CASE REPORT

A fatal complex case of extensive digestive necrosis – was it trauma, intoxication or ischemia?

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Abstract

Introduction: Extensive necrosis of the upper digestive tract is an uncommon and life-threatening condition, usually associated with severe ischemia, toxic agents, or considerable abdominal trauma. Determining the primary cause might be challenging when other factors, such as systemic vascular disease or delayed hospital presentation, are involved. Instances of simultaneous necrosis in multiple organs are rarely recorded in the literature. We present a fatal case of widespread digestive necrosis in a patient with recent thoraco-abdominal trauma, history of homemade herbal products use, and extensive hyaline arteriolosclerosis.

Case Report: A 66-year-old woman with a history of type II diabetes mellitus and previous colectomy presented to the emergency room four days after a domestic fall. Clinical and imaging assessments revealed fluido-pneumothorax, marked stomach distension, hemoperitoneum, several mesenteric and splenic hematomas. An emergency exploratory laparotomy confirmed the intra-abdominal trauma, but also extensive necrosis in the esophagus, gastric mucosa and duodenum. A total gastrectomy, terminal abdominal esophagostomy, segmental small bowel resection, splenectomy, and jejunostomy were performed. Frozen section examination revealed transmural necrosis of the esophagus, stomach, and duodenum, severe ischemic enteritis, and considerable haemorrhagic extravasation. Despite the extensive postoperative care, the patient experienced significant hemodynamic instability, resulting in cardiac arrest and subsequent death. A forensic autopsy was required and performed at the Institute of Forensic Medicine - Târgu Mureș, due to trauma sustained prior to admission. A notable microscopic finding was generalized hyaline arteriolosclerosis involving small-calibre vessels in multiple organs, including the heart, kidneys, lungs, and gastrointestinal tract. Comprehensive toxicological testing of the blood and specimens yielded no evidence supporting a toxic etiology.

Conclusions: The present case illustrates how trauma, delayed hospital presentation, and significant microvascular disease may converge to produce extensive digestive necrosis. Recognition of underlying vascular pathology is crucial in the evaluation of complex abdominal trauma, given its potential impact on disease evolution and prognosis.

Keywords: Digestive necrosis; abdominal trauma; hyaline arteriolosclerosis; forensic autopsy.

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Introduction

Extensive necrosis of the upper digestive tract is a rare condition that may be associated with severe ischemia, trauma, or the accidental/intentional ingestion of toxic substances [1,2,3]. Blunt abdominal trauma involving the stomach is rare, with an incidence of 0.4%–1.7% [4]. Gastric stasis with a consequent increase intragastric pressure, along with the presence of other conditions such as generalized hyaline arteriolosclerosis, may more rapidly lead to ischemia and subsequently to gastric necrosis. According to data reported in the medical literature, gastric necrosis may occur 1–2 days after the onset of the triggering factor responsible for gastric dilation [5]. Hyaline arteriolosclerosis may occur in both normotensive individuals, but it is more common in those with arterial hypertension and diabetes mellitus; this pathology involves thickening of the arteriolar wall due to the deposition of hyaline material within the subendothelial space and the tunica media of small arteries and arterioles with progressive narrowing of the lumen and even complete obliteration of the vascular structures [6,7]. The kidney and spleen are the organs most frequently affected by hyaline arteriolosclerosis, but it may occur in all organs [6,7,8,9].

We present a fatal case of extensive esophago-gastro-duodenal necrosis in an individual who sustained a recent thoracoabdominal trauma following a fall from the same level, with a history of consuming homemade herbal drinks, in whom severe hyaline arteriolosclerosis involving multiple organs was identified.

Case Report

A 66-year-old female patient, with type II diabetes mellitus, history of colectomy and hysterectomy with adnexectomy, was brought by ambulance to the Emergency Department of the County Clinical Emergency Hospital in Târgu Mureș from another regional hospital in Mureș County. The patient complained of general malaise, pain

