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ACUTE ESOPHAGEAL NECROSIS: A RARE BLACK ESOPHAGUS SYNDROME

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ABSTRACT

Background: Acute esophageal necrosis is a rare condition characterized by diffuse black discoloration of the distal esophageal mucosa with abrupt demarcation at the gastroesophageal junction. It develops due to the combined effects of ischemia, impaired mucosal defense and chemical injury from refluxed gastric contents. The condition is typically associated with upper gastrointestinal bleeding and severe systemic comorbidities.

Aim: The aim of this review is to summarize current knowledge on acute esophageal necrosis and to highlight clinically relevant aspects of diagnosis, risk assessment and management that may support clinical decision making.

Material and Methods: A narrative literature review was performed using PubMed, Scopus, Web of Science and Google Scholar. Peer reviewed full text publications in English from 1990 to 2025 were included. Search terms comprised acute esophageal necrosis, black esophagus, ischemic esophagitis, gastrointestinal hemorrhage, endoscopic findings, management and outcomes.

Results: Acute esophageal necrosis is a rare but serious cause of upper gastrointestinal bleeding with an incidence between 0.01 percent and 0.28 percent. It predominantly affects elderly or critically ill patients with multiple comorbidities. Available data indicate a multifactorial pathogenesis involving ischemic injury, loss of mucosal integrity and chemical damage from reflux. Mortality of approximately 32 percent is mainly related to underlying systemic disease rather than the esophageal necrosis itself.

Conclusions: Acute esophageal necrosis carries a poor prognosis despite its low incidence. Early recognition, urgent endoscopic evaluation and timely supportive therapy are essential for improving outcomes. Current evidence is based primarily on case reports and small retrospective series. Larger studies are required to refine diagnostic criteria, risk stratification and management strategies.

Keywords: acute esophageal necrosis, black esophagus, ischemia, endoscopy, esophageal diseases, gastrointestinal hemorrhage, esophagitis

INTRODUCTION

Acute Esophageal Necrosis (AEN), also known as Black Esophagus, Acute Necrotizing Esophagitis or Gurvits Syndrome is a rare but dangerous disease of the esophagus that has diffuse and circumferential black discoloration of the mucosa that usually ends abruptly at the junction. This disease was first described in 1990 as a most severe form of esophageal injury due to ischemic insult combined with impaired mucosal defenses and chemical injury from gastric reflux. Although less common, with incidence rates ranging from 0.01% to 0.28%, it significantly differs by a mortality risk due to big underlying comorbidities. According to most cases, the symptomatology involves the upper symptoms of gastrointestinal bleeding, including hematemesis, melena or coffee-ground emesis [1]. These diseases usually affect old men with many chronic illnesses like diabetes mellitus, cardiovascular disease, renal insufficiency and malnutrition-all of which conditions predispose to vascular compromise and mucosal vulnerability. The blackened mucosa primarily involves the distal third of the esophagus endoscopically, reflecting the region's relatively poor blood supply [2]. AEN is mainly supportive in treatment, focusing on hemodynamic stabilization, proton pump inhibitor therapy, mucosal protection, and treatment of underlying systemic illness. Surgical intervention is reserved for those with complications by perforation or mediastinitis. Diagnosis is usually delayed because the symptoms that arise are vague and nonspecific. Many diseases have similar symptoms, making differential diagnosis very important. Despite advances in recognition and care, overall mortality is high, emphasizing the importance of early diagnosis and comprehensive management [3, 4]. The condition is very rare, and its subtle signs become reasons for careful observation. The rationale behind this literature review will be to update the epidemiology, pathophysiology, clinical presentation, and diagnostic and management strategies that control the course of acute esophageal necrosis in order to better inform clinical practice and improve patient outcomes.

RELEVANCE

Acute esophageal necrosis remains a clinically significant condition due to its high mortality, frequent association with severe comorbidities and the risk of delayed diagnosis caused by nonspecific symptoms. Although the syndrome is rare, it is regularly encountered in patients with shock, diabetic ketoacidosis, malnutrition and multiorgan disease, which makes timely recognition essential. The overlap of symptoms with other causes of upper gastrointestinal bleeding underscores the need for clear diagnostic guidance and structured clinical decision making.

NOVELTY

The novelty of this review lies in its focus on clinically relevant aspects of acute esophageal necrosis, including early diagnostic criteria, practical risk stratification, differential diagnostic markers, structured initial management and indicators for follow up endoscopy. By summarizing these elements within a single clinical framework, the review highlights features that are directly applicable to practice and supports informed decision making in patients with suspected or confirmed acute esophageal necrosis.

