Septic shock recognition by bedside chest ultrasonography in the emergency department

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Abstract
The case of a young man who presented in the emergency department complaining of sudden onset of cough, fever and haemoptysis is described. Chest ultrasonography rapidly diagnosed an acute respiratory distress syndrome picture, which was confirmed on computed tomography. This allowed prompt management of the patient. Blood cultures yielded \textit{Streptococcus pyogenes} and pharyngeal exudates were positive for influenza B virus.

Keywords
Septic shock; chest ultrasonography; interstitial syndrome; pneumonia; influenza B infection; \textit{Streptococcus pyogenes} pneumonia.

Introduction
The diagnostic use of point-of-care ultrasonography (US) has become widespread in emergency departments in the last few years because it provides a rapid, accurate, non-invasive, painless and low cost examination at the bedside, and has an increasing role in many different diseases\(^1\). Emergency US should be considered a goal-directed focused examination in addition to the physical examination and performed by the emergency physician and intensivist in any emergency setting to answer brief and important clinical questions\(^2\). Using chest US, some conditions can be diagnosed immediately (e.g. pneumonia, atelectasis, pulmonary embolism, pleural as well as pericardial effusion, and pneumothorax) or suspected (e.g. diffuse parenchymal lung disease). The US examination may serve as a guide for the next diagnostic or therapeutic steps\(^3\).

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Case presentation

A 39-year-old man, with a history of asthma and occasional use of bronchodilators, presented at 04:42 h to the emergency department complaining of sudden onset of cough with haemoptysis and fever. He complained of flu symptoms for 1 week for which he had taken non-steroidal antiinflammatory drugs. He had tachycardia (heart rate 124 bpm), tachypnoea (32 apm), and moderate confusion (Glasgow Coma Score (GCS) 14); his blood pressure was 140/70 mmHg, and his oxygen saturation on normal breathing was 90%. Physical examination revealed crackles particularly in the right lung; heart tones were clear and no murmurs were found. An electrocardiogram documented a sinus tachycardia with incomplete right branch block.

Suddenly, he presented repeated haemoptysis attacks. His blood pressure dropped to 60-30 mmHg and he become more and more confused (GCS 11). His oxygen saturation decreased to 60%, so prompt oral intubation was performed with 100% oxygen delivery and he was placed on volume-controlled ventilation. Two peripheral venous lines were achieved through which laboratory tests were drawn, and 1L of crystalloid was rapidly infused. Blood gas analysis revealed pH 7.3, Pco2 31.3 mmHg, Po2 48.1 mmHg, bicarbonates 15.6 mmol/L, lactates 7.4 mmol/L.

Bedside chest ultrasonography, performed according to recent reports for detection of pneumonia in the emergency department[6], showed confluent B lines (white lung) involving almost all the right lung in the absence of fluid effusion; the left lung showed distinct B lines particularly in the middle-basal fields, with an inhomogeneous distribution and evidence of spared areas; associated pleural line abnormalities were detected bilaterally (Fig. 1). Superficial alveolar consolidation areas were found on the posterior scans, predominantly on the right side. Integration with focused cardiac ultrasonography (FOCUS), performed in subcostal view, revealed normal global cardiac systolic function without pericardial effusion, no right or left ventricular...
enlargement or hypertrophy, no significant valvular defects and a collapsed inferior caval vein, according to the reduced volume status\(^7\). These ultrasonography findings provided evidence for a wide interstitial syndrome, and the picture was interpreted as an acute respiratory distress syndrome (ARDS), according to previous studies\(^8\).

Blood tests showed impaired renal and heart function, with serum creatinine 3.02 mg/dL (normal value [NV] 0.67–1.17 mg/dL), sodium 132 mEq/L (NV 135–145 mEq/L), lactate dehydrogenase 386 IU/L (NV <250 IU/L), creatine phosphokinase 123 IU/L (NV 30–170 IU/L), alanine aminotransferase 50 IU/L (NV 7–45 IU/L), N-terminal pro brain natriuretic peptide 7511 pg/mL (NV <150 pg/mL), troponin T high sensitivity 0.011 ng/mL (NV <0.014 ng/mL). The blood count revealed haemoglobin 17 g/dL (NV 13–17 g/dL), platelets 125 \(\times 10^9\)/L (NV 140–450 \(\times 10^9\)/L), severe leucopaenia 0.91 \(\times 10^9\)/L (NV 4.10–9.80 \(\times 10^9\)/L), with 48% of neutrophils over 50% blood cells, 28% lymphocytes and 24% immature forms. C-reactive protein was 22.3 mg/dL (NV <3 mg/dL), procalcitonin 5.88 ng/mL (NV <0.50 ng/mL).