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in the right hemithorax and dyspnea; reportedly, four days earlier, she had sustained a falling trauma, without presenting to the hospital at that time. Following the medical history and objective clinical examination, blood tests and imaging investigations were performed, including a native thoracic CT scan and an abdominopelvic CT scan, which revealed bilateral hydropneumothorax, a collapsed right lung with minimal partial air bronchogram, pneumoperitoneum with multiple subcentimeter air bubbles in the perisigmoid and anteroinferior pelvic regions, pelvic fluid collection, the stomach distended with air and semisolid content, gastric stasis, and the urinary bladder displaced posterolateral because of the pelvic fluid collection. Admission to the Surgery Department was decided, and emergency surgical intervention was performed, consisting of bilateral thoracic drainage and exploratory laparotomy. Upon inspection, a moderate hemoperitoneum was identified, with 300 ml of blood evacuated, extensive hematomas in the peritoneal cavity, splenic hilum, gastrocolic ligament, and mesentery, a partial rupture of the mesentery, and a considerably distended stomach with violaceous, thinned, and atonic walls. Intraoperatively, the gastroenterology team was consulted to perform an intraoperative gastroscopy, which revealed necrosis of the gastric mucosa along the greater curvature. A partial small bowel resection and splenectomy were performed, and upon mobilization of the abdominal esophagus, increased wall friability was noted, raising suspicion of esophageal necrosis. Consequently, a total gastrectomy with terminal abdominal esophagostomy and feeding jejunostomy was performed, along with partial resection of a pelvic cyst. Fragments from the esophagus, stomach, duodenum, small intestine, spleen and omentum were collected for histopathological examination. After the surgical procedure, the patient was transferred to the ICU in critical condition, without reversal of general anesthesia, intubated and mechanically ventilated, with mydriatic pupils, hemodynamically unstable with vasoactive support, with a blood pressure of 48/34 mmHg and a heart rate of 76 bpm. Specific life-support treatment was settled, but the patient's condition deteriorated, and a few hours postoperatively, cardiorespiratory arrest occurred, with no response to resuscitation.

Given the recent history of traumatic event, a forensic autopsy was requested at the Institute of Forensic Medicine in Târgu Mureș, according to the Romanian legislation.

Autopsy Findings

During the autopsy, external examination revealed traumatic injuries, including an ecchymosis on the left frontal region, a hematoma in the left axillary region extending to the upper third of the left lateral hemithorax, and a hematoma in the sacrococcygeal region extending to the right gluteal area. The external examination revealed signs of medical treatment, including bilateral pleural drains, a 28 cm long post-surgical xipho-umbilico-pubic incision, abdominal drainage tubes, a jejunostomy and esophagostomy. Internal examination identified minimal bilateral hydrothorax, subpleural hemorrhagic petechiae, pulmonary congestion and acute edema, emphysema and areas of pulmonary fibrosis, minimal pericardial fluid, myocardial sclerosis, left ventricular hypertrophy, mild/moderate coronary atherosclerosis, as well as diaphragmatic and mesenteric blood infiltration, status post recent gastrectomy and splenectomy, duodenum ligated at its initial segment, end-to-end jejunal anastomosis following segmental small bowel resection, and an omega-shaped jejunal loop with a jejunostomy tube exteriorized through the left abdominal wall. At the esophageal level, ligation was observed in the lower third, with an end esophagostomy tube exteriorized through the left abdominal wall. Additionally, sternal and rib fractures caused by mechanical resuscitation were identified. During the medicolegal autopsy, tissue samples were collected from various organs, for histopathological examination, as detailed in Table I.

The investigative authorities provided the forensic toxicology laboratory three bottles containing liquids of different quantities and colors, which, according to the relatives, were frequently consumed by the deceased person. The results are presented in Table II.

Conclusions of the forensic autopsy report

Based on the autopsy findings and the correlation of all complementary medicolegal examination results, it was concluded that the death was violent, resulting from multiorgan failure due to extensive necrosis of the esophagus, stomach, duodenum, and connective-adipose tissue, in the context of a global hypoperfusion syndrome, secondary to a blunt thoraco-abdominal trauma to a person with generalized hyaline arteriolosclerosis, arterial hypertension, and diabetes mellitus.

The external traumatic injuries (left frontal contusion, left lateral thoracic hematoma, left sacrococcygeal hematoma) and internal injuries (left diaphragmatic blood infiltration; hemoperitoneum; extensive hematomas in the peritoneal cavity, splenic hilum, gastrocolic ligament, and mesentery; partial mesenteric rupture) could have resulted from impact against hard surfaces, possibly in the context of a fall. A direct conditional causal link was established between the traumatic injuries and death, with the conditional factor being the extensive and generalized hyaline arteriolosclerosis.

Discussions

1. Traumatic and Hemodynamic Mechanisms

Although this anatomical region has an efficient local blood supply and a rich collateral vascularisation, the occurrence of extensive necrosis at this level suggests that it is not merely a localized or isolated lesion, but rather the result of a complex, multisystem pathophysiological mechanism capable of producing such catastrophic consequences. Secondary hypovolemia due to post-traumatic hemo-

Table 1. Histopathological findings observed in the evaluated organ fragments.

