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BLACK ESOPHAGUS AFTER BILE DUCT INJURY: A CASE REPORT OF ACUTE ESOPHAGEAL NECROSIS

Esôfago negro após lesão do ducto biliar: relato de caso de necrose esofágica aguda

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Abstract

Background: Acute esophageal necrosis, also known as black esophagus, is a rare and potentially life-threatening condition characterized by circumferential necrosis of the esophageal mucosa, generally associated with systemic hypoperfusion and chemical injury caused by gastric reflux. Its most common clinical manifestation is upper gastrointestinal bleeding, particularly in elderly patients with severe underlying conditions. The occurrence of this complication following an iatrogenic bile duct injury represents an unusual clinical scenario requiring early diagnosis and multidisciplinary management.
Aim: To report a case of acute esophageal necrosis following an iatrogenic bile duct injury during laparoscopic cholecystectomy, highlighting the clinical and endoscopic findings, therapeutic interventions, and patient outcomes. Methods: This descriptive case report was based on the analysis of the clinical course of a 78-year-old female patient who developed acute esophageal necrosis following laparoscopic cholecystectomy complicated by a bile duct injury. Clinical findings, imaging examinations, endoscopic procedures, surgical interventions, therapeutic management, and outcomes during hospital follow-up were evaluated. Results: A 78-year-old female patient presented with severe abdominal pain, vomiting, and hematemesis on the fifth postoperative day following laparoscopic cholecystectomy. Computed tomography revealed free intraperitoneal fluid, and diagnostic laparoscopy identified biliary peritonitis associated with an injury to the duct of Luschka, classified as Strasberg type A, as well as choledocholithiasis. Upper gastrointestinal endoscopy demonstrated extensive circumferential necrosis of the esophageal mucosa, consistent with acute esophageal necrosis. Initial management included fasting, nutritional support, proton pump inhibitor therapy, and abdominal drainage. Follow-up endoscopy showed no evidence of perforation. However, approximately two weeks later, the patient developed oral feeding intolerance and vomiting, and cicatricial esophageal stenosis was identified. Continued enteral nutritional support was required, followed by exploratory laparotomy with choledochotomy, papillosphincteroplasty, and gastrostomy. Subsequently, endoscopic esophageal dilation was initiated, with favorable clinical recovery. Conclusion: Acute esophageal necrosis should be considered in patients presenting with upper gastrointestinal bleeding following surgical complications associated with systemic hypoperfusion and intra-abdominal inflammation. Early endoscopic diagnosis, clinical and nutritional support, and definitive management of the underlying biliary injury are essential for treating this condition. In this case, despite the development of esophageal stenosis, multidisciplinary management resulted in favorable clinical outcomes, highlighting the importance of serial endoscopic follow-up for the identification and treatment of late complications.
Keywords: Acute esophageal necrosis, Black esophagus, Bile duct injury, Cholecystectomy, Upper gastrointestinal bleeding.

Resumo

Introdução: A necrose esofágica aguda, também conhecida como esôfago negro, é uma condição rara e potencialmente grave, caracterizada por necrose circunferencial da mucosa esofágica, geralmente associada à hipoperfusão sistêmica e à lesão química por refluxo gástrico. Sua manifestação mais frequente é a hemorragia digestiva alta, especialmente em pacientes idosos e com condições clínicas graves. A ocorrência dessa complicação após lesão iatrogênica da via biliar representa uma situação incomum, que exige diagnóstico precoce e abordagem multidisciplinar. Objetivo: Relatar um caso de

