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Acute Diffuse Phlegmonous Esophagitis Involving the Entire Esophagus with Septic Shock: A Case Report and Literature Review

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Abstract:

Background: Acute diffuse phlegmonous esophagitis is a relatively rare bacterial inflammation of the esophagus. It is characterized by an acute, diffuse, purulent infection involving the submucosal and serosal layers, resulting in phlegmonous-like features. There are only a few case reports in the medical literature, and early diagnosis of this condition presents challenges.

Case Presentation: This report presents a 73-year-old male patient with a history of type 2 diabetes, hypertension, gout, and chronic alcohol use, with poorly controlled blood glucose. The patient presented with upper abdominal pain, fever, shortness of breath, and dysphagia. Initial symptoms included severe upper abdominal pain, fever, and respiratory distress. Chest and abdominal enhanced CT scans revealed thickening and swelling along the entire esophagus, with narrowing of the lumen. After contrast enhancement, the esophagus showed ring-like enhancement. Upper gastrointestinal endoscopy showed mucosal swelling. There was no ulceration or perforation. The diagnosis of acute diffuse phlegmonous esophagitis was considered after excluding other common acute abdominal and chest pain conditions. The condition progressed rapidly, leading to septic shock. Next-Generation Sequencing of blood was positive for hypervirulent Klebsiella pneumoniae (hvKp). Despite active treatment, the patient's condition was initially controlled; however, a follow-up chest CT showed multiple air pockets around the esophagus. The patient subsequently developed septic shock again and died despite active treatment.

Conclusion: Hypervirulent Klebsiella pneumoniae infection causing acute diffuse phlegmonous esophagitis involving the entire esophagus is a rapidly progressing condition that can quickly lead to septic shock. This report aims to alert clinicians to this rare disease and emphasizes the need for heightened awareness in cases of severe upper abdominal pain, where atypical abdominal pain may mask potentially fatal chest pain conditions. Early and accurate diagnosis and timely treatment are essential.

Keywords

Acute Diffuse Phlegmonous Esophagitis, Septic Shock, Hypervirulent Klebsiella Pneumoniae

Abbreviations

CT: Computer Tomography; hvKp: Hypervirulent Klebsiella Pneumonia; NGS: Next-Generation Sequencing; NGT: Nasogastric Tube; MRI: Magnetic Resonance Imagin

Introduction

The most common site for gastrointestinal phlegmon is the stomach, while phlegmonous inflammation affecting the esophagus, small intestine, or colon is relatively rare [1,2]. Acute diffuse phlegmonous esophagitis is a rare but potentially life-threatening bacterial esophageal inflammation characterized by diffuse infection of the submucosal and serosal layers [3,4]. Delayed early diagnosis and treatment can lead to severe complications, including esophageal perforation, mediastinal infection, pleural effusion, septic shock, respiratory failure, and even death [5–7]. Long-term complications may include esophageal stricture and fistula formation [8]. This case report presents an acute diffuse phlegmonous esophagitis involving the entire esophagus due to hypervirulent *Klebsiella pneumoniae* infection, leading to septic shock. Additionally, the clinical features and treatment strategies for this condition are reviewed in light of existing literature.

Case Presentation

A 73-year-old male patient presented to the emergency department (ED) with a three-day history of upper abdominal pain, progressive fever, and dyspnea. The patient had a medical history of hypertension, type 2 diabetes, gout, chronic alcohol consumption, and poorly controlled glucose levels. On examination, the patient was conscious. Vital signs were as follows: body temperature of 38.5°C, heart rate of 82 beats per minute, respiratory rate of 22 breaths per minute, blood pressure of 161/75 mmHg, and oxygen saturation of 95%. The abdominal physical examination revealed that the whole abdomen was soft, with tenderness noted in the area below the xiphoid process. Furthermore, the presence of rebound tenderness and muscle tension was not observed. Lung auscultation revealed coarse breath sounds, while other system examinations showed no significant abnormalities.