This review aims to summarize current knowledge on acute esophageal necrosis and to identify clinically relevant aspects of diagnosis, risk assessment and patient management that can support decision making and improve outcomes.

RESEARCH TASKS:

1. To identify the key clinical signs that require urgent endoscopic evaluation in suspected AEN.
2. To classify major risk groups and determine simple criteria for risk stratification at hospital admission.
3. To analyze the differential diagnostic options and outline practical criteria that distinguish AEN from similar conditions.
4. To summarize evidence based principles of initial management and to formulate a stepwise clinical approach for early stabilization and prevention of complications.
5. To define indications for conservative and surgical treatment strategies in patients with AEN.
6. To clarify the role and timing of follow up endoscopy and its relevance for detecting complications.
7. To characterize typical clinical scenarios in which AEN should be considered early in the diagnostic process.
8. To outline recommendations for post episode monitoring aimed at reducing the risk of stricture formation.

METHODOLOGY

This narrative review was conducted to synthesize current knowledge on the epidemiology, pathogenesis, clinical presentation, diagnosis, management and outcomes of acute esophageal necrosis. A comprehensive literature search was performed in PubMed, Scopus, Web of Science and Google Scholar. The search covered publications from 1990 to

2025. Earlier sources were included selectively for historical context. Search terms included acute esophageal necrosis, black esophagus, ischemic esophagitis, pathogenesis of AEN, clinical presentation, gastrointestinal hemorrhage, endoscopic findings, management and outcomes. Titles and abstracts were screened for relevance.

A total of 112 publications were identified. After applying predefined inclusion and exclusion criteria, 54 full text articles were retained.

Inclusion criteria comprised peer reviewed full text publications in English, including original studies, clinical case reports, systematic or narrative reviews and case series that addressed etiology, pathophysiology, diagnosis, treatment, prognosis or complications of acute esophageal necrosis. Exclusion criteria comprised conference abstracts, editorials, animal only studies and papers without specific focus on acute esophageal necrosis.

References of the selected articles were hand searched to identify additional relevant sources. Extracted data were organized into thematic domains that included epidemiology and risk factors, pathophysiology, clinical features and diagnostic approach, management principles and outcomes. The findings were summarized descriptively with attention to recurring clinical patterns, discrepancies between reports and remaining gaps in knowledge.

FINDINGS

DEFINITION AND EPIDEMIOLOGY

AEN is a rare but serious condition characterised by sudden, dark necrosis of the esophageal mucosa. It classically affects the distal third of the oesophagus and ends abruptly at the gastro-oesophageal junction. The incidence of AEN is 0.01–0.28% of patients undergoing esophagogastroduodenoscopy (EGD). The term Black Esophagus emphasises the striking endoscopic appearance of extensive mucosal necrosis, seen in a setting of critical illness [1, 2, 3]. The disease affects men more often with male predominance up to 4:1. The average age of diagnosis is in the elderly category (usually 60–70 years), most often in people with comorbidities and general debilitation [4]. While the exact incidence is difficult to estimate, AEN appears to be a disease whose prevalence may be higher than generally believed. Diagnosis is being made with increasing endoscopy and awareness of the syndrome. Studies suggest that it may occur transiently in patients with upper gastrointestinal bleeding [5, 6]. It is also underestimated due to the frequent subclinical form of the disease and early healing of the esophageal mucosa. Furthermore, the correct diagnosis is often not made due to comorbidities, especially in patients with reflux esophagitis [1]. AEN usually occurs in the setting of marked physiologic stress (e.g., shock states, multiorgan failure), some authors suggest the true prevalence may be higher than reported, with cases missed or attributed to other causes of upper gastrointestinal bleeding [6, 7].

PATHOPHYSIOLOGY

The pathogenesis of Black Esophagus is considered to be a compilation of multiple factors, and several interacting mechanisms have been proposed to explain the characteristic necrosis of the distal esophagus. One of these factors has been shown to be the "two-hit" theory; that is, initial hypoperfusion/ischemia of the poorly vascularized distal esophagus with subsequent superimposed damage to the overlying mucosa due to either regurgitation of gastric juice or to luminal injury [1, 8]. Firstly, there is the aspect of vascular compromise. The distal part of the esophagus receives rather poor blood supply (via the left gastric and inferior phrenic arteries) compared to other segments. As such, it becomes susceptible to ischemic damage particularly in conditions of low flow and hypotension. The process of cellular damage begins with mucosal and submucosal hypoxia due to ATP depletion, dysfunction of the sodium-potassium pump, damage to tight junctions, and activation of the process of coagulative necrosis. In patients with multiple conditions that make them susceptible to ischemic disorders, hypotension becomes potentially damaging to the esophagus due to hypoperfusion and ischemic necrosis [8, 9, 10].

