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A Case of Esophageal Infectious Shock Precipitated by Klebsiella pneumoniae Acute Esophagitis: Presenting with Refractory Epigastric Pain

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Abstract

Background: Acute epigastric pain is a common emergency presentation, but infectious esophagitis as its etiology is rare and easily overlooked [1]. We report a rare case of *Klebsiella pneumoniae* infectious esophagitis presenting as refractory epigastric pain and rapidly progressing to septic shock.

Case Presentation: A 52-year-old man with uncontrolled diabetes presented with severe epigastric pain unresponsive to multiple analgesics. Computed tomography (CT) revealed diffuse esophageal wall thickening, and he rapidly deteriorated into septic shock, rendering endoscopy contraindicated. Blood next-generation sequencing (NGS) identified *Klebsiella pneumoniae* (resistant to third-generation cephalosporins), leading to targeted therapy with meropenem and continuous renal replacement therapy (CRRT). Subsequent endoscopy confirmed inflammatory esophageal swelling.

Conclusion: Refractory epigastric pain unresponsive to conventional analgesics should alert clinicians to severe atypical infections such as *K. pneumoniae* esophagitis, especially in immunocompromised patients. Early CT evaluation and NGS are crucial for rapid pathogen identification and targeted therapy to prevent fatal septic shock.

Keywords

Acute Epigastric Pain, Esophageal Inflammation, Septic Shock, Next-Generation Sequencing (NGS)

Introduction

Acute epigastric pain is one of the most common chief complaints in the emergency department (ED), with a complex etiology involving multiple systems, such as the gastrointestinal and cardiovascular systems [2]. Clinically, "refractory epigastric pain," which is unresponsive to conventional antispasmodic and analgesic treatments, often prompts a high suspicion of fatal emergencies such as acute myocardial infarction, aortic dissection, severe acute pancreatitis, or hollow organ perforation. However, when these common causes are excluded, esophageal emergencies are easily overlooked [3]. Due to neuroanatomical reflex pathways, some acute esophageal lesions can present with isolated epigastric pain radiating to the back, rather than typical retrosternal pain or dysphagia, leading to early misdiagnosis or missed diagnosis [4].

Infectious esophagitis usually occurs secondary to immunocompromised hosts, such as patients receiving

long-term immunosuppressants, those with malignancies, or those with uncontrolled diabetes [5]. Although *Candida*, herpes simplex virus, and cytomegalovirus are the most common pathogens, bacterial esophagitis should not be underestimated. *Klebsiella pneumoniae* (*K. pneumoniae*), an opportunistic pathogen of the Enterobacteriaceae family, commonly colonizes the human gastrointestinal tract and can cause microbial translocation and invasive infections when the mucosal barrier is compromised and immunity is impaired [6]. Recently, the global spread of hypervirulent *K. pneumoniae* (hvKp) has attracted widespread attention; its infections progress rapidly, are highly prone to triggering sepsis and septic shock, and have extremely high mortality rates. However, cases of acute infectious esophagitis caused by *K. pneumoniae* rapidly progressing to septic shock are extremely rare in clinical practice, with only a few reports in the literature.

In critically ill patients with hemodynamic instability, endoscopy, the "gold standard" for diagnosing esophagitis, is often contraindicated, posing a significant challenge for early etiological diagnosis. Metagenomic next-generation sequencing (mNGS), with its advantages of being culture-independent, having high throughput, a low detection limit, and the ability to identify unknown pathogens, provides a rapid and precise etiological diagnostic approach for severe infections of unknown origin [7].

This article reports a critical case of acute *K. pneumoniae* infectious esophagitis confirmed by blood next-generation sequencing (NGS), which presented with intractable epigastric pain and previously undiagnosed diabetes, eventually progressing to septic shock. By analyzing the diagnostic and therapeutic process of this case, we aim to explore the clinical characteristics of atypical esophageal epigastric pain, the pathogenic mechanism of invasive *K. pneumoniae* infection in immunocompromised hosts, and the core value of NGS in early precision-targeted therapy [8], hoping to provide a reference for the clinical management of such occult and life-threatening emergencies.

Case Presentation

A 52-year-old man presented to the ED with a 3-day history of abdominal pain that had worsened over the 3 hours prior to admission. Three days earlier, he developed dull epigastric pain with back discomfort without any apparent provocation, which was tolerable and initially ignored. Three hours before admission, the epigastric pain and back pain progressively worsened, accompanied by dry mouth, thirst, dizziness, cough, expectoration, fever, dyspnea, dysphagia, palpitations, and fatigue. He took analgesics at home (details unknown) without relief. He denied nausea, vomiting, hematemesis, cessation of flatus or bowel movements, and melena. His past medical history included hypertension, hyperlipidemia, and gout for several years, with irregular medication use.

