**Sudden cardiac death in a young man**

**Case Report**

**Case presentation**

A 35-year-old man presented to the accident and emergency department with history of productive cough, breathlessness and some weight loss over several weeks. He had a past medical history of asthma and eczema. He mentioned that, at times, he had been expectorating sputum with some haemoptysis over the past few months. He was of Bangladeshi origin, but had been resident in the UK since 1986 and last visited Bangladesh a year ago. He was a smoker of 10–15 cigarettes per day. He also admitted to smoking heroin. In addition to his respiratory symptoms he also complained of vomiting, which was precipitated by eating. He denied bowel or urinary symptoms. He was married, lived with his wife and three young children, denied any alcohol use and had no previous contact with tuberculosis (TB) patients.

**Investigations**

On clinical examination the patient was unwell. His temperature was 35.8°C. His blood pressure was 98/62 mmHg, heart rate 110 beats·min⁻¹, respiratory rate 16 breaths·min⁻¹. He had bilateral pedal oedema with no clinical evidence of deep vein thrombosis. There were no palpable lymph nodes. Examination of the cardiovascular system was normal.

His blood tests showed a marginally raised white cell count (WCC) of 12.5×10⁹ per L and a hyponatraemia of 133 mmol·L⁻¹. The patient had deranged liver function tests and a low glucose level of 2.1 mmol·L⁻¹ on admission, which was corrected with intravenous dextrose.

**Task 1**

What would be your initial investigations for a patient like this?

**Conflict of interest**

None declared.
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**Answer 1**
Bloods (full blood count, urea and electrolytes, and C-reactive protein), chest radiography, ECG, echocardiogram, and sputum for acid-fast bacilli

**Answer 2**
Haziness is noted on the right, cardiac shadow is enlarged, and prominent bilateral hilar vasculature

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**Task 2**
Can you name three abnormalities on the chest radiograph (fig. 1)?

**Task 3**
What would be the initial diagnosis?

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His chest radiograph showed cardiomegaly and inflammatory changes in the right middle lobe. He was started on antibiotics (amoxicillin). The plan was to exclude TB by sending sputum for tests; blood samples were also sent for hepatitis and autoimmune screening.

An ultrasound scan of his abdomen showed that the gall bladder was thick walled and had sludge with pericholecystic fluid. The liver parenchyma appeared unremarkable. It was also noted that there was hepatic venous and inferior vena cava (IVC) congestion, and bilateral pleural effusions.

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**Figure 1**
Chest radiograph.

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**Answer 3**
TB, respiratory tract infection, myocarditis

**Answer 4**
Addisonian crisis

**Outcome and follow-up**
Unfortunately the patient became agitated and suffered a cardiac arrest. He underwent cardiorespiratory resuscitation for over 45 min, but could not be resuscitated from his pulseless electrical activity arrest and unfortunately died. His post mortem showed that the lungs were markedly oedematous throughout. There was no bronchopneumonic consolidation. The pleura appeared healthy, but bilateral straw-coloured pleural effusions were present. The heart was enlarged weighing 450 g. All the valves were unremarkable. Both ventricles appeared dilated. A thrombus was adherent to the wall of the left ventricle and the ventricle muscle underlying this area of thrombus appeared pale. Sections from this abnormal area of myocardium showed florid granulomatous inflammation with multinuclear giant cells. This was consistent with TB. The lungs showed pulmonary oedema and mild emphysema, but no histological evidence of TB.

**Task 4**
What could be a possible cause of low blood glucose in the patient?

**Task 5**
What are the hallmark histological findings of patients with TB?
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Discussion

TB-related sudden death is mainly due to bronchopneumonia, massive haemoptysis and, rarely, cardiac complications of TB or adrenal gland involvement [1]. Only a few cases of sudden cardiac death due to TB myocarditis have been reported [2, 3], all of which have been diagnosed at autopsy. It has been postulated that the mechanism of death in these patients is due to a ventricular arrhythmia. Horn and Saphir [4] have described three histological types of myocardial TB. An interesting point to note is that it has been proposed that the myocardium can be affected either by direct extension, lymphatic drainage, or direct spread from tuberculous pericarditis [3]. It seems as though anatomically the right-sided mediastinal nodes make the myocardium most vulnerable to direct spread [5]. A point to note is that patients can remain clinically asymptomatic until quite late in the disease process.

The three histological types of myocardial TB are as follows:

1) Nodular tubercles (tuberculomas) of the myocardium, varying “from pea to egg size” with central caseation usually affecting the right side of the heart, particularly the right atrium.
2) Miliary tubercles of the myocardium complicating generalised miliary disease.
3) Uncommon diffuse infiltrative type, usually associated with tuberculous pericarditis, in which the myocardium is diffusely infiltrated by granulation tissue containing giant cells, endothelial cells and lymphocytes.

TB myocarditis is rarely diagnosed during life. It should be suspected in a patient with TB if patients present with a cardiac arrhythmia such as atrial fibrillation, paroxysmal ventricular tachycardia, ventricular fibrillation [6] or atrioventricular block [7]. Patients may continue to develop congestive heart failure [8], valve dysfunction or superior vena cava (SVC) obstruction [9]. Magnetic resonance imaging may be quite useful for detecting involvement of the myocardium [10, 11].

Key points

- TB-related sudden deaths are mainly due to bronchopneumonia, massive haemoptysis, Addisonian crisis, and arrhythmias secondary to myocarditis.
- TB myocarditis is an uncommon presentation in the acute setting.
- Myocarditis is a known complication and should be considered.
- These patients may present with cardiac arrhythmia which if not addressed may lead to cardiac arrest.
- TB treatment and high-dose steroids remain the mainstay of therapy.

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