In the second place, there is luminal injury and intraluminal insult. The distal esophagus is potentially susceptible to corrosive damage due to the regurgitation of highly acidic gastric juices, duodenogastric reflux, and possibly obstructive gastric emptying. In the context of diminished blood flow, the chemical damage can be disproportionately severe and potentially beyond the capacity of the esophageal epithelium to withstand. Furthermore, dysfunction in esophageal peristalsis and lower esophageal sphincter function due to hiatal hernias or obstruction generally leads to AEN [1, 11].

Thirdly, impairment of mucosal defence and repair mechanisms is critical. Many patients have comorbidities and general debilitation including malnutrition, diabetes mellitus, alcohol abuse, congestive heart failure, chronic pulmonary disease, cancer, immunosuppression or systemic inflammatory states, each of which compromises mucosal integrity, diminishes bicarbonate or mucus protective secretion, and delays epithelial regeneration. These factors render the esophagus less resilient to ischemic or chemical assaults [1, 4, 10]. Furthermore, after the inception of necrosis, there might also be the presence of hemorrhage due to the fragile mucosa potentially giving way to infections translocating due to the breach in barriers. The histopathology findings include evidence of necrosis in the squamous epithelium and submucosa/muscularis propria layers with the absence of viable squamous

epithelium with hemorrhage [11, 12]. Though the core pathogenic triad of AEN includes ischemia, massive acid refluxes, and damage to the barrier function/mucosal barrier function, other contributory factors in vulnerable patients include severe hypothermia, vasoconstrictors, thrombotic occlusion of the microvascular beds in the esophagus, alcohol-related lactic acidosis, and micro-infections (such as Candida and HSV infections) [13].

In conclusion, therefore, the mechanism underlying AEN can be succinctly defined as the 'cumulative damage to the distal esophagus in the context of systemic predisposition with ischemia as the final common pathway. Early unmasking and reversal of underlying triggering factors (including hemodynamic disturbance and uncontrolled refluxes), therefore, becomes essential to avert further progression from superficial injury to full-thickness infarction [1, 4, 10, 11, 13]. Notably, AEN may also occur in the absence of shock or hypotension, indicating that although hemodynamic instability increases risk, it is not a necessary condition for disease development. Across reported cases, however, at least two of the following have been consistently present: acute ischemia, pre-existing vascular disease, or substantial local injury [10, 14, 15]. Table 1 outlines key mechanisms of acute esophageal necrosis with their examples, functional consequences and supporting references.

Table 1. Mechanisms dominating in the pathogenesis of Acute Esophageal Necrosis.

Mechanism	Examples	Functional consequence	Reference number
Ischemia	Shock, hypotension, vasculopathy, post-arrest hypoperfusion	Hypoxia-driven barrier failure and onset of coagulative necrosis	[1, 8, 13]
Luminal corrosive injury	Acid / duodenogastric reflux, outlet obstruction, vomiting	Superimposed chemical burn on hypoxic mucosa → accelerated necrosis	[3, 4, 6, 9, 11]
Weak mucosal defence	Diabetes/DKA, malnutrition, alcohol, immunosuppression	Lower resilience and delayed repair → lower injury threshold	[7, 8, 28]
Additional mechanisms	Hypothermia, vasoconstrictors, microthrombosis, infection major surgery/trauma, →Hemorrhage, barrier loss, translocation	Converts subclinical injury into full-blown necrosis →Deep extension (submucosa/muscle), infection, perforation risk	[10, 13, 14, 15]

DKA-diabetic ketoacidosis;

RISK FACTORS AND PREDISPOSING FACTORS

The development of Acute Esophageal Necrosis is typically associated with multiple converging risk factors. These factors can be grouped into hemodynamic/ischemic insults, luminal injury and reflux and impaired mucosal defence and repair. A "red-flag" mindset is required-patients presenting with upper gastrointestinal bleeding in the setting of these risk factors should raise suspicion for AEN. It is a rare disease and recognition of red-flag combinations is critical [4]. Hemodynamic compromise and ischemia play a central role in the pathogenesis of acute esophageal necrosis. The condition is frequently reported in patients who present with significant hemodynamic instability, often necessitating admission to the intensive care unit. Low-flow states such as shock, hypotension, systemic vasculopathy, and circulatory failure represent some of the most significant predisposing factors for AEN. In patients with underlying vascular disease, hypotension can lead to inadequate tissue perfusion and ultimately result in ischemic necrosis of the esophageal mucosa. Several retrospective studies have identified compromised hemodynamic states-such as septic or hypovolemic shock as the most common precipitating events leading to AEN [1, 16, 17]. It also has been frequently reported in association with septic shock, where generalized vasodilation mediated by inflammatory cytokines causes profound hemodynamic compromise and reduced perfusion pressure. Reported primary sources of infection include skin and soft tissue infections, urinary tract infections, and necrotizing fasciitis. Cardiogenic shock represents another major cause of AEN, typically occurring secondary to acute myocardial infarction, stress-induced cardiomyopathy, or cardiac arrest [16, 17, 18].

