CASE REPORT

Therapeutic dilemma in a case of acute coronary syndrome (ACS)

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SUMMARY

An adult male labourer, a smoker and alcoholic was admitted to our hospital with a short history of fever, myalgia, breathlessness and oliguria. On examination he was icteric and hypotensive. Calf muscle tenderness was present. A provisional diagnosis of leptospirosis was made and he was started on treatment with crystalline penicillin. Blood pressure (BP) did not improve with fluids. Inotropes were started. The patient was taken for Slow Low Efficiency Daily Dialysis (SLEDD) during which he developed chest pain. ECG showed an anterolateral myocardial infarction (MI). He also complained of breathlessness and haemoptysis. Antiplatelets were withheld in view of thrombocytopenia and haemoptysis; heparin could not be given because of the deranged coagulation parameters. The patient was managed symptomatically with nitrates. After the BP improved SLEDD was restarted. On day 3 of admission the patient became tachypnoeic and hypoxic, bilateral coarse crackles were present on auscultation. He was intubated and mechanically ventilated. Suctioning of endotracheal tube revealed fresh blood, and chest CT revealed alveolar haemorrhage. In spite of aggressive resuscitative measures, mechanical ventilation and antibiotics, the patient expired on the 12th day following admission.

BACKGROUND

We present this case to illustrate that in a patient with risk factors, acute coronary syndrome (ACS) can complicate even infectious diseases. The management of such cases with multiple complications of both the infectious process and ACS is difficult.

CASE PRESENTATION

The patient, a 48-year-old manual labourer presented to us with a 4-day history of high fever with chills and rigours, myalgia and cough productive of minimal amounts of mucoid sputum. He also complained of gradually progressive breathlessness for 2 days preceding admission. Medical history and family history were not contributory.

On examination the patient was febrile and icteric. He had a pulse rate of 100/min. The systolic blood pressure was 80 mm Hg by palpation.

Bilateral calf muscle tenderness (which is indicative of leptospirosis) was present. Auscultation of the lungs revealed bilateral fine crepitations. The patient was conscious but restless. There was no focal neurological deficit or neck stiffness.

INVESTIGATIONS

- ▶ Blood urea 160.0 mg/dl (10–50 mg%)
- ▶ Serum creatine 6.2 mg/dl (0.7–1.2 mg%)

- ▶ Serum total bilirubin 6.9 mg/dl (up to 1.2 mg%)
- ▶ Serum aspartate aminotransferase (serum glutamic oxaloacetic transaminase) 53.0 U/l (up to 35 U/l)
- ▶ Serum alanine aminotransferase (serum glutamic pyruvic transaminase) 17.0 U/l (up to 45 U/l)
- ▶ Serum alkaline phosphatase 101.0 U/l (40–130 U/l)
- ▶ PT—control—13.5 s Test—27 s
- ▶ Urine dark ground microscopy—positive for leptospirosis
- ▶ Haemoglobin 11.0 g%
- ▶ White blood cell count (total) 8000/mm³
- ▶ Differential leukocyte count—N95%L2% E02%M01%
- ▶ Erythrocyte sedimentation rate 65 mm (1st hour) (0–10 mm/h)
- ▶ Platelet count 7000/mm³
- ▶ Blood culture—no growth
- ▶ Echocardiogram—lateral wall hypokinesia. Ejection fraction—50%. No left ventricular hypertrophy

TREATMENT

With a provisional diagnosis of leptospirosis the patient was started on crystalline penicillin at 15, 00, 000 units sixth hourly after negative test dose. The blood pressure did not improve even after a fluid challenge of 200 ml bolus three times, and norepinephrine was infused at 10 µg/min, after which the blood pressure improved to 100/60 mm Hg. However the oliguria persisted. Renal parameters worsened and the patient was taken for SLEDD (Slow Low Efficiency Daily Dialysis). During dialysis the patient complained of severe substernal chest pain and breathlessness. The patient was tachypnoeic and diaphoretic and systolic blood pressure was 80 mm Hg. An ECG was recorded and it showed ST elevation in leads I, aVL and V4–V6 and ST depression in lead V1. Dialysis was terminated and the patient was shifted back to the medical intensive care unit. Chest pain was relieved with morphine, however, the cough increased and was now associated with haemoptysis. Aspirin and clopidogrel were withheld in view of haemoptysis and thrombocytopenia; heparin was not given because of elevated PT/INR and the patient refused consent for thrombolysis and primary angioplasty. Chest x-ray was suggestive of acute respiratory distress syndrome. The patient became tachypnoeic and hypoxic and he was intubated and mechanically ventilated. Pulsed methylprednisolone was administered for pulmonary haemorrhage.

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Figure 1 ECG showing anterolateral myocardial infarction.

Platelets and fresh frozen plasma were transfused, and antibiotics and inotropes were continued. On day 12 the patient suffered a cardiac arrest from which he could not be revived, and was declared dead.

DISCUSSION

Leptospirosis is a zoonosis caused by spirochetes of the genus *Leptospira*. Severe leptospirosis is characterised by hepatic and renal dysfunction, hypotension and pulmonary haemorrhage.

Haemorrhagic manifestations in leptospirosis can be due to endothelial disruption leading to increased vascular permeability. Thrombocytopenia and impaired coagulation parameters due to hepatic injury may also contribute to haemorrhagic tendency. Thrombocytopenia is common in leptospirosis¹ and may be due to immune-mediated platelet destruction and increased peripheral consumption.^{2,3}

Cardiovascular involvement in leptospirosis includes arrhythmias and pericarditis.⁴ Though ECG changes including atrial fibrillation, atrioventricular block and non specific repolarisation abnormalities are common,⁵ left ventricular function as assessed by echocardiogram is usually normal.⁶ Autopsy reveals that cardiac involvement in leptospirosis includes cardiomegaly and interstitial myocarditis.⁷ However, