Severe respiratory illness in an intravenous drug abuser

Case report
A 36-year-old female presented with a 3-day history of increasing shortness of breath, cough with purulent sputum, haemoptysis, pleuritic chest pain, nausea, vomiting and rigors. The patient was an intravenous drug user (IVDU) who smoked 20 cigarettes per day and denied drinking alcohol. Her only past medical history was of mild asthma, for which salbutamol was taken as needed.

On examination, the patient appeared distressed and unwell. Her temperature was 39.3°C, and she was tachycardic (110 beats per minute), hypotensive (98/74 mmHg) and hypoxic (O₂ saturation 86% on air with a respiratory rate of 20 breaths per minute). There were needle marks in both groins and forearms. Heart sounds were normal with no murmurs. A chest examination showed only decreased breath sounds at both bases. Abdominal and neurological examinations were unremarkable.

Investigations
Initial investigations were as follows: white blood cells 11.2×10⁹·L⁻¹, neutrophils 9.7×10⁹·L⁻¹, haemoglobin 11.3 g·dL⁻¹, platelets 71×10⁹·L⁻¹, international normalised ratio of 1.5, creatinine 135 μmol·L⁻¹, alkaline phosphatase 195 IU·L⁻¹, alanine aminotransferase 63 IU·L⁻¹, and C-reactive protein 285 mg·L⁻¹. Arterial blood gases on air showed type 1 respiratory failure: pH 7.4, partial pressure of O₂ 7.79 kPa, partial pressure of CO₂ 4.40 kPa and bicarbonate 20. There were patchy opacities at both lung bases, which extended to the midzones on chest radiography (not shown).

The patient was initially treated for severe community-acquired pneumonia and a computed tomography (CT) scan of the chest was obtained (figure 1).
On the basis of the CT findings, the diagnosis of infected pulmonary emboli was considered and right-sided infective endocarditis (IE) was suspected. This was supported by the blood culture result, as *Staphylococcus aureus* was present. However, the transthoracic echocardiogram (TTE) showed no vegetations.

Despite treatment with appropriate high-dose intravenous antibiotics, the patient deteriorated progressively, becoming confused and agitated. She remained febrile, hypotensive and hypoxic. Approximately, 72 hours post-admission, she developed a rash (figure 2). This began distally on both legs and slowly extended to involve the torso.

The patient’s level of consciousness continued to deteriorate such that the airway could no longer be protected. Subsequently, she was transferred to the intensive care unit (ICU) where she was sedated and intubated. Inotropic support was required. While in the ICU, a transoesophageal echocardiogram (TOE) was organised (figure 3).

**Task 2**
How would you describe the patient’s rash?

**Answer 2**
The patient has an extensive petechiae/vasculitic rash in both lower limbs.

**Task 3**
Describe the abnormal finding you can see on TOE.

**Task 4**
What is the sensitivity of TTE versus TOE in detecting vegetations?
For the next 10 days, despite appropriate antibiotic and supportive therapy, the patient failed to improve. She developed spontaneous pneumothoraces and several other complications, including anaemia, profound hypoalbuminaemia (albumin 9 g·L⁻¹), massive oedema of all limbs and severe lower limb ulceration.

Improvement then began gradually over the next 7 days. She required less ventilatory support and was weaned off inotropes. However, she remained unresponsive despite cessation of all sedation; hence, a CT scan of the brain was obtained and the results are shown in figure 4.

Over the course of several weeks, the patient gradually regained consciousness, intelligent speech, and motor function on the left side. However, the right upper and lower limbs remained weak. During this recovery period, she continuously and aimlessly moved her left hand, and repeatedly put her hand in her mouth and frequently groped around her anus and external genitalia. On several occasions, her left hand spread faeces over her body. The patient insisted that she was not doing this purposefully and expressed frustration that she could not stop her hand from moving.

**Task 7**
What would your neurological diagnosis be?

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**Answer 3**
A large multi-lobulated mass/vegetation can be seen in the right ventricle.

**Answer 4**
The sensitivity of TTE compared with TOE is 40–63% versus 90–100%.

**Answer 5**
The CT scan reveals two lesions, one in the left basal ganglia area with surrounding oedema and another in the left frontal lobe. The features are consistent with multiple brain abscesses.