On admission, his vital signs were as follows: heart rate (HR), 82 bpm; respiratory rate (RR), 20 breaths/min; blood pressure (BP), 161/87 mmHg; and peripheral oxygen saturation (SpO_2), 96%. Physical examination revealed a soft abdomen with epigastric tenderness, no rebound tenderness, a non-palpable liver and spleen, a negative Murphy's sign, and active bowel sounds. The emergency electrocardiogram (ECG) showed no abnormalities. Random blood glucose was 24.36 mmol/L. Other physical findings and laboratory indices were unremarkable. Non-contrast computed tomography (CT) of the upper abdomen showed a slightly enlarged gallbladder, no pancreatic swelling or peripancreatic exudation, splenomegaly, bilateral renal cysts, small bilateral renal stones (largest 0.3 cm), significant gastric contents, calcification of the abdominal aorta and its branches, and thickening of the lower esophageal wall.

He was initially treated with non-invasive ventilation, an insulin pump for glycemic control, fluid resuscitation, electrolyte supplementation, and empirical ceftriaxone. For severe pain, he was sequentially administered dezocine and phloroglucinol, but the persistent epigastric pain remained unrelieved and was accompanied by persistent thirst and polydipsia. An intramuscular injection of pethidine provided transient relief, but the severe pain recurred (pain score: 9/10), necessitating a continuous intravenous morphine infusion. Four hours after admission, his temperature spiked to 39°C and was

managed with antipyretics. Myoglobin and troponin T were mildly elevated, creatine kinase-MB (CK-MB) was normal, and D-dimer was 2.55 mg/L.

An urgent multidisciplinary team (MDT) consultation concluded that there was no indication for cardiac intervention. Gastroenterologists noted the CT finding of esophageal thickening but advised against endoscopy because of hemodynamic instability, suggesting that it be performed once the patient had stabilized. The surgical team found no acute indication for intervention. Subsequently, a contrast-enhanced CT of the chest and whole abdomen, including pulmonary CT angiography (CTA), demonstrated diffuse and marked thickening of the entire esophageal wall with luminal narrowing, blurred surrounding fat planes with streaky opacities, a soft tissue nodule lateral to the trachea and upper esophagus (short-axis diameter approximately 2.2 cm, likely an enlarged lymph node), and multiple enlarged mediastinal lymph nodes.

Due to markedly elevated infection markers, severe shock, and lactic acidosis, a diagnosis of septic shock was established. He was emergently transferred to the intensive care unit (ICU), intubated for mechanical ventilation, and received fluid resuscitation and continuous renal replacement therapy (CRRT) for endotoxin adsorption. Empirical antimicrobial therapy was sequentially escalated with imipenem, vancomycin, levofloxacin, polymyxin, and caspofungin. Supportive measures included inhaled nitric oxide (NO) to improve ventilation-perfusion matching, bronchoscopy for sputum drainage, red blood cell and plasma transfusions, anti-inflammatory therapy, antispasmodics, and glycemic control.

On the day of ICU admission, fungal (1,3)-β-D-glucan and galactomannan (GM) tests were negative, and blood samples were sent for NGS. The NGS results revealed *Klebsiella pneumoniae* (6,124 reads), *Staphylococcus aureus* (48 reads), JC polyomavirus (43 reads), Epstein-Barr virus (20 reads), and *Candida parapsilosis* (161 reads). The resistance genes of *K. pneumoniae* indicated resistance to penicillins and third-generation cephalosporins. Bilateral pleural effusions progressively accumulated, and percutaneous drainage was performed. Cytopathological examination of the bronchoalveolar lavage fluid (BALF) showed no malignant cells but revealed fungal organisms morphologically consistent with *Candida*.