In these conditions, the inability of the heart to maintain effective cardiac output leads to systemic hypoperfusion and ischemia of the gastrointestinal tract, including the esophagus [18, 19, 20, 21]. Hemorrhagic shock has also been

implicated in the development of AEN due to acute blood loss caused by conditions such as bleeding varices, rupture of a thoracic or abdominal aortic aneurysm, traumatic vascular injury, or postoperative bleeding (following liver transplantation or cholecystectomy) [22, 23, 24]. Other less common causes associated with AEN are hemodynamic compromise due to excessive use of antihypertensive medication-especially combinations of different drug classes-and hypovolemia due to gastrointestinal losses in the form of vomiting or diarrhea [25, 26].

Other important predisposing factors in the development of black esophagus include gastric outlet obstruction, reflux, and corrosive luminal injury. Reflux of acidic gastric and duodenogastric contents into a vulnerable esophagus, especially in conditions of delayed gastric emptying or mechanical obstruction at the gastric outlet, may cause severe damage and necrosis of the mucosa. Conditions such as gastric outlet obstruction, gastric dilatation, gastric volvulus, and episodes of massive or recurrent vomiting have been shown to promote backflow of acidic and bile-rich contents, thereby aggravating ischemic injury to the esophageal mucosa [3, 4, 9, 11]. In postoperative observations, further factors that may contribute to black esophagus include duodenal or gastric ulcer disease and reflux of gastric or biliary secretions. These conditions raise intragastric pressure and promote the retrograde movement of corrosive contents into the esophagus, compounding preexisting mucosal vulnerability caused by systemic hypoperfusion or ischemia [6]. The important predisponing factors in the development of AEN are impaired mucosal defense and systemic vulnerability. Indeed, many affected patients usually have multiple comorbidities that impair mucosal integrity and systemic resilience, such as advanced age, male sex predominance, diabetes mellitus, hypertension, chronic kidney disease, alcohol abuse, malignancy, and malnutrition [3, 7, 16]. Case series demonstrated that AEN predominantly affects patients with significant comorbidities such as hypertension, diabetes mellitus, dyslipidemia, chronic kidney disease, coronary artery disease, and malignancy. Among these, diabetes particularly when complicated by diabetic ketoacidosis (DKA) has been repeatedly implicated as an important predisposing factor. Several reviews have emphasized DKA, alcohol intoxication, and multiorgan failure as common antecedent states leading to AEN [3, 16, 17, 27]. Furthermore, chronic alcohol abuse and malnutrition, both of which impair mucosal defense and compromise tissue repair mechanisms, have been identified as additional risk factors. Studies highlighted the association between AEN and these conditions, suggesting that systemic nutritional and metabolic deficiencies play a crucial role in weakening the esophageal mucosa and increasing susceptibility to necrotic injury [7, 8, 28]. A variety of additional triggering factors have been implicated, broadening the spectrum of predisposing conditions. Case reports and series have identified surgery, particularly major operations and transplants trauma, vasoconstrictive drug use, local infections, and gastric ulceration as important contributors [1, 4, 6]. Inflammatory and infectious diseases such as acute pancreatitis, cholecystitis, pneumonia, ischemic colitis, and peritonitis may trigger AEN through inflammatory vasodilatation, fluid shifts, and reduced perfusion pressure. Disseminated infections such as bacteremia caused by Escherichia coli, Klebsiella oxytoca, Staphylococcus aureus, and fungemia caused by Candida glabrata can further increase ischemia. Local infections of the esophagus due to Herpes simplex virus, Cytomegalovirus, Candida, and other opportunistic pathogens can immediately injure the esophageal mucosa. These are supported by the role of biopsy to establish microbiologically directed therapy [1, 4, 6, 29, 30]. Malnutrition and neoplasm are other significant risk factors causing underlying susceptibility and repairing resistance to the esophageal mucosal barrier. Cachectic patients with underlying conditions such as malignancies, cirrhosis, renal failure, and heart failure are particularly susceptible to AIN [8, 29]. Approximately 10% of AEN cases are evident in cancer patients with malignancies such as those affecting the esophagus, colon, pancreas, and lungs; these are postulated to be fragile due to malignancy and stress induced by chemotherapeutic regimens [1]. These observations highlight that AEN is due to the resultant interplay between underlying susceptibility factors and hemodynamically induced tension in conjunction with direct injury to the esophageal barrier [3, 4, 8]. These risk and predisposing factors can be identified to attempt to prevent and manage patients in its initial stages, as shown in Table 2.

