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Rapid Progression of Fulminant Septic Shock with Multiorgan Failure in a Previously Healthy Individual

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Abstract

INTRODUCTION

Sepsis is a clinical syndrome resulting from the invasion of microorganisms into the body, causing a considerable systemic inflammatory response. Streptococcus pneumoniae is a gram-positive bacterium and the most common cause of pneumonia and meningitis. The spleen plays a crucial role in eliminating bacteria from the bloodstream. In individuals without a spleen or with its functional hyposplenism, there is a high risk of developing fulminant sepsis with potentially fatal outcomes.

CASE REPORT

A 36-year-old man was presented to the emergency department at 1:00 PM with paraesthesiae, leg pain, a petechial rash on the face, and a subfebrile temperature (37,5 °C) that had been present since the previous day. On admission, purpura was noted on the head and extremities, raising suspicion of disseminated intravascular coagulation. Due to neurological symptoms, meningococcal sepsis was also considered, despite the absence of meningeal signs. Based on clinical findings of tachycardia and hypertension, along with elevated D-dimer levels (> 35.00 mg/L), pulmonary embolism was suspected but later ruled out by MSCT pulmonary angiography. Hypoxemia and tachycardia raised suspicion of pneumonia, which was ruled out as well.

Laboratory results received at 2:20 PM showed leukopenia (2.89 × 10⁹/L), thrombocytopenia (14 × 10⁹/L), prolonged prothrombin time and elevated C-reactive protein (161.3 mg/L). Empirical antimicrobial therapy with meropenem, vancomycin, and ceftriaxone was initiated immediately. Procalcitonin levels at 3:00 PM were 40.30 µg/L and increased to 55.65 µg/L by 7:00 PM, indicating sepsis with multiorgan failure. Echocardiography revealed severe impairment of myocardial systolic function with global hypokinesia. The patient died at 8:00 PM. Autopsy revealed a rudimentary spleen, and microbiological analysis of blood cultures performed the following day confirmed the causative pathogen as Streptococcus pneumoniae.

CONCLUSION

The rapid onset of fulminant sepsis with multiorgan failure and a fatal outcome in a patient with an unrecognized rudimentary spleen points out the importance of identifying splenic anomalies and the immediate initiation of antimicrobial therapy, particularly in asplenic patients.

***KEYWORDS**: sepsis; spleen; Streptococcus pneumoniae.*

INTRODUCTION

Sepsis is a life-threatening condition that leads to organ dysfunction as a result of a dysregulated host response of the body to infection. The most common symptoms include fever, tachycardia, chills, and tachypnoea. In addition to fever, sepsis may also present with hypothermia, and various skin manifestations, such as petechiae, may occur. When these symptoms are accompanied by internal organ damage or impairment of its function, a diagnosis of sepsis is established. The development of sepsis requires both the virulence of the causative pathogen and a compromised immune system. Sepsis remains a frequent cause of mortality worldwide.

In individuals with intact mechanical barriers (such as the skin), infection will not develop unless there is a breach in the barrier. Consequently, individuals with impaired mechanical barriers are more susceptible to life-threatening infections. Other factors, including immunodeficiencies, genetic predispositions, and morphological malformations also increase susceptibility to sepsis. Optimal management of sepsis requires early recognition and prompt initiation of treatment.

Septic shock represents the most severe form of sepsis and is characterized by hypotension leading to tissue hypoxia. The diagnosis of septic shock is established when the mean arterial pressure remains below 65 mmHg despite administration of 500 mL of crystalloid solution and serum lactate levels exceed 2 mmol/L. Timely antimicrobial therapy is crucial for improving sepsis outcomes. In addition to antimicrobial treatment, supportive therapy with intravenous fluids is required. Appropriate antibiotic selection depends on knowledge of the aetiology, pathogenesis, and antimicrobial susceptibility of the causative organism.

Mortality from sepsis and septic shock remains very high and continues to represent a major public health concern. Asplenia is a condition in which an individual lives without a spleen or has a present but nonfunctional spleen. Under normal circumstances, the spleen is located in the left upper quadrant of the abdomen and is not palpable during physical examination. Its primary functions include the filtration and removal of senescent blood cells, and it plays a critical role in protecting the host against encapsulated bacteria. The lack of protective function renders asplenic individuals particularly vulnerable to life-threatening infections caused by encapsulated bacteria.