No.	Organ	Histopathological changes in the organ fragments collected intraoperatively	Histopathological changes of the organ fragments collected during the medicolegal autopsy
1.	Heart	-	- Myocardial sclerosis; many myocardial fibers with acute ischemic lesions - Many small blood vessels with markedly thickened, hyalinized walls and narrowed lumen
2.	Lung	-	- Diffuse alveolar damage, exudative phase - Acute and chronic congestion - Many small blood vessel with markedly thickened and hyalinized walls
3.	Liver	-	- Many small blood vessels with markedly thickened and hyalinized walls
4.	Kidney	-	- Severe nephrosclerosis and chronic interstitial nephritis with parenchymal atrophy - Granular degeneration - Acute tubular necrosis -Small blood vessels with markedly thickened, hyalinized walls
5.	Pancreas	-	- Small blood vessels with thickened, hyalinized walls and narrowed lumen
6.	Diaphragm	-	- Numerous recent hemorrhagic infiltrates - Small blood vessels with thickened, hyalinized walls and narrowed lumen
7.	Esophagus	- Abundant hemorrhagic extravasation involving the submucosa and muscularis propria	- Recent hemorrhagic infiltrates involving the submucosa, muscularis propria, and adventitia - Small blood vessels with thickened, hyalinized walls and narrowed lumen
8.	Duodenum	- Duodenal resection margin with transmural hemorrhagic extravasation	- Recent hemorrhagic infiltrates in the submucosa and muscularis propria - Small blood vessels with thickened, hyalinized walls and narrowed lumen
9.	Omentum	-With abundant hemorrhagic extravasation	-
10.	Small intestine	- Congested blood vessels - Areas of focal necrosis	-
11.	Spleen	- Arterial vessels showing parietal hyalinosis with homogeneous thickening of the vascular wall	-
12.	Stomach	-Abundant transmural hemorrhagic extravasation and focal areas of transmural necrosis	-
13.	Right paracardial lymph node	- Extensive areas of necrosis with abundant hemorrhagic extravasation	-
14.	Cyst wall	- Zones of fibrosis and areas of necrosis	-

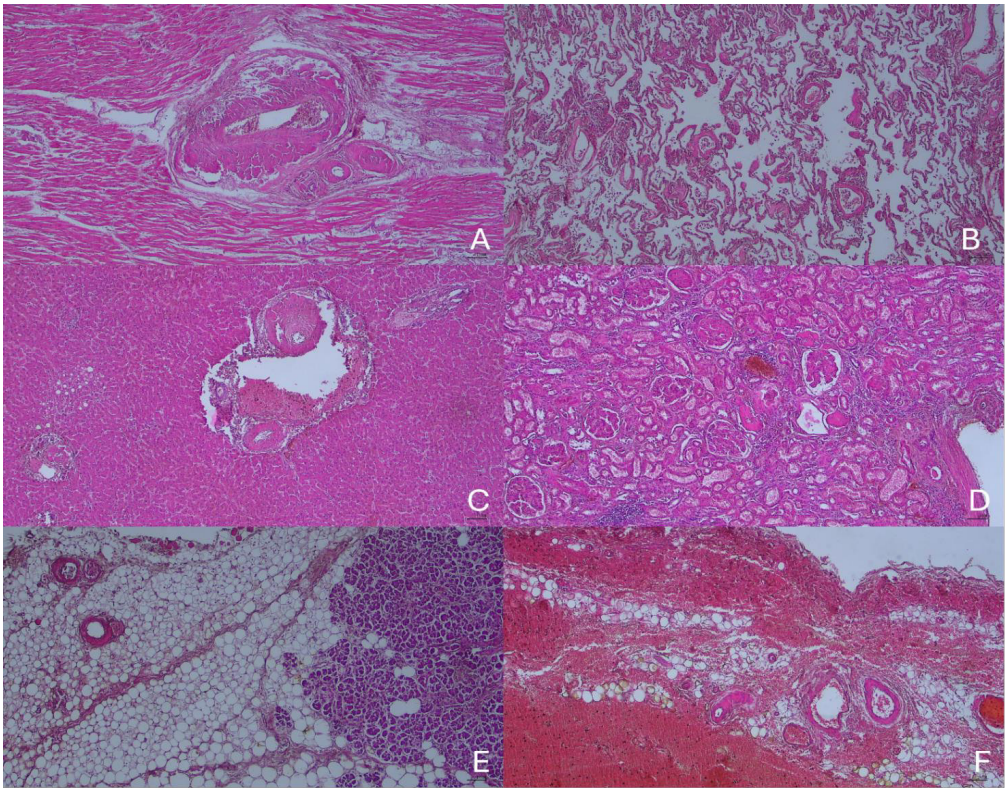


Fig. 1. Mediastinal goiter; 2. Trachea; 3. Esophagus

Table 2. Toxicological examination results.