Table 2. Classification of risk and predisponing factors.

Category	Included factors	Reference number
Major predisponing factor	systemic hypoperfusion, shock, vascular disease or end-organ compromise, diabetes, DKA, alcohol abuse, malnutrition	[18, 19, 20, 21]
Local injury factors	gastric or duodenogastric reflux, gastric outlet obstruction, massive vomiting, postoperative reflux	[3, 4, 6]
Situational triggers	major surgery or trauma, vasoconstrictive drugs; cardiac arrest, local infections (Candida/HSV), severe ulcer disease	[1, 4, 6, 29, 30]

Clinical red flags	hemodynamic instability and UGI bleeding in a high-risk host (older male, DM, CKD, vascular disease)	[22, 23, 24, 25, 26]

DKA-diabetic ketoacidosis; HSV-herpes simplex virus; DM-diabetes mellitus; CKD-chronic kidney disease; UGI bleeding-upper gastrointestinal bleeding;

CLINICAL PRESENTATION AND DIAGNOSIS

Acute Esophageal Necrosis is an unusual but potentially fatal condition that manifests with subtle to extensive black pigmentation involving the entire circumference mainly within the distal one-third with clear demarcation present at the esophagogastric junction. The usual presentation includes acute upper gastrointestinal bleed with hematemesis caused mainly due to acute onset and more subtle presentation with melena [6, 28, 29]. Symptoms include abdominal pain and epigastric pain, vomiting, abdominal distention with fever and syncope. Symptoms that manifest prominently include those due to hemodynamic instability such as tachycardia and hypotension suggestive of blood loss and shock respectively [4, 6]. Laboratory parameters include anemia due to acute blood loss with accompanying elevated WBC due to possible infectious/inflammatory causes [4]. The most striking observation has been that most patients with Acute Esophageal Necrosis possess numerous concomitant diseases such as diabetes mellitus, cardiovascular diseases, sepsis, and immunosuppression [1, 3, 4].

The confirmation of AEN diagnosis is best made with an Esophagogastroduodenoscopy (EGD). The key endoscopic finding is the appearance of the circumferential black necrotic mucosa that most affects the distal esophagus with clear-cut delineation between the esophagus and the gastroesophageal junction. The extension to the esophagus may be patchy to diffusely involve the esophagus. Other endoscopic findings include active hemorrhage, edema, ulcers within the duodenum, gastritis, and evidence suggesting gastric outlet obstruction [8, 14, 20, 31, 32]. Biopsy and histopathology are not essential diagnostics; however, these may aid in other differential diagnoses that include malignant melanoma, pseudomelanosis, and melanosis in patients with AEN with black esophagus. Characteristic findings include complete loss of viable epithelium, extensive mucosal and submucosal necrosis, vascular thrombosis, and intense inflammatory infiltration. In some cases, necrosis extends into the muscularis propria. Biopsy samples should be cultured for bacterial, fungal, and viral pathogens to identify potential infectious contributors [8, 33, 34].

The differential diagnosis includes conditions that cause black discoloration of the esophagus. The most frequently mentioned in differential diagnosis that can present with similar endoscopic findings are Malignant melanoma, Acanthosis nigricans, Coal dust deposition, Pseudomelanosis, Melanosis of the Esophagus, Black dye ingestion, Direct caustic injury [4, 11, 13, 35, 36, 37]. Additionally, corrosive esophagitis due to ingestion of caustic substances must be excluded. Endoscopic appearance supported by biopsy or cytology along with clinical history assists in distinguishing AEN from these entities [14, 33, 38]. What is common in differentiating these diseases from AEN is a much longer, slow-wave course, which does not produce such sudden symptoms as in the case of AEN and is detected during scheduled gastroscopies. In these entities, the stage of necrosis has rarely onset, as is the sudden case with acute esophageal necrosis. The characteristic distal esophageal involvement with abrupt GE junction transition may also be a valuable clue [3, 13, 39]. Accurate differentiation from these conditions is essential for appropriate management. Table 3 summarizes the main clinical features, diagnostic criteria, differential diagnoses and associated findings of acute esophageal necrosis with supporting references.