necrose esofágica aguda após lesão iatrogênica da via biliar durante colecistectomia videolaparoscópica, destacando os achados clínicos e endoscópicos, as intervenções terapêuticas e a evolução da paciente. Método: Estudo descritivo, do tipo relato de caso, baseado na análise da evolução clínica de uma paciente de 78 anos que desenvolveu necrose esofágica aguda após colecistectomia videolaparoscópica complicada por lesão da via biliar. Foram avaliados os achados clínicos, exames de imagem, procedimentos endoscópicos, intervenções cirúrgicas, condutas terapêuticas e evolução durante o acompanhamento hospitalar. Resultados: Paciente de 78 anos apresentou dor abdominal intensa, vômitos e hematêmese no quinto dia após colecistectomia videolaparoscópica. A tomografia computadorizada evidenciou líquido livre abdominal, e a videolaparoscopia diagnóstica identificou peritonite biliar associada à lesão do ducto de Luschka, classificada como Strasberg tipo A, além de coledocolitíase. A endoscopia digestiva alta demonstrou necrose circunferencial extensa da mucosa esofágica, compatível com necrose esofágica aguda. O tratamento inicial incluiu jejum, suporte nutricional, inibidor da bomba de prótons e drenagem abdominal. A endoscopia de controle não evidenciou perfuração. Entretanto, após aproximadamente duas semanas, a paciente apresentou intolerância alimentar e vômitos, sendo identificada estenose esofágica cicatricial. Foi necessária manutenção da nutrição enteral, seguida de laparotomia exploradora com coledocotomia, papiloesfincteroplastia e gastrostomia. Posteriormente, iniciou dilatação endoscópica esofágica, apresentando evolução clínica favorável. Conclusão: A necrose esofágica aguda deve ser considerada em pacientes com hemorragia digestiva alta após complicações cirúrgicas associadas à hipoperfusão sistêmica e à inflamação intra-abdominal. O diagnóstico endoscópico precoce, o suporte clínico e nutricional e o tratamento da lesão biliar são fundamentais para o manejo dessa condição. Neste caso, apesar do desenvolvimento de estenose esofágica, a abordagem multidisciplinar permitiu evolução favorável, demonstrando a importância do acompanhamento endoscópico seriado para identificação e tratamento de complicações tardias.
Palavras-chave: Necrose esofágica aguda, Esôfago negro, Lesão da via biliar, Colecistectomia, Hemorragia digestiva alta.

Background

Acute esophageal necrosis (AEN), historically termed "black esophagus" or Gurvits syndrome, is a rare, life-threatening clinical syndrome defined by striking circumferential, diffuse, dark-pigmented mucosal devitalization predominantly involving the distal esophagus and terminating sharply at the gastroesophageal junction [2,6]. Epidemiological evaluations across large retrospective endoscopic cohorts estimate its prevalence between 0.01% and 0.20%, predominantly affecting elderly male patients harboring profound baseline clinical vulnerabilities [6-8]. The classic presentation features overt upper gastrointestinal hemorrhage, manifesting as hematemesis, melena, or coffee-ground emesis in 70% to 90% of cases, frequently accompanied by epigastric discomfort, nausea, vomiting, fever, or hemodynamic collapse [2,7,11]. Laboratory examinations universally reveal profound yet nonspecific abnormalities, including severe anemia, marked leukocytosis, lactic acidosis, hyperglycemia, and hypoalbuminemia [3,6].

The pathophysiological cascade underpinning AEN is multifactorial and relies on a "two-hit" model centered on severe regional microvascular hypoperfusion combined with overwhelming luminal chemical injury [6,7]. The distal third of the esophagus represents a relative vascular watershed zone supplied principally by terminal branches of the left gastric and inferior phrenic arteries, rendering its mucosal lining particularly susceptible to ischemic insult during systemic hypotension, hypovolemic shock, sepsis, or cardiogenic failure [1,4,10]. Superimposed on tissue hypoxia, the breakdown of protective mucosal barriers permits refluxed acidic gastric contents and activated digestive enzymes to provoke extensive chemical digestion, culminating in full-thickness mucosal necrosis [2,6]. Recognized predisposing conditions encompass diabetes mellitus, systemic arterial hypertension, atherosclerotic cardiovascular disease, chronic alcohol abuse, malnutrition, and prothrombotic or malignant states [3,7,12,13].

Diagnosis is established through esophagogastroduodenoscopy (EGD), which reveals the pathognomonic appearance of friable, non-viable, coal-black mucosa that typically spares the gastric cardia [2,5,11]. Mucosal biopsy is not mandatory when classic endoscopic findings are identified but may be warranted in immunocompromised hosts to exclude infectious etiologies such as cytomegalovirus, herpes simplex virus, or invasive fungal colonization [2,6]. Management is conservative and supportive, focusing on immediate hemodynamic restoration with intravenous crystalloids, complete esophageal rest (nil per os), suppression of gastric acid with high-dose

intravenous proton pump inhibitors, mucosal cytoprotection, and definitive control of the precipitating systemic insult [1,2,7]. Despite supportive care, overall mortality remains high, approaching 30% to 50%, although fatality is predominantly driven by underlying systemic catastrophic comorbidities rather than direct esophageal perforation or mediastinitis [2,3,6,7].