	Sample analyzed	Method	Result
Biological samples	Blood (Blood alcohol level)	GC	Negative
	Blood (Toxicology)	GC-MS	Positive: Ketamine
Evidence specimens	Bottle 1 (330 mL) – 150 mL of contents	GC-MS	Positive: Stylopine (an alkaloid from Chelidonium majus); Asperuloside (a glycoside from Galium vernum); Menthol; Linalool; Chlorophyll.
	Bottle 2 (1000 mL) – 250 mL of contents	GC-MS	Positive: Ursolic acid (a triterpenoid from plants of the Lamiaceae family); Chlorophyll
	Bottle 3 (500 mL) – 350 mL of contents	Visual inspection	Rubbing alcohol

peritoneum, combined with generalized vasoconstriction, possible compressive effects from multiple intra-abdominal post-traumatic hematomas, as well as gastric stasis and distension (which, through increased intragastric pressure, could have further promoted and exacerbated ischemia), along with the delayed hospital presentation (four days after the traumatic event), most likely contributed to extensive ischemia at these sites [2,10]. Ischemic gastric necrosis most commonly occurs along the lesser curvature of the stomach, whereas necrosis of the greater curvature is characteristic in cases of gastric distension, suggesting two synergistic mechanisms in the progression of gastric ischemia; the location of gastric mucosal necrosis along the greater curvature, identified during intraoperative endoscopy, corresponds to the location described in the case reported by Ibrahim Aydin et al. in the 2013 article titled "Gastric Necrosis due to Acute Massive Gastric Dilatation."

2. Ischemic Susceptibility and Vascular Factors.

Generalized hyaline arteriolosclerosis, confirmed histopathologically, impaired microvascular autoregulation and reduced the capacity of arterioles to maintain tissue perfusion under stress. The post-traumatic hypovolemic state and generalized vasoconstriction likely produced systemic hypoperfusion, which, in the setting of rigid, narrowed arterioles, could not be compensated. This led to severe hypoperfusion of the esophago-gastro-duodenal mucosa and, together with local compressive effects and gastric distension, contributed to extensive ischemic necrosis. Arteriosclerosis and hypoperfusion are among the most important factors involved in gastrointestinal ischemia, as also observed by Flavio Paterno et al. in the study "The Etiology and Pathogenesis of Vascular Disorders of the Intestine", and diagnosis and treatment remain a multidisciplinary challenge [11]. In this context, the arteriolar wall thickening and luminal obliteration observed histologically would have significantly limited the ability of the microcirculation to respond to hemodynamic challenges. The combination of systemic factors (hypovolemia, vasoconstriction, delayed resuscitation) and local mechanical factors (compressive hematomas, gastric distension, increased intragastric pressure) created a synergistic effect that overwhelmed the already impaired microvascular compensatory capacity.

3. Possible Toxicological Contribution.

The homemade beverages consumed contained various plant species, which were identified by GC-MS analysis, including stylopine (an alkaloid from Chelidonium majus), asperuloside (a glycoside from Galium vernum), and ursolic acid (a triterpenoid from plants of the Lamiaceae family). These compounds can have various systemic effects, including potential impacts on the gastric mucosa. In particular, in addition to the anti-inflammatory, antitumor, and antimicrobial effects of Chelidonium majus, it also exhibits antispasmodic activity, as described by Zielińska S. et al. in the study "The Activity of Isoquinoline Alkaloids and Extracts from Chelidonium majus against Pathogenic Bacteria and Candida sp." [12]. The potential antispasmodic effect at the gastric level could have influenced gastric motility, subsequently resulting in gastric stasis and distension, representing an additional contributing factor alongside the abdominal trauma and generalized hyaline arteriolosclerosis. However, toxicological analysis did not detect these substances in the blood, likely due to the time interval between ingestion, hospital admission, and death. Furthermore, the timing and quantity of ingestion remain unknown, limiting the ability to establish a direct causal relationship.

Conclusion

The particularity of this case lies in the combination of thoraco-abdominal trauma, extensive necrosis of the esophagus, stomach, and duodenum, and extensive generalized hyaline arteriolosclerosis—a synergistic triad that responded poorly to medical and surgical treatment, rapidly leading to the patient's death. The hypothesis of intoxication was also investigated; however, the negative toxicological results could not objectively support it. The forensic autopsy and post-mortem multiorgan histopathological examination contributed decisively to the identification of generalized hyaline arteriolosclerosis as a significant pathological substrate, providing an explanation for the unfavourable clinical outcome despite a complex multidisciplinary approach.

Authors' contributions

AH – Writing – Original draft, Data curation, Investigation.
ȘAL – Conceptualization, Investigation, Data curation.
LC – Writing – review & editing, Resources, Visualization.
BAS – Methodology, Investigation, Visualization.
TH – Methodology, Investigation, Software.
CCR – Writing – review & editing, Supervision, Visualization.
CC – Conceptualization, Writing – review & editing, Validation.

Conflict of interest

None to declare.

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