Table 3. Summary of key points for Acute Esophageal Necrosis.

Aspect	Details	Reference number
Clinical Presentation	Acute upper GI bleeding (hematemesis, melena), abdominal pain, nausea, syncope, hemodynamic instability	[4, 6, 28, 29]
Diagnostics	Endoscopy showing circumferential black mucosa of distal esophagus; biopsy supportive but not essential	[8, 14, 20, 31, 32]
Differential diagnosis	Malignant melanoma, acanthosis nigricans, pseudomelanosis, caustic esophagitis	[4, 11, 13, 35, 36, 37]
Associated Findings	Duodenal ulcers, gastritis, gastric outlet obstruction, anemia, leukocytosis	[8, 14, 20, 31, 32]

Acute upper GI bleeding - acute upper gastrointestinal bleeding;

MANAGEMENT AND TREATMENT

The management process does not include any form of definitive management. It is mainly supportive and aims to normalize hemodynamics, prevent damage to the esophageal membrane lining, and manage risk factors. In most cases that lead to AEN arising from critical conditions such as the Black Esophagus Syndrome, the early detection and management are critical to making significant improvements to morbidity and mortality rates. There is special emphasis on the causes that lead to AEN, besides intravenous hydration that aims to shield the esophagus from further potential damage [1, 11, 40, 41]. Large fluid challenges with crystalloids and blood transfusion with packed red blood cells are needed to increase blood volume and alleviate anemia caused by acute gastrointestinal hemorrhage. The patient must be made nil per os (NPO), and nasogastric intubation should be avoided due to the possible risk of perforation. Intravenous proton pump inhibitors (PPIs) are the mainstay of therapy, reducing gastric acid secretion and protecting the compromised esophageal mucosa, additional sucralfate [6, 17, 19, 42]. In selected cases, prophylactic antibiotics may be administered to high-risk patients, routine antibiotic use in uncomplicated AEN is not recommended [1]. Patients should receive total parenteral nutrition (TPN) during the acute phase to facilitate mucosal healing while minimizing mechanical irritation. Enteral nutrition and enteral tube placement must be postponed until there is evidence of mucosal healing. Once the patient's condition has stabilized, oral medication can be gradually resumed under endoscopic guidance [6, 8]. Follow-up endoscopy is advised to assess healing and exclude complications such as stricture development; this can be done after 4 to 6 weeks post-episode. Endoscopic hemostatic interventions, including adrenaline injection or placement of covered self-expanding metallic stents, may be employed for active bleeding [6, 43]. Balloon tamponade should be avoided due to perforation risk. Late complications such as esophageal strictures develop in up to 10% of patients and may be treated effectively with endoscopic balloon dilation. Refractory strictures may need repeated dilation procedures or stent placement [44, 45, 46]. Surgery is only required in those patients who develop potentially life-threatening complications such as esophageal perforations, mediastinitis, and abscess development [8]. In these instances, subtotal to complete esophagectomies with diversion may be required. Following stabilization of these patients, reconstruction procedures are carried out. The requirement for surgical management has been seen to occur in 4-7% of instances. These instances are also associated with significant morbidity and mortality rates [9]. Other minimally invasive approaches such as video-thoracoscopic drainage and stent placements have been shown to be successful in these instances [47, 48, 49, 50].

PROGNOSIS AND OUTCOMES

This disease carries a significant risk of morbidity and mortality, with prognosis linked closely to the extent of esophageal damage, underlying patient comorbidities, and complications such as infection or perforation. The prognosis is generally poor [3, 4]. The direct mortality due to esophageal necrosis was about 6%. The mortality rate was roughly 30-32%, mainly due to other critical conditions superimposed rather than AEN itself. The survival rates increase markedly if diagnosed and managed appropriately with supportive measures with more than 60% obtaining beneficial results [6, 11, 17]. The outcome becomes worse if AEN is further complicated with sepsis, esophageal perforation, and multi-organ failure. The most dreadful complications are those due to esophageal perforation with 7% incidence and more than 40% mortality rates [51, 52]. Stricture and stenosis occur in the recovery stage and might result in morbidity requiring repeated endoscopic dilations and even surgical esophagectomy in resistant instances [3, 10]. The recurrence has been found to be due to unremitted duodenal diseases with gastric outlet obstruction [53]. Long-term sequelae of AEN are poorly documented due to limited follow-up data. Persistent strictures and esophageal dysfunction are the principal chronic complications [4, 17, 54].

RESULTS

The analysis of the selected 54 publications allowed identification of several clinically relevant aspects of acute esophageal necrosis that can be directly derived from current evidence.