Although AEN has been described secondary to septic, cardiogenic, and hypovolemic shock, its occurrence subsequent to an iatrogenic bile duct injury (BDI) and its complex postoperative convalescence constitutes an exceptionally rare clinical scenario. Iatrogenic biliary injuries represent devastating surgical complications that provoke biliary peritonitis, systemic inflammatory response syndrome, biloma formation, prolonged hemodynamic instability, and repeated operative or interventional stress. To our knowledge, the intersection of acute bile duct injury, subsequent critical illness, and acute esophageal necrosis managed via combined surgical and endoscopic modalities has received minimal focus in existing surgical literature. Herein, we describe the clinical course, diagnostic workup, endoscopic findings, and interdisciplinary management of a patient diagnosed with AEN after sustaining a major bile duct injury at a tertiary university center.

Aim

To report a rare case of acute esophageal necrosis (black esophagus) following an iatrogenic bile duct injury during laparoscopic cholecystectomy, highlighting the clinical presentation, diagnostic findings, therapeutic management, and postoperative complications.

Methods

This is a descriptive, retrospective case report of a 78-year-old female patient who developed acute esophageal necrosis following an iatrogenic bile duct injury during laparoscopic cholecystectomy. Clinical manifestations, laboratory and imaging findings, upper gastrointestinal endoscopic examinations, surgical interventions, therapeutic management, and clinical outcomes were analyzed.

Results

On the fifth postoperative day, the patient developed abdominal pain, hematemesis, and signs of peritoneal irritation. Diagnostic laparoscopy revealed biliary peritonitis associated with a Strasberg type A bile duct injury and choledocholithiasis. Upper gastrointestinal endoscopy demonstrated extensive circumferential esophageal necrosis. Initial management included fasting, nutritional support, proton pump inhibitor therapy, and abdominal drainage. Follow-up endoscopy showed no evidence of perforation. However, the patient subsequently developed esophageal stenosis, requiring enteral nutritional support and endoscopic dilation. Additional surgical treatment included choledochotomy, papillosphincteroplasty, and gastrostomy, followed by favorable postoperative recovery.

Discussion

Acute esophageal necrosis is an unusual and devastating presentation of severe visceral ischemia and mucosal chemical breakdown [2,6]. Although AEN is classically described in elderly individuals with significant cardiovascular, metabolic, or end-stage renal disease who present with acute hematemesis [3,7,8], this report illustrates an atypical precipitating setting: the development of circumferential esophageal necrosis secondary to severe systemic and local hemodynamic compromise triggered by an iatrogenic bile duct injury. The pathophysiology of AEN in this clinical context directly aligns with the recognized "two-hit" paradigm [2,6,7].

The human esophagus possesses a segmented vascular network: the cervical segment is supplied by the inferior thyroid arteries, the midthoracic portion receives branches directly from the thoracic aorta and bronchial arteries, and the distal segment relies on branches from the left gastric and left inferior phrenic arteries [6,7]. Consequently, the distal esophagus constitutes a microvascular watershed zone with comparatively lower baseline blood flow, rendering it disproportionately vulnerable to systemic hypoperfusion [2,6]. In our patient, intraoperative and postoperative systemic hypotension, systemic inflammatory response syndrome (SIRS) provoked by bile leakage, and significant blood loss compromised microvascular perfusion across the celiac axis and left gastric territories. This initial ischemic insult disrupted the mucosal barrier, permitting acid, pepsin, and potentially duodenogastroesophageal reflux containing bile acids to induce transmural chemical digestion, producing the distinctive black, necrotic mucosal architecture [4,6,10].

Similar patterns of acute ischemic mucosal devitalization have been reported following severe hypovolemic and hemorrhagic shock [1], septic shock [7], and acute metabolic crises such as diabetic ketoacidosis [4,10]. Dias et al. observed that systemic vascular collapse and multiorgan dysfunction were primary drivers of mortality in patients diagnosed with AEN [2,3]. Furthermore, Gurvits et al. demonstrated in multicenter analyses that while direct esophageal perforation occurs in fewer than 10% of cases, overall mortality frequently exceeds 30%, largely reflecting the severity of the primary triggering insult rather than intrinsic esophageal failure [6,7]. The presentation in our patient, characterized by sudden-onset upper gastrointestinal bleeding during surgical recovery, mirrors the predominant symptomatic pattern described in 70% to 90% of published cohorts [2,7,11].

