

Unexpected Fatal Empyema in a Previously Healthy Woman

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Abstract

Streptococcus pyogenes is gram + aerob coccus and it is described as group A *Streptococcus* (GAS) according to the Lancefield classification. GAS may cause pneumonia and pleural effusion rarely. Herein we reported a 43 years old previously healthy woman presented to the Emergency Department (ED) with the complaint of chest-back pain and shortness of breath. During her follow up she developed septic shock due to the empyema. 13rd hour of her admission, cardiopulmonary arrest developed and she died in spite of the appropriate and immediate treatment. 2 days later after her admission, streptococcus pyogenes was detected in her pleural effusion and blood culture. In conclusion clinicians should be aware of this potential fatal condition also in healthy patients and start the treatment immediately.

Case Presentation

A 43 years old woman presented to the Emergency Department (ED) with the complaint of chest-back pain and shortness of breath. Her symptoms had begun 3-4 days ago. Her vital signs were: Blood Pressure: 81/52 mmHg, Heart Rate: 140/min, Respiratory rate: 30/min, O₂ sat: 97% (without O₂ support). Her Glasgow Coma Scale score was 15 but she was agitated. In her initial examination: The lung sounds decreased in right hemithorax, the distal peripheral pulses couldn't be palpable. The upper extremities were cold and pale. Her ECG showed only sinus tachycardia. Her arterial blood gases analyses (ABG) were as following: pH: 7.32, pO₂:82, pCO₂: 25, O₂ sat: 98, HCO₃: 15, lactate : 8,9 Aortic dissection and pulmonary embolism were firstly predicted in the differential diagnosis. The triple rule out Computerized Tomography (CT) was applied to the patient. After the CT procedure, bradichardia was developed in her cardiac monitorization at 2nd hour of her admission. She was administered 0.5 mg Atropine iv. Then she was arrested and the arrest rhythm was pulseless electrical activity. After 1 cyclus of the CPR, ROSC was gained. The first ABG after the ROSC revealed ph: 6.82, pO₂:51, pCO₂:77, O₂ sat: 56%, HCO₃:12, lactate:13. The acute phase reactants results were as following: Leucocyte count: 2600 , C-reactive protein:45,9 mg/dL, procalcitonin: 11,98 ng/mL. The CT was reported as massive pleural effusion on the right side of the lung, no pathologic finding on the coronary and pulmonary arteries and also on aorta by the radiologist (Figure 1). A

thoracostomy tube was inserted to the right side of the lung and approximately 2000 cc purulent effusion was drained (Figure 2). Meropenem was administered to the patient for the empyema. She was administered vazopressor agent by titrating the dosage but hypotension was resistant. pH value couldn't be reached over 6,8 during the follow up in spite of NaHCO₃ infusion.

Her relatives were asked whether she took any medications in overdose, recently having an influenza infection including fever, cough; any acutely ill patient in the home living with them. They told she had a boy having cerebral palsy and he didn't hospitalized for a long time. The possible cause of the empyema couldn't be found. Approximately

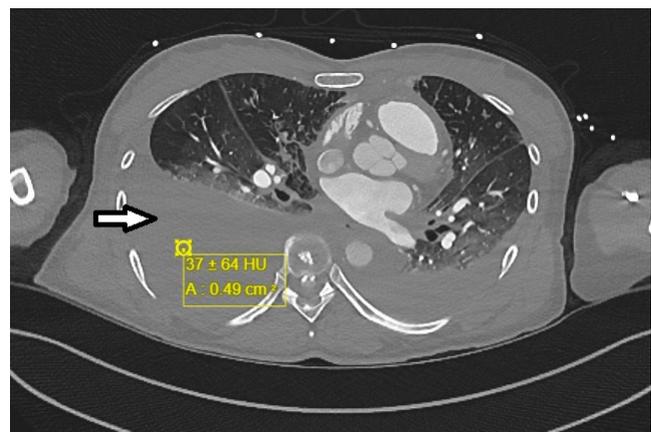


Figure 1: The massive pleural effusion on the right side of the lung with high HU*.