Clinical indicators for immediate endoscopic evaluation

Across reviewed sources, hematemesis, melena, syncope, hemodynamic instability, tachycardia and acute anemia consistently appeared as indications for urgent esophagogastroduodenoscopy. These manifestations occurred in the majority of documented cases and represented the earliest opportunity for diagnosis.

Initial clinical management sequence

The reviewed material demonstrated a reproducible pattern of initial stabilization. Key elements included hemodynamic support with crystalloids and blood transfusion, nil per os status, avoidance of nasogastric intubation due to perforation risk, and early initiation of intravenous proton pump inhibitor therapy. Sucralfate and parenteral nutrition were used to support mucosal healing. This sequence was consistently reflected in published case series and reviews.

Risk stratification at admission

The literature indicated that advanced age, male sex, diabetes mellitus especially in the setting of ketoacidosis, chronic kidney disease, cardiovascular disease, sepsis, alcohol abuse and malnutrition represented dominant predictors of severe course. The combination of upper gastrointestinal bleeding with hemodynamic instability in a patient with these comorbidities repeatedly preceded the diagnosis. These factors allow formation of a simple admission risk profile.

Differential diagnostic criteria

Comparison of endoscopic and clinical features across studies enabled distinction between AEN and conditions producing similar black discoloration such as malignant melanoma, acanthosis nigricans, pseudomelanosis, melanosis and caustic injury. The abrupt distal involvement with clear demarcation at the gastroesophageal junction and acute clinical presentation were consistently specific for AEN, whereas alternative entities presented with gradual course and incidental endoscopic discovery.

Criteria for conservative versus surgical management

Published evidence showed uniform criteria for escalation. Conservative therapy was appropriate in cases limited to mucosal and submucosal necrosis without perforation or mediastinal complications. Surgical intervention was consistently indicated in perforation, mediastinitis or abscess formation. These thresholds were repeatedly confirmed across case series.

Role and timing of follow up endoscopy

Multiple publications supported a control endoscopy at four to six weeks to verify mucosal healing, detect strictures and guide reintroduction of oral intake. Earlier endoscopy was performed only in cases of deterioration or suspected complications.

Typical clinical scenarios requiring early suspicion of AEN

The synthesis of clinical data identified recurrent patterns. These included gastrointestinal bleeding during shock states, diabetic ketoacidosis, severe vomiting, postoperative reflux, gastric outlet obstruction and multiorgan failure. In these settings AEN appeared repeatedly and should be considered early.

Post episode monitoring to prevent stricture formation

The included literature indicated that up to ten percent of patients develop esophageal strictures. Early identification by follow up endoscopy and subsequent endoscopic dilation were effective strategies. Patients with persistent reflux, outlet obstruction or nutritional deficiencies required closer monitoring.

DISCUSSION

The synthesis of the reviewed literature allows a more clinically oriented interpretation of acute esophageal necrosis, emphasizing the practical implications identified in the Results section. The condition remains rare, but the collected data consistently underline that its clinical presentation is dominated by upper gastrointestinal bleeding, hemodynamic compromise and acute anemia, which together form the most reliable triggers for urgent endoscopic evaluation. These signs appear early and repeatedly across published cases, making them central to timely diagnosis [4, 6, 8, 17, 28, 29].

The structured analysis of management principles demonstrates that initial stabilization follows a reproducible sequence. Hemodynamic support with fluids and blood products, strict avoidance of gastric intubation because of perforation risk, early initiation of intravenous proton pump inhibition and mucosal protection reflect the core of immediate care. This unified pattern across heterogeneous case reports confirms that treatment relies on supportive measures aimed at restoring perfusion and minimizing ongoing mucosal damage [6, 17, 19, 42].

Risk stratification also emerges as clinically relevant. The literature shows a persistent association between severe outcomes and the presence of diabetes mellitus, ketoacidosis, chronic kidney disease, cardiovascular disease, malnutrition, alcohol misuse and sepsis [1, 4, 7, 8, 10]. These comorbidities, when combined with gastrointestinal bleeding, allow early identification of high risk patients. This observation provides a simple framework for estimating severity at admission, even in the absence of formal scoring systems [16, 28].

Differential diagnosis continues to be essential because multiple conditions can mimic the black appearance of the esophageal mucosa. The abrupt demarcation at the gastroesophageal junction, the acute clinical onset and the typical distal distribution distinguish AEN from pseudomelanosis, acanthosis nigricans, malignant melanoma and caustic injury. These distinctions are supported across multiple reports, allowing clinicians to narrow the diagnostic field when

facing black esophageal mucosa [11, 13, 35, 36, 37].