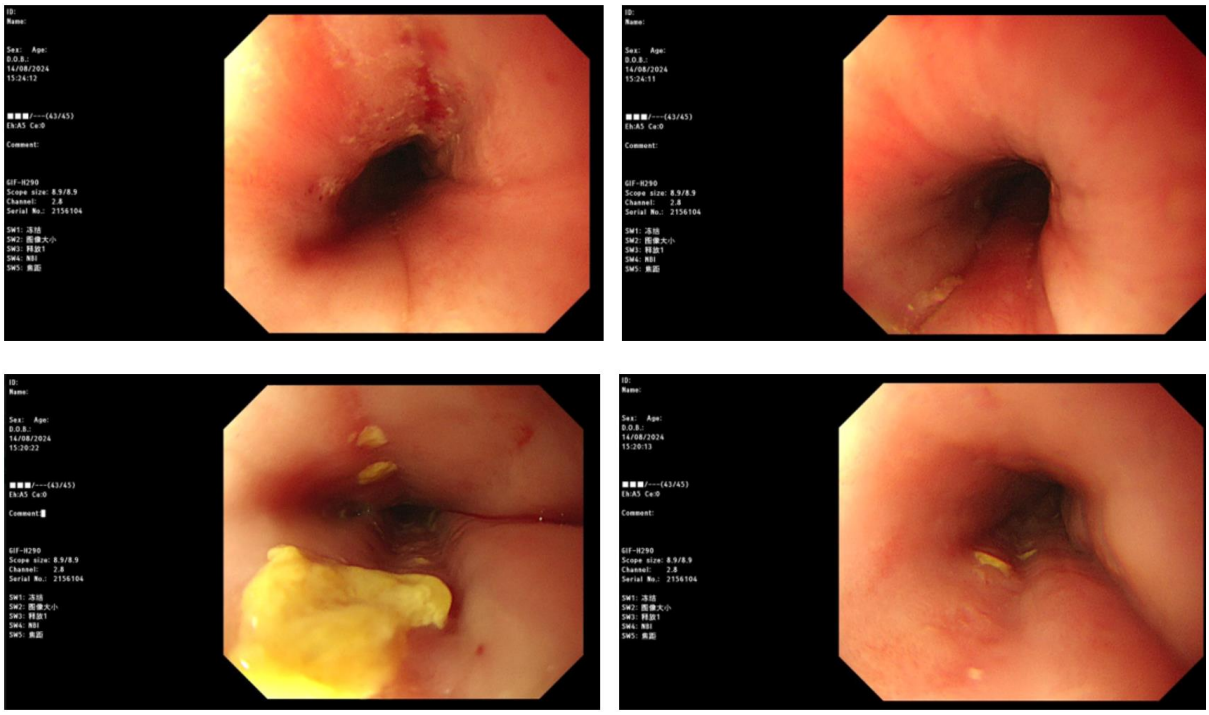

Fig-1:

Gastroscopy revealed that the surface of the esophagus was covered with a large amount of purulent substances that could be flushed away, and the esophageal mucosa was swollen.

After treatment, the septic shock and acute respiratory distress syndrome (ARDS) improved. Vasopressors and ventilatory support were gradually weaned, and infection markers decreased. On day 7, when the patient's vital signs had stabilized, CRRT and inhaled NO were discontinued, and endoscopy was performed. It revealed that the esophageal lumen was coated with copious white mucus and yellow-white flocculent material, which could be washed away (**Fig-1**). The mucosa was swollen but smooth and soft, with poor distensibility upon insufflation and blurred vascular patterns, without abnormalities under narrow-band imaging (NBI). Multiple biopsy specimens were soft. The endoscopic diagnosis was suspected inflammatory esophageal mucosal swelling and chronic atrophic active gastritis. Histopathological examination confirmed inflammatory changes.

However, a follow-up computed tomography (CT) scan on August 21, 2024, showed slight persistent thickening of the entire esophageal wall with a drainage tube in situ, blurred periesophageal fat spaces, and patchy low-density shadows with gas loculi lateral to the mid-upper esophagus, poorly demarcated from the esophagus, suggesting localized fluid collection and mediastinal infection with slightly enlarged mediastinal lymph nodes. Unfortunately, on August 28, 2024, the patient developed severe shock again. Despite the administration of metaraminol, pituitrin, fluid resuscitation, and sodium bicarbonate, the family requested discharge after being informed of the critical prognosis and treatment risks.

Discussion

The Diagnostic Fog: Esophageal Emergencies Characterized by Refractory Epigastric Pain:

Acute epigastric pain is a common ED presentation with a complex etiology and is highly susceptible to delayed or missed diagnosis. This patient presented with "intractable epigastric pain radiating to the back," unresponsive to multiple analgesics, including phloroglucinol, dezocine, pethidine, and even morphine. Such refractoriness to conventional analgesia strongly indicates a critical condition. Typically, epigastric pain prioritizes the exclusion of biliary diseases, acute pancreatitis, hollow organ perforation, and fatal cardiovascular events (e.g., myocardial infarction (MI) and aortic dissection) [2]. In this case, the initial electrocardiogram (ECG) and cardiac markers were unremarkable, and CT ruled out biliopancreatic and vascular emergencies but notably indicated "thickening of the lower esophageal wall." Within hours, the patient rapidly developed high fever, shock, and coagulopathy. This serves as a crucial clinical caveat: when common acute abdominal emergencies are excluded and empirical analgesia fails, severe infection at an atypical site must be highly suspected. If CT shows esophageal abnormalities coupled with escalating infection markers, esophageal-origin infection should be integrated into the differential diagnosis rather than dismissed as an incidental finding.