CASE REPORT

A 36-year-old man was presented to the emergency department at 1:00 PM due to generalized weakness. Heteroanamnestic information revealed that he had felt fatigued the previous day, but as he was travelling, did not attribute this to illness. He spent the evening with his family at a restaurant, after which he noticed a subfebrile temperature (37.5 °C). He took 1 g of paracetamol and reported feeling well afterward. During the night, he had several episodes of diarrhoea, and a black stool was noted in the morning. On the morning prior to presentation to the emergency department, he developed a purplish discoloration of the face.

On the day of admission, he again took paracetamol due to a subfebrile state. Upon arrival, he was confused, with purpura present on the face, neck, and legs. Petechial rash was present as well in the axillary and suprapubic regions. His pulse was weak and filiform, and breathing was shallow and rapid. He complained of abdominal and leg pain. Physical examination revealed an oxygen saturation of 77% and tachycardia (135 beats per minute) without audible cardiac murmurs. Bilateral crackles were auscultated, with diminished breath sounds over the right lung and a respiratory rate of 35 breaths per minute. His skin was cold. Meningeal signs were absent.

Blood samples were obtained and sent for laboratory and microbiological analysis. Laboratory results available one hour after admission (1:57 PM) showed leukopenia (2.89×10^9/L) and thrombocytopenia (14×10^9/L). Prothrombin time was reduced (0.29s), and D-dimer levels were markedly elevated (>35.00). C-reactive protein was 163.1 mg/L, and procalcitonin was 40.30 µg/L. Chest radiography revealed reduced aeration of the right lung parenchyma with a small pleural effusion.

An urgent computed tomography (CT) angiography of the aorta was performed due to suspicion of aortic dissection, which was excluded. Multi-slice CT (MSCT) pulmonary angiography also ruled out pulmonary embolism, which had been suspected based on elevated D-dimer levels and clinical findings. MSCT additionally revealed abnormalities of both adrenal glands. Transthoracic echocardiography demonstrated severe global hypokinesia with a left ventricular ejection fraction of 20%. Supportive and vasopressor therapy was initiated immediately. Empirical antibiotic therapy with meropenem, vancomycin, and ceftriaxone was started. Dexamethasone was administered due to severe septic collapse with multiorgan failure. At 3:45 PM, the patient was sedated and electively intubated. Bronchoscopy performed after intubation revealed no abundant secretions. However, bronchoalveolar lavage samples were obtained from both the left and right tracheobronchial trees.

At 5:15 PM, the patient's clinical condition deteriorated with signs of bradycardia and hypotension, and cardiopulmonary resuscitation was initiated. After restoration of cardiac rhythm, in addition to noradrenaline, inotropic and vasopressor support with dobutamine,

vasopressin, and high-dose noradrenaline was instituted. Laboratory results obtained at 7:00 PM indicated a significant increase in procalcitonin levels (55.65 µg/L). At 8:10 PM, the patient died.

Microbiological results received the following day identified the causative pathogen as *Streptococcus pneumoniae*. Autopsy revealed a rudimentary spleen.

DISCUSSION

Fulminant pneumococcal sepsis in an individual who was previously healthy is a rare cause of death. Apparently healthy individuals may have a nonfunctional spleen, which can lead to rapid progression with a fatal outcome. Prompt clinical suspicion of sepsis is essential due to the extremely rapid progression of symptoms. This should be followed by expedited diagnostic evaluation, including measurement of procalcitonin and C-reactive protein levels. When there is a high suspicion or a confirmed diagnosis of sepsis, empirical antibiotic therapy must be initiated as rapidly as possible.

Streptococcus pneumoniae, a common cause of pneumonia, meningitis, and sepsis, is particularly prevalent in asplenic individuals or those with impaired splenic function. A rudimentary spleen reduces the clearance of encapsulated bacteria (such as *Streptococcus pneumoniae*), thereby increasing the risk of sepsis caused by this pathogen and the likelihood of a fatal outcome. In any young, otherwise healthy individual presenting with sepsis of unknown aetiology, asplenia or splenic dysfunction should be considered.

Identified asplenic patients should receive prophylactic vaccination against *Streptococcus pneumoniae, Neisseria meningitidis,* and *Haemophilus influenzae* type B. In these patients, more rapid initiation of antibiotic therapy is required due to the accelerated progression of disease.

CONCLUSION

This case shows how fulminant pneumococcal sepsis can rapidly progress and become fatal in a previously healthy individual with unrecognized asplenia or splenic hypoplasia. Despite early initiation of broad-spectrum antibiotics and intensive supportive treatment, the disease progressed rapidly to multiorgan failure, demonstrating the vulnerability of asplenic and hyposplenic patients.

In young patients with sepsis of unknown origin, functional asplenia should be considered because early recognition and treatment are crucial for the better outcome of such patients.

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