The decision between conservative and surgical treatment is confirmed to follow a consistent line. Conservative management remains appropriate when necrosis is confined to the mucosal layers without signs of perforation. Surgical intervention is uniformly indicated when perforation, mediastinitis or abscess formation is present. This threshold appears repeatedly across the reviewed studies and represents the clearest practical recommendation available [3, 4, 9].

The role of follow up endoscopy is another point of agreement. A control procedure at approximately four to six weeks allows clinicians to confirm mucosal healing and identify strictures early. This timing correlates with the natural evolution of mucosal recovery and the onset of narrowing. In cases of clinical deterioration, earlier evaluation is justified, although this scenario is less common [8, 14, 20, 31, 32].

Typical clinical settings in which AEN should be suspected include gastrointestinal bleeding during shock, ketoacidosis, prolonged vomiting, postoperative reflux and multiorgan dysfunction. These recurring clinical contexts underline the importance of maintaining a high level of suspicion in patients with systemic instability and abrupt gastrointestinal symptoms.

Finally, the occurrence of post necrotic strictures in a subset of patients indicates the need for structured follow up. Early detection through surveillance endoscopy and subsequent endoscopic dilation, when required, represent the main tools for preventing long term morbidity [1, 2, 4, 11, 40, 41].

Taken together, these findings demonstrate that although the literature on acute esophageal necrosis remains largely descriptive and based on small heterogeneous cohorts, it provides a coherent set of clinically applicable principles. The integration of these elements addresses key diagnostic and therapeutic uncertainties and aligns the review with practical clinical needs. Table 3 summarizes the core clinical features, diagnostic findings, differential diagnoses and associated conditions in acute esophageal necrosis.

Table 3. Summary of key points.

Aspect	Details
Clinical Presentation	Acute upper GI bleeding (hematemesis, melena), abdominal pain, nausea, syncope, hemodynamic instability
Diagnostics	Endoscopy showing circumferential black mucosa of distal esophagus; biopsy supportive but not essential
Differential diagnosis	Malignant melanoma, acanthosis nigricans, pseudomelanosis, caustic esophagitis
Associated Findings	Duodenal ulcers, gastritis, gastric outlet obstruction, anemia, leukocytosis

Legend: Acute upper GI bleeding - acute upper gastrointestinal bleeding

This table is a summary of key points relevant to the topic. It gives an overview of the principal clinical, diagnostic and associated features of AEN. Clinical presentation usually consists of acute upper gastrointestinal bleeding presented as hematemesis or melena, often accompanied by abdominal pain, nausea, syncope, and signs of hemodynamic instability. Diagnosis is made by endoscopy, which shows circumferential black discoloration of the distal esophageal mucosa with an abrupt transition at the gastroesophageal junction. Biopsy is supportive but not obligatory. The main differential diagnoses are other causes of esophageal pigmentation or necrosis, including malignant melanoma, acanthosis nigricans, pseudomelanosis, and caustic esophagitis. Associated findings may include duodenal ulcers and/or gastritis, gastric outlet obstruction, anemia from blood loss, and leukocytosis reflecting inflammation or infection.

CONCLUSIONS

Acute esophageal necrosis represents a rare clinical condition associated with upper gastrointestinal bleeding and severe systemic illness. Available evidence indicates that the disorder arises from the combined effects of ischemia, impaired mucosal defense and chemical injury from refluxed gastric contents. Although the incidence is low, the condition carries a high mortality that is determined mainly by underlying comorbidities rather than by esophageal necrosis itself. Early recognition and urgent endoscopy are essential for diagnosis, since most patients present with bleeding and hemodynamic instability.

Management relies on supportive therapy that includes hemodynamic stabilization, intravenous acid suppression, mucosal protection and treatment of the precipitating systemic disorder. Surgical intervention is reserved for cases complicated by perforation or mediastinitis. Follow up endoscopy is important for confirming mucosal recovery and identifying strictures that may require dilation.

Current knowledge is based largely on case reports and small retrospective series, which limits interpretation and prevents firm conclusions regarding risk prediction and optimal management strategies. Larger and well designed studies are needed to clarify epidemiology, refine risk stratification and better define clinical pathways. Greater clinical awareness of characteristic presentations and differential diagnostic features may contribute to earlier detection and improved outcomes.

DISCLOSURE

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The authors declare no conflict of interest.

USE OF AI

The authors used artificial intelligence tools to assist with language editing and structural refinement. All AI-assisted content was reviewed and revised by the authors to ensure that its use did not influence the scientific integrity or substantive content of the work. Artificial intelligence was not used to extract data from primary studies, all information was checked manually.

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