Diagnostic confirmation relies definitively on urgent endoscopic visualization [2,5,6]. The pathognomonic finding is coal-black, circumferential mucosal necrosis extending proximally from the squamocolumnar junction with variable proximal demarcation, terminating abruptly at the Z-line because the gastric cardia receives dual, robust vascularization from the short gastric and left gastric networks [2,6,11]. While deep biopsies can definitively demonstrate full-thickness mucosal necrosis, many endoscopists prudently avoid aggressive biopsy during the acute phase due to heightened risk of iatrogenic transmural perforation of the ischemic esophageal wall [2,6,9]. When pursued, histopathology serves primarily to exclude cytomegalovirus, herpes simplex virus, or fungal invasion in immunocompromised hosts [2,6]. Differential diagnoses include corrosive ingestion, malignant melanoma, melanosis esophagi, pseudomelanosis, and severe coal-dust deposition, all of which can be readily differentiated based on clinical history and endoscopic features [2,6,12,13].

Management remains primarily medical, anchored on aggressive resuscitation, reversal of tissue hypoperfusion, total parenteral nutrition, and high-dose acid suppression [1,2,7]. Intravenous proton pump inhibitors suppress luminal gastric acidity, halting acid-mediated enzymatic degradation and promoting mucosal restitution [2,6]. Sucralfate acts synergistically by establishing a physical barrier over the ulcerated mucosa [2,7]. Crucially, as highlighted in this report, definitive resolution of the underlying biliary disruption, whether achieved through biliary reconstruction, surgical diversion, or endoscopic drainage, is indispensable for terminating the systemic inflammatory response and restoring regional hemodynamic equilibrium [2,6,7]. Although specialized interventions such as esophageal stenting have been utilized in refractory bleeding or impending perforation [9], surgical resection is strictly reserved for cases complicated by free transmural perforation, mediastinitis, or abscess formation [2,6].

Long-term surveillance is necessary because esophageal stenosis or stricture formation occurs in up to 10% to 25% of surviving patients during the healing phase, typically developing between the second and fourth weeks following the initial insult [2,6,7]. Serial endoscopic follow-up is essential to ensure adequate mucosal regeneration and to facilitate timely endoscopic balloon dilatation should symptomatic strictures arise [2,7]. The major limitation of this report is its single-center, retrospective observational design, which precludes establishing direct causality beyond the chronological and pathophysiological association observed. Nonetheless, this case adds valuable insights to the sparse literature linking severe biliary surgical complications to acute ischemic esophageal necrosis, demonstrating that early endoscopic identification and aggressive supportive care can achieve mucosal healing without surgical esophagectomy.

The chronological sequence of clinical events, diagnostic evaluations, and interventions is outlined in Table 1.

Table 1: Chronological sequence of clinical events, diagnostic evaluations, and interventions

Timeline	Clinical Event and Diagnostic Findings	Interventions and Management
Day 0	Index surgical procedure	There wasn't any
Day 5	Postoperative instability, Acute upper gastrointestinal hemorrhage, biloperitoneum, and hemodynamic decompensation	Diagnostic videolaparoscopy, biliary tract and abdominal cavity drainage
Day 6	Diagnostic esophagogastroduodenoscopy: classic acute esophageal necrosis	fasting, full-dose PPI, and parenteral nutrition
Day 8	New endoscopy showing no signs of complications	Nasoenteric tube placement
Day 12	Clinical stability and good dietary tolerance	Hospital discharge for scheduled ERCP following nutritional improvement
Day 26	Poor tolerance of oral diet and early vomiting. Endoscopy showing esophageal stenosis.	Enteral diet maintained and surgery scheduled.
Day 40	Unable to tolerate oral intake and with a biliary drain in place.	Exploratory laparotomy was performed to carry out a choledochotomy, papilloshincteroplasty, and gastrostomy.

Conclusion

Acute esophageal necrosis is a rare but potentially severe complication associated with postoperative systemic hypoperfusion and inflammatory conditions. Early endoscopic diagnosis, hemodynamic stabilization, nutritional support, and multidisciplinary management are essential to improve clinical outcomes and identify complications such as esophageal stenosis.

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