Chest and abdominal enhanced CT scans showed a small amount of bilateral pleural effusion, marked thickening of the esophageal wall along its entire length, narrowing of the lumen, and blurring of the surrounding fat spaces with streaky shadows. Additionally, there was an increase in the number of lymph nodes in the upper trachea, esophagus, and mediastinum. Arterial blood gas analysis revealed hypoxemia with a partial pressure of oxygen (PO_2) of 60 mmHg and a lactate level of 2.0 mmol/L. Laboratory examination revealed a white blood cell count of 8.47 × 10^9/L with 84.8% neutrophils. Hemoglobin was 153 g/L, and platelet count was 75 × 10^9/L. Albumin was 30.4 g/L, creatinine was 142 μmol/L, and urea was 7.9 mmol/L. Interleukin-6 was elevated at >5000.00 pg/ml, and procalcitonin was >100.000 ng/ml. The patient's HbA1c level was 9.9%. Myocardial markers were as follows: myoglobin 250.00 ng/ml, B-type natriuretic peptide precursor 4343 ng/L, and troponin-T 27.5 ng/L.

On admission, the patient presented with persistent severe pain, rated 8 on the visual analog scale. Despite the administration of tramadol, dezocine, phloroglucinol, and morphine, the patient's pain remained inadequately controlled. Upon admission, piperacillin-tazobactam was started for infection control, along with fluid therapy and nutritional support. On day 2 of hospitalization, the patient's dyspnea worsened progressively, with a decreased oxygenation index and declining blood pressure. The patient underwent endotracheal intubation and was placed on ventilator-assisted breathing, while vasoactive drugs were administered for shock. The patient was prescribed meropenem, a more potent antibiotic, and also underwent endotoxin adsorption therapy. Continuous infusion of hydrocortisone was administered to improve shock, along with dobutamine, levosimendan, and digoxin to support cardiac function. Additionally, anticoagulation was adjusted, blood glucose controlled, and electrolyte balance and acid-base status maintained.

Blood NGS testing was positive for hypervirulent *Klebsiella pneumoniae*. The organism showed resistance to penicillin and third-generation cephalosporins. Virulence gene screening revealed positivity for *iutA* and *rmpA2* genes. On the seventh day, an upper gastrointestinal endoscopy was performed, which revealed significant amounts of white mucus and yellow-white secretions in the esophagus. The secretion was removed upon irrigation, but the mucosa remained swollen, smooth in appearance, and soft in texture. The mucosa was poorly distensible upon insufflation. Pathology revealed squamous epithelial hyperplasia.

After treatment, the patient's shock and acute respiratory distress syndrome improved. The vasopressors and inotropic drugs were gradually reduced, and ventilator support was tapered. On day 13, the endotracheal tube was removed, and the patient was placed on non-invasive ventilation as part of sequential therapy. On day 14, a follow-up chest and abdominal CT scan revealed slight thickening of the entire esophageal wall, blurred fat spaces around the esophagus, and streaky shadows. Multiple air collections were observed around the esophagus with poorly defined borders and possible localized pleural effusion. Bilateral pleural effusions were also noted (**Fig-1** and **Fig-2**).

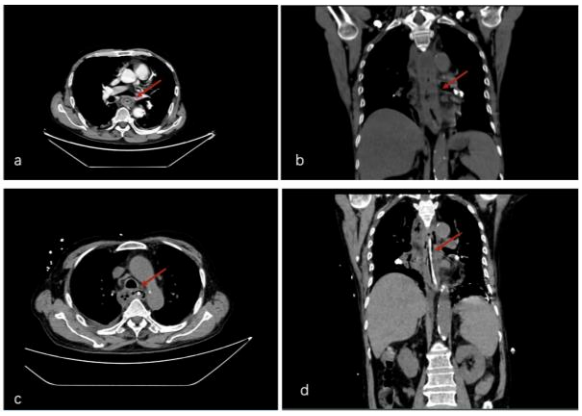

Fig-1:

Enhanced chest CT on admission showing significant thickening of the esophageal wall, narrowing of the lumen, and edge enhancement (a,b); Chest CT on day 14 showing mild thickening of the esophageal wall, with multiple air collections around the esophagus and localized pleural effusion (c,d).