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Figure 2: The drainage chest tube of the patient including purulent effusion.

13rd hour of her follow up, cardiopulmonary arrest developed and ROSC could not be achieved and she died. 2 days later after her admission, streptococcus pyogenes was detected in her pleural effusion and blood culture.

Discussion:

Streptococcus pyogenes is gram + aerob coccus and it is described as group A *Streptococcus* (GAS) according to the Lancefield classification. The exotoxin of this microorganism may cause toxic shock syndrome (TSS). GAS may cause pneumonia rarely and mostly secondary to the viral infection¹. After the development of the pneumonia pleural effusion may occur rapidly and occasionally in the left side of the lung and frequently it is empyema. The prevalence of GAS associated pleural effusion is 0,7% in all pleural effusions². Bacteriemia may develop 80% in pneumonia due to GAS. The 25% of these patients will develop TSS with high mortality rates³. Shock and multiorgan failure will develop in hours because of its exotoxins which cause tissue necrosis rapidly.

The mostly of these patients having a risk factor (minor trauma, routine non steroidal antiinflammatory drug usage, recent surgeries, viral infections [influenza, varicella, HIV], iv drug abuse, malignancy, burns, diabetes mellitus and immunosuppression.). The risk of the GAS associated TSS development in a healthy patient is so low.

Hypotension is resistant in spite of the vasopressor treatment. Our patient had also resistant hypotension and meta-

bolic acidosis. Mortality rate is 30-70%³. The absolute diagnosis can be made by the GAS grown in sterile area cultures like blood, pleural effusion, pericardial effusion, cerebrospinal fluid or surgical wound.

The management includes the treatment of septic shock, the debridement of the tissue necrosis if there is and antibiotic therapy.

Tamayo et al could show *Streptococcus pyogenes* in only one patient among 40 GAS associated pneumonia³. During the 2009 influenza A (H1N1) pandemic, there were increase in hospitalization of the children with the pleural effusions⁴.

Asai et al reported a patient which presented with a severe *Streptococcus pyogenes* empyema following influenza A infection. This patient had no medical history apart from influenza. He was recovered by drainage with intrapleural urokinase and antibiotic therapy⁵.

Sakaia T et al reported a case report in which previously healthy patient presented with empyema and TSS due to *Streptococcus pyogenes* and recovered after the intensive combined therapies including drainage, anti-DIC agent and antibiotics⁶. Our patient was also a healthy woman and despite the detailed history and examinations we couldn't explain the possible cause of this fatal infection. In conclusion clinicians should be aware of this potential fatal condition also in healthy patients and start the treatment immediately.

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References

1. Muller MP, Low DE, Green KA, Simor AE, Loeb M et al. Clinical and epidemiologic features of group A streptococcal pneumonia in Ontario, Canada. *Arch Intern Med* 2003;163:467-72.) *Arch Intern Med* 2003;163:467-72
2. Birch C, Gowardman J. *Streptococcus pyogenes*: a forgotten cause of severe community-acquired pneumonia. *Anaesth Intensive Care* 2000;28:87-90
3. Tamayo E, Montes M, Vicente D, Pérez-Trallero E. *Streptococcus pyogenes* pneumonia in adults: clinical presentation and molecular characterization of isolates. 2006-2015. *PLoS One* 2016;11:e0152640
4. Ampofo K, Herbener A, Blaschke AJ, Heyrend C, Poritz M et al. Association of 2009 pandemic influenza A (H1N1) infection and increased hospitalization with parapneumonic empyema in children in Utah. *Pediatr Infect Dis J*. 2010;29:905-9.
5. Asai N, Suematsu H, Sakanashi D, Kato H, Hagihara M et al. A severe case of *Streptococcus pyogenes* empyema following influenza A infection. *BMC Pulmonary Medicine* (2019) 19:25
6. Sakaia, T, Taniyama D, Takahashi S, Nakamura M, Takahashi T. Pleural empyema and streptococcal toxic shock syndrome due to *Streptococcus pyogenes* in a healthy Spanish traveler in Japan. *IDCases* 9 (2017) 85-88