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Recent fever, sudden dyspnoea and ST elevation with raised cardiac enzymes

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DESCRIPTION

A 45-year-old male farmer presented with a history of fever since 1 week associated with dyspnoea and dry cough since 5 days, followed by bodyache, jaundice and redness in both eyes since 2 days. There was no history of similar complaints, or any history of hypertension, diabetes, tuberculosis, etc. For his current symptoms he consulted a local health worker but was referred to us for further management when he did not improve.

On admission, the patient was conscious and oriented. He was febrile with a temperature of 100 F, pulse 102/min and blood pressure of 116/86 mm Hg. He was tachypnoeic with a respiratory rate of 26/min and hypoxic with an oxygen saturation of 80% at room air.

He had conjunctival suffusion and there was no pallor, cyanosis, lymphadenopathy, pedal oedema or raised jugular venous pressure. On cardiac auscultation his heart sounds were normal. There was no cardiac murmur. Respiratory system examination revealed presence of bilateral wheeze.

Laboratory investigations revealed haemoglobin of 14 g%, a total leucocyte count of 4400/mm³ and thrombocytopaenia (platelets were 27 000/mm³). He had marked azotaemia with a urea of 147 mg% and creatinine 6.5 mg%. His urine routine and microscopy showed 30–40 pus cells with active sediment evidenced by the presence of 5–6 red blood cells along with moderate macroalbuminuria suggesting an active glomerular injury. He did not have any documented oliguria.

He had marked jaundice with a total bilirubin of 25 U/l (direct bilirubin 14.0, indirect bilirubin 11.0) along with mild hepatitis evidenced by mild elevation of liver enzymes (aspartate aminotransferase /alanine transaminase/alkaline phosphatase (ALP) 123/79/136). He had marked hypoalbuminemia (total protein/albumin 4.9/2.5). His viral markers (hepatitis B surface antigen and anti-Hepatitis C virus) and peripheral smear for malaria parasite were negative. His glycemic status was normal with fasting blood sugar/post prandial blood sugar of 62/103.

His arterial blood gas showed metabolic acidosis with compensatory respiratory alkalosis pH 7.40, PO₂ 72.1, PCO₂ 24.6, HCO₃ 15.4. His electrolytes were fairly normal at Na⁺/K⁺ 129/4.0.

Chest x-ray showed bilateral prominent pulmonary vasculature but no obvious pulmonary oedema (figure 1). His ECG revealed an ST elevation of 3 mm in V2 and V4 (figure 2) which subsequently subsided on ECG 2 days later (figure 3). Cardiac echocardiography showed dyskinetic

basal inferior septum with normal left ventricular function. His creatine phosphokinase (CPKMB) cardiac enzyme was elevated at 34 U/l (normal 24 U/l). He was treated as a non-Q/non-ST elevation myocardial infarction (STEMI)/subendocardial myocardial infarction with addition of anticoagulants (low molecular heparin), antiplatelets (aspirin) and statins (atorvastatin).

In view of his hypoxia, thrombocytopaenia and azotaemia, the patient was shifted to the ICU where he received 4 units of platelet transfusions along with antibiotics and two sessions of haemodialysis. Three days later his platelet counts improved to 93 000/mm³ along with a drastic reduction in azotaemia to normal. He was discharged after a week after all his disease parameters normalised.

DISCUSSION

This patient had a classical multisystem involvement with respiratory, hepatic, renal and cardiac illness and a

Figure 1 Admission day chest x-ray postero-anterior (PA) view.

Figure 2 First ECG on admission.

Figure 3 ECG after 2 days.

clinical diagnosis of leptospirosis was made. This was supported serologically by high immunoglobulin M leptospira antibody titers of 80 U/ml (normal range <15U/ml). His findings of ST elevation and elevated cardiac enzymes were initially thought to be due to a myocardial infarction and he was managed accordingly although thrombolytics were not given as he had presented after the window period. In retrospect, however, this constellation of cardiac findings could have been due to leptospira myocarditis that have been described in significant numbers in autopsy series of patients with suspected leptospirosis.^{1 2} Acute myocardial infarction like clinical presentation of myocarditis has been reported in prior case series.³ This report was to document a patient of leptospira myocarditis mimicking a myocardial infarction.

Competing interests None.

Patient consent Obtained.

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