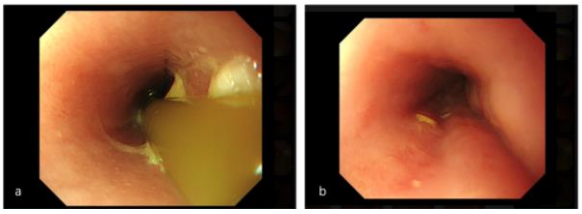

Fig-2:

a: Endoscopic view showing a large amount of yellow-white and fibrinous material covering the esophageal mucosa; b: After irrigation, esophageal mucosal swelling was observed.

Despite all supportive treatments, on day 15 of hospitalization, the patient experienced recurrent chills and high fever, followed by worsening shock, and ultimately succumbed to septic shock and passed away. A follow-up review on day 21 showed normalization of the laboratory findings, as indicated in **Table-1**.

Discussion

Acute diffuse phlegmonous esophagitis is a rare and rapidly progressing life-threatening condition. To date, only a small number of case reports exist. Among the 35 cases of acute phlegmonous esophagitis reported in the literature, 3 patients died [6]. The hallmark of phlegmonous esophagitis is the infection of the submucosal layer of the esophagus, rather than inflammation of the mucosal layer [9]. Common risk factors for phlegmonous esophagitis include alcohol abuse, diabetes, a history of endoscopic procedures, malignancy, and immune deficiency states [9,10]. In our case, the patient's risk factors included uncontrolled type 2 diabetes and chronic alcohol abuse. The normal esophageal microbiota is predominantly composed of Gram-positive bacteria, especially streptococci, while the microbiota in esophageal inflammation is mainly comprised of Gram-negative bacteria [11]. Pathogens identified in case reports of phlegmonous esophagitis include *Streptococcus* species [5,12], *Bacillus* species [13,14], *Acinetobacter baumannii* [3], oral anaerobes [15], *Pseudomonas aeruginosa* [16], and *Klebsiella pneumoniae* [2,4,16–18], with *Klebsiella pneumoniae* being the most commonly identified pathogen. Based on virulence and pathogenic characteristics, *Klebsiella pneumoniae* can be categorized into common and hypervirulent strains [19].

In our case, the patient's blood culture and next-generation sequencing (NGS) indicated *Klebsiella pneumoniae* positivity, and the virulence gene screening revealed the presence of the *iutA* and *rmpA2* genes.

Table-1: Blood Investigations of the Patient

Laboratory tests	On addimition	Day 2	Day 5	Day 7	Day 14	Day 21
White Blood Cell(10^9/L)	8.47	6.48	7.15	4.94	3.56	3.57
Neutrophils(%)	0.85	0.77	0.76	0.91	0.82	0.78
Platelet Count(10^9/L)	75	80	17	57	79	56
Procalcitonin (ng/ml)	82.5	>100	70.11	22.9	10.5	21.1
Interleukin-6 (pg/ml)	N/A	>5000	473	1060	265	1340

These virulent genes are encoded by plasmids in hypervirulent *Klebsiella pneumoniae* (hvKP) and serve as molecular markers of this strain. Compared to the common *Klebsiella pneumoniae*, hvKP is more commonly associated with community-acquired infections and is more likely to cause invasive infections or metastatic spread, leading to worse prognosis [20]. The patient ultimately succumbed to septic shock, which is likely related to the widespread infection caused by hypervirulent *Klebsiella pneumoniae* affecting the entire esophagus.

