortality rates exceeding 50%, mainly due to delayed diagnosis and limited therapeutic window [1, 16].

Clinically, early diagnosis remains the cornerstone of survival, but the nonspecific presentation of AMI often results in missed or delayed recognition. Abdominal pain "out of proportion" to physical findings is the classic hallmark, yet this feature is frequently subtle or absent in critically ill or sedated patients [6, 7]. The diagnostic "gold standard" is multidetector CTA, which provides near 90% sensitivity and allows visualization of both vascular and bowel wall changes [6]. During the pandemic, rapid CT assessment became indispensable for differentiating thrombotic *versus* inflammatory causes of acute abdomen in COVID-19-positive patients [8].

Therapeutic strategies depend on the underlying etiology and the degree of intestinal viability. Early revascularization – whether by open surgical embolectomy, endovascular thrombectomy, or hybrid techniques – remains the most effective intervention for arterial occlusive forms [9]. In contrast, nonocclusive or microthrombotic AMI, particularly in COVID-19 patients, often benefits from aggressive systemic anticoagulation, hemodynamic optimization, and correction of precipitating factors such as vasopressor-induced hypoperfusion [10, 11].

Postoperative outcomes remain dismal. Sepsis, multiple organ failure, and short bowel syndrome are the major causes of postoperative morbidity and mortality [13]. Studies have consistently identified the time to intervention, preoperative hemodynamic instability, and extent of intestinal

necrosis as independent predictors of mortality [12–14]. Importantly, COVID-19 appears to exacerbate these outcomes through persistent coagulopathy, cytokine-driven endothelial dysfunction, and impaired tissue oxygenation [14, 15].

Elderly patients and those with preexisting cardiovascular disease represent the most vulnerable group, not only due to compromised mesenteric perfusion but also because of reduced physiological reserve and the tendency toward delayed diagnosis [13]. In these populations, a high index of suspicion and a multidisciplinary response – combining surgical, radiological, and intensive care expertise – are critical for improving prognosis [8, 9].

Recent studies emphasize the importance of standardized management pathways for suspected AMI. Implementation of "mesenteric ischemia protocols" involving early CTA, prompt surgical evaluation, and liberal use of anticoagulation has demonstrated improved survival rates in tertiary centers [8, 10]. Nevertheless, outcomes remain highly dependent on early recognition, as every hour of delay in revascularization increases mortality by approximately 10–15% [1, 11].

In the COVID-19 era, AMI exemplifies the systemic consequences of viral-induced coagulopathy and endothelial injury. The interplay between infection, inflammation, and thrombosis underscores the necessity for early prophylactic anticoagulation and vigilant monitoring of gastrointestinal symptoms in infected patients [2–5, 14]. Multicenter collaboration and data sharing are essential to refine prognostic models and tailor management strategies in this complex and evolving clinical entity.

In summary, AMI continues to represent a devastating abdominal emergency with persistently high mortality, despite advances in imaging. COVID-19 has unveiled new pathogenic pathways, highlighting the need for heightened vigilance, multidisciplinary coordination, and early intervention. Timely diagnosis, aggressive revascularization when feasible, and comprehensive postoperative care remain the key determinants of survival in this challenging condition.

Nevertheless, morphopathological examination remains essential for reconstructing the sequence of events leading to death in fatal acute abdomen. It allows differentiation between acute and chronic ischemic processes, identification of perforation mechanisms, assessment of tumoral involvement, and recognition of COVID-19-related endothelial pathology. Integration of macroscopic and microscopic findings is particularly valuable when clinical data are incomplete or nonspecific. As emphasized in surgical and forensic pathology literature, systematic morphopathological analysis is indispensable for understanding lethal pathways and improving preventive and diagnostic strategies [19].

Conclusions

Acute abdominal emergencies, including mesenteric ischemia, perforated ulcers, and neoplastic peritonitis, carry high mortality, often before surgical intervention. The COVID-19 pandemic further emphasized the role of thrombotic and endothelial mechanisms, particularly in mesenteric ischemia. Clinicopathological correlation demonstrates that rapid progression from localized abdominal injury to sepsis or hemorrhagic shock underlies most fatal outcomes. Postmortem morphopathological analysis is essential for elucidating these mechanisms, refining diagnostic accuracy, and supporting the development of improved clinical pathways for early recognition and management. Early recognition, rapid triage, and multidisciplinary management remain essential to improve survival.

Conflict of interests

The authors declare no conflict of interests.

Institutional Review Board Statement

The study was conducted in accordance with the Declaration of Helsinki and approved by the Ethics Committee of the Emergency City Hospital, Timişoara, Romania (Approval No. E-4676/11.11.2025).

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