As it was a matter of septic shock, aggressive volume replacement and high-dose vasopressors (dopamine and noradrenaline) were used, but, despite all efforts his blood pressure remained low. A computed tomography (CT) scan of the lungs showed wide areas of lung consolidation involving both lungs but more represented in the right lung, with ground glass zones in the peripheral areas (Fig. 2). Pulmonary embolism and vascular malformations were excluded. After blood cultures were drawn and bronchoalveolar lavage was performed, broad-spectrum antibiotic therapy, including ceftriaxone (4 g/day) and levofloxacin (750 mg/day), together with oseltamivir 75 mg (twice daily), was started. Bloody secretions throughout the ventilator circuit were always observed, so extracorporeal membrane oxygenation (ECMO) procedures were organized, according to recent reports\(^9\). Two central lines were achieved, and high-dose adrenaline was continually infused without results. Repeat arterial blood gas measurements revealed a worsening condition, with pH 6.9, Pco\(_2\) 56.3 mmHg, Po\(_2\) 50.7 mmHg, So\(_2\) 62%, lactates 7 mmol/L, bicarbonates 13.3 mmol/L despite mechanical ventilation. At 08:40 h, a huge bleed from the tube was observed before beginning ECMO; at 08:50 h the patient had a cardiac arrest in asystole. Prompt reanimation according to Advanced Life Support guidelines was started but no return of spontaneous circulation was obtained and the patient was pronounced dead at 09:30 h.

\textit{Streptococcus pyogenes} was identified in all the blood cultures and reverse transcriptase polymerase chain reaction on pharyngeal exudates was positive for influenza B virus. Rheumatology workup, including antinuclear antibody, anti-double stranded antibody, antineutrophil cytoplasmic antibodies, and anti-glomerular basement membrane antibody, were negative. Autopsy showed a wide and complete alveolar haemorrhage, with 2 L of blood collected in the ribcage. Microscopically, alveoli were filled with pus and exudates with macrophages and germs. No other signs of vasculitis were found in other organs; these findings suggest a case of viral alveolar haemorrhage with a superinfection due to \textit{Streptococcus pyogenes}, which finally led to sepsis.

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{Fig2.png}
\caption{Lung CT scan showing involvement of the right lung more than the left lung, with ground glass appearance in the peripheral fields.}
\end{figure}
**Discussion**

Invasive group A streptococcal infections cause significant morbidity and mortality, suggesting the need for further understanding of streptococcal biology\[10\]. Moreover, it is known that the co-infection of streptococci and influenza B can lead to rapid onset of septic shock and death even in healthy individuals. Three cases of healthy young women with influenza B who developed a severe streptococcal infection leading to septic shock and death whose infection was sustained by *S. pyogenes* are described in the literature\[11\]. Influenza B virus, as the first action, causes damage to the epithelium of the bronchi and lungs, allowing microaspirated bacterial pathogens to establish infection. On the other hand, influenza B virus modifies the host immune response, so allowing bacterial overgrowth\[12\]. Commonly, influenza A is associated with a several co-infections, but influenza B leads to milder disease\[13\]. In a study by Okamoto et al\[14\], conducted in mice, it was demonstrated that intranasal infection with sublethal doses of influenza A virus resulted in lethal infection after further intranasal infection with sublethal doses of group A *S. pyogenes* strains.

In the young women reported with severe septic shock sustained by co-infection of influenza B and streptococci, the strain of influenza B was not the common one during that period (B/Jiangu/10/03) and so it is possible that no immunologic memory for this infection was present\[11\]. In our case, we have not matched the influenza B strain, but the lack of immunologic memory in this patient can explain the rapid development of infection in a previously healthy man.

This is the first case report of alveolar haemorrhage sustained by a co-infection of influenza B virus and *S. pyogenes* investigated by chest ultrasonography in the emergency department. Bedside chest ultrasonography allowed early depiction of lung abnormalities and recognition of their multilobar and bilateral involvement, so suggesting the severity of the picture. Moreover, FOCUS excluded substantial heart impairment, confirming intravascular volume depletion. Thus, the bedside ultrasonography findings contributed to guide the next diagnostic and therapeutic steps (e.g., CT scan and aggressive volume replacement). The presence of the typical ultrasonographic pattern, as previously observed in influenza A (H1N1)v pneumonia, suggests the feasibility of this examination for all patients complaining of respiratory distress\[6\].

B lines are the well defined rough vertical ultrasonographic artefacts arising from the visceral pleura and spreading up to the edge of the screen without fading\[6\]. They are a sign of subpleural interlobular septal thickening and are produced by repeated reflection between interfaces of tissues with a large acoustic impedance difference, such as fluid and air\[15\]. The identification of the B lines pattern in the diagnosis of interstitial syndrome is considered easy to learn, fast to depict, and highly feasible, reproducible, and reliable. Unfortunately, it is not specific, other than in cases of pneumonia\[6\] and ARDS\[8\] in situations such as cardiogenic pulmonary oedema\[16\], lung contusion\[17\], and lung fibrosis\[18\]. Subcutaneous emphysema and pneumothorax, as well as difficulty detecting central, supradiaphragmatic, retroscapular, or parahilar lung fields because of physical and anatomic obstacles, are the main limitations of this ultrasonography technique\[19\].

In conclusion, cases of streptococcal infection causing severe sepsis and fatal outcome are increasing; seasonal influenza, epidemiologically occurring from December to March in the northern hemisphere\[20\], can serve as the substratum for streptococcal co-infection. The prompt recognition of this potentially fatal picture, with the aid of bedside ultrasonography, can shorten the time to diagnosis and lead to prompt management.

**Conflict of interest**

The authors declare that they have no competing interests.

**References**