According to previous case reports, the diagnosis of acute phlegmonous esophagitis relies on endoscopic examination and CT imaging. Endoscopy may reveal esophageal mucosal edema without purulent discharge or perforation [5], or it may show ulcer formation with purulent exudate leaking from the ulcer [4,14]. In the later stages of treatment, some patients may develop esophageal strictures [8]. In this case, endoscopy revealed the presence of yellow-white mucus adhering to the esophageal mucosa, which could be removed upon irrigation. The entire esophageal mucosa appeared swollen without obvious ulcers or fistulas. On enhanced CT, acute phlegmonous esophagitis typically presents as a circumferential low-density area within the esophageal wall, with surrounding esophageal and gastric wall enhancement [17]. If there are bubbles within the thickened esophageal wall, this suggests infection by gas-producing pathogens, which is a distinguishing feature of phlegmonous esophagitis [4]. A case reported by Motohiro Shimizu was the first to confirm that magnetic resonance imaging (MRI) could be used in the diagnosis of acute phlegmonous esophagitis to differentiate between esophageal abscesses and edema [5]. However, there is still a lack of additional cases to support the notion that MRI is superior to enhanced CT in diagnosing acute diffuse phlegmonous esophagitis.

Although early enteral nutrition via a nasogastric tube (NGT) can help critically ill patients achieve adequate nutrition, caution is necessary for patients with esophageal involvement, particularly those with esophageal mucosal damage. Whether early enteral nutrition through a gastric tube is necessary in such cases remains a matter of careful consideration. Po-Chih Chang et al. reported a case of acute phlegmonous esophagitis secondary to deep cervical infection, where insertion of an NGT led to the formation of an esophageal wall dissection and subsequent rupture of the esophageal mucosa. This complication unexpectedly resulted in drainage of a submucosal abscess. While the final outcome was beneficial, this case highlights the need for caution when placing an NGT in patients with potential esophageal pathology, to avoid catastrophic complications such as gastrointestinal injury or esophageal perforation [18]. For patients expected to require long-term enteral nutrition, a jejunostomy tube may be considered. This approach bypasses the esophagus, alleviating mechanical irritation and reducing the risk of aspiration or vomiting due to swallowing difficulties. Moreover, compared to total parenteral nutrition, early enteral nutrition helps maintain normal physiological function of the gut, preserving intestinal barrier integrity, preventing intestinal atrophy, reducing dysbiosis, and lowering the risk of intestinal infections [21].

The treatment of phlegmonous esophagitis primarily involves broad-spectrum antibiotics, endoscopic mucosal debridement with intraluminal drainage [4], endoscopic esophageal drainage [22,23], and surgical intervention [24]. Endoscopic abscess drainage is a minimally invasive and effective procedure. However, due to potential esophageal mucosal injury, complications such as esophageal stricture may arise. To minimize the risk of complications, it is recommended to perform an incision at the base of the abscess, limiting the extent of esophageal mucosal resection [8].

For elderly patients presenting to the emergency department with a history of chronic conditions such as type 2 diabetes, chest CT may reveal esophageal thickening with swelling and severe pain that is unbearable. In addition to considering acute phlegmonous esophagitis, other critical conditions with atypical clinical symptoms, such as acute coronary syndrome, aortic syndrome, and pulmonary embolism, must also be considered. Furthermore, some cases of acute phlegmonous esophagitis may present with chest pain [3]. Therefore, it is essential to rule out these life-threatening conditions when diagnosing phlegmonous esophagitis. Acute phlegmonous esophagitis has a rapid onset and a short course. It progresses quickly in some

patients, leading to a poor prognosis [13,25]. Early identification and prompt and effective treatment may help improve the patient's prognosis.

Conclusion

Hypervirulent *Klebsiella pneumoniae* infection causing acute diffuse phlegmonous esophagitis involving the entire esophagus is a rapidly progressing condition that can quickly lead to septic shock. This report aims to alert clinicians to this rare disease and emphasizes the need for heightened awareness in cases of severe upper abdominal pain, where atypical abdominal pain may mask potentially fatal chest pain conditions. Early and accurate diagnosis and timely treatment are essential.

Consent for Publication

Written informed consent was obtained from the patient's son for the publication of this case report and any accompanying images.

Ethics Approval and Consent to Participate

This study was approved by the Ethics Committee of West China Hospital of Sichuan University. Written informed consent was obtained from the individual and his son for the publication of any potentially identifiable images or data included in this article.

Data Availability Statement

The original contributions generated for the study are included in the article; further inquiries can be directed to the corresponding authors.

Conflict of Interest

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

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