Etiology and Pathogenesis: K. pneumoniae Invasion in the Setting of Diabetes:

Infectious esophagitis (IE) is typically observed in immunocompromised individuals and is rare in healthy populations [9]. Although this patient denied a history of diabetes, his admission random blood glucose was 24.36 mmol/L, suggesting long-term poor glycemic control. Hyperglycemia-induced impairment of immune defense (e.g., diminished neutrophil chemotaxis and phagocytosis) laid the foundation for this opportunistic infection. *Klebsiella pneumoniae* (*K. pneumoniae*), an opportunistic pathogen of the Enterobacteriaceae family, commonly colonizes the nasopharynx and gastrointestinal (GI) tract [10]. Blood NGS confirmed *K. pneumoniae* as the pathogen. Combined with his history of consuming wild mushrooms and fermented tofu prior to symptom onset, the proposed pathogenic mechanism is as follows: binge eating or irritating foods caused physical or chemical damage to the esophageal mucosa, allowing colonized *K. pneumoniae* in the GI tract to translocate and invade the compromised mucosa; alternatively, the ingestion of *K. pneumoniae*-contaminated food directly caused the infection. In the setting of compromised immunity, *K. pneumoniae* invaded the esophageal wall, triggering acute inflammation. Unable to be cleared by the immune system, the pathogen rapidly breached the mucosal barrier into the bloodstream, precipitating systemic inflammatory response syndrome (SIRS) and septic shock, and eventually spreading to the mediastinum and pleural cavities (bilateral pleural effusions and secondary *Candida* infection) [11].

Advancing the Diagnostic Timeline: The Value of Imaging and NGS:

Flexible high-resolution endoscopy is the gold standard for diagnosing infectious esophagitis. However, in the early phase, endoscopy lacked a clear indication; by the time it was considered to clarify the diagnosis, the patient's hemodynamic instability rendered it absolutely contraindicated. Under such circumstances, imaging and etiological testing became the keys to breaking the impasse. The contrast-enhanced CT not only ruled out fatal conditions such as pulmonary embolism (PE) and aortic dissection but also clearly delineated "diffuse esophageal wall thickening with blurred periesophageal fat spaces," providing vital radiological clues for an esophageal source. Etiologically, traditional cultures are time-consuming and have limited positivity rates. The application of NGS rapidly detected *K. pneumoniae* (6,124 reads) in the blood. NGS boasts high throughput, requires no preselected targets, and has a low detection limit. For severe infections of unknown origin that progress rapidly or involve mixed infections (such as the *Candida parapsilosis* detected later in this case), early NGS significantly improves pathogen detection rates, guiding the transition from empirical to targeted antimicrobial therapy and gaining precious time for early precision treatment [8].

Therapeutic Reflections: Early Antimicrobial Therapy and Source Control:

The core of treating bacterial esophagitis lies in early, adequate broad-spectrum antibiotics and aggressive source control. Although empirical ceftriaxone was administered initially, the patient's condition deteriorated precipitously. The reasons are twofold: first, *K. pneumoniae* is prone to drug resistance, and the initially used third-generation cephalosporin failed to provide adequate coverage [12]; second, the infectious source lacked timely physical intervention. After ICU admission, under hemodynamic support, antimicrobial therapy was immediately escalated to meropenem, combined with continuous renal replacement therapy (CRRT) for endotoxin adsorption and inflammatory factor clearance, alongside anti-inflammatory agents. This successfully stabilized the patient's vital signs, creating conditions for subsequent endoscopy and pleural drainage. This case warns that, for patients highly suspected of severe esophageal infection, clinicians should not wait for culture results; broad-spectrum carbapenems should be initiated immediately after specimen collection, alongside aggressive complication management, striving for early infection control before the condition spirals out of control.

Conclusion

This case reports an instance of acute *K. pneumoniae* infectious esophagitis presenting with refractory epigastric pain and complicated by septic shock. It highlights that atypical presentations of acute esophageal infection are potential pitfalls in the ED. When confronting patients with refractory epigastric pain, clinicians should establish the following diagnostic logic. First, "treatment response is a diagnostic clue"; ineffectiveness of conventional analgesia often portends organic damage or severe infection. Second, "immunodeficiency reshapes the disease spectrum"; in immunocompromised populations, such as those with diabetes, opportunistic pathogens (such as *K. pneumoniae*) can breach mucosal barriers to cause primary esophageal infections that rapidly evolve into septic shock. Third, "technology empowers precision intervention"; when hemodynamic instability precludes endoscopy, CT evidence of esophageal wall thickening serves as a crucial breakthrough, while blood NGS offers an irreplaceable shortcut for early etiological tracing in severe infections. Early recognition, broadened differential thinking, and precise etiological identification are the keys to curbing the deterioration of such occult infectious foci.

Conflict of Interest

The authors have read and approved the final version of the manuscript. The authors declare no conflicts of interest.

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