**Answer 6**
The possible mechanisms are as follows:
1. Concurrent involvement of both left and right ventricles.
2. Paradoxical embolism.
3. Acquired pulmonary arteriovenous malformation.
4. Metastatic as part of generalised sepsicaemia.

**Task 5**
Describe what you can see on the CT scan of the brain.

**Task 6**
Suggest possible mechanisms that could explain systemic embolisation in right-sided IE.
After 8 weeks in hospital, she was transferred to a rehabilitation facility. On discharge from this facility, she was able to communicate intelligently, mobilise without assistance and was fully independent. She had also regained reasonable control of her left hand.

Discussion
This case is a classic example of severe sepsis, which may occur as a consequence of intravenous drug abuse. It demonstrates an unusual cause of a rare but well-documented neurological syndrome. It also illustrates the remarkable recovery that is possible after severe but reversible brain injury, particularly in those younger patients with a significant degree of neurological plasticity.

Infection is a common complication amongst IVDUs. Skin and soft tissue infections occur most frequently. The commonest causes of mortality in IVDUs are accidental overdose, alcohol/other drug abuse, hepatic disease, trauma and sepsis [1].

As superficial veins become harder to access, deeper vascular structures are used with increasing risks of severe systemic sepsis. Endocarditis occurs frequently in IVDUs. It usually affects the right side of the heart, particularly the tricuspid valve and often presents with septic pulmonary emboli [2]. Septic emboli may also arise from areas of septic thrombophlebitis or dental sepsis in these patients. The microorganisms involved in the skin and local vascular infections vary, but S. aureus is the main agent, followed by various streptococci, aerobic Gram-negative rods, anaerobic cocci and bacilli [3–7].

Making the diagnosis of infected pulmonary emboli and endocarditis may be difficult. Blood cultures are not always positive and there may not be an audible murmur. Chest radiography appearances are often non-specific. On TTE, vegetations <4 mm in diameter may not be seen. Evidence of other septic emboli, such as petechiae or splenomegaly, may be useful clues. It should be remembered that IVDUs are also at an increased risk of community-acquired pneumonias, and pneumonia is, in fact, more common than endocarditis in this population [2]. However, if an appropriately treated pneumonia fails to resolve, other causes should be considered.

The chest radiograph in the presence of septic pulmonary emboli classically shows multiple ill-defined round or wedge-shaped opacities, usually towards the periphery of the lung. There is often cavitation with visible air-fluid levels. Reactive hilar lymphadenopathy and pleural collections, often representing empyema, may be present. CT scanning is more sensitive than chest radiography and may allow detection of septic emboli in situations where the chest radiograph will pass as normal. Recognised CT findings include, in descending order of prevalence: peripheral parenchymal nodules, which may contain air bronchograms; a “feeding vessel sign”; cavitation; and wedge-shaped lesions abutting the pleura [8].

Cerebral sepsis is the most serious complication of endocarditis; the organisms involved are usually pyogenic bacteria or fungi. The septic emboli in the case presented here caused AHS, in which the patient perceives one upper limb as foreign to them, having a will of its own. The syndrome was first identified in 1908 by Kurt Goldstein who described the case of a female with ischaemic lesions affecting the right hemisphere and corpus callosum. Three variants have now been described: corpus callosum damage only, causing intermanual conflict; dominant medial frontal cortex damage plus or minus corpus callosum involvement, associated with grasping and groping; and right posterior cerebral artery territory damage causing a sensory form with purposeless movements [9]. In all cases the involved limb, usually the left, is felt to be dissociated from the body and out of control. As in the case presented here, the limb is often criticised for its behaviour. Spontaneous recovery can occur and an individualised neuropsychological rehabilitation programme may be indicated as a possible therapy in AHS [10].
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CASE PRESENTATION

Teaching points

1. Endocarditis is common in IVDUs and can cause catastrophic septic embolisation.
2. Endocarditis may be difficult to clearly diagnose.
3. Antibiotics use should cover *S. aureus* in septic patients known to abuse intravenous drugs, but positive microbiology must be sought as polymicrobial and fungal infections are common.
4. Patients can make a full recovery despite overwhelming sepsis and neurological damage, and should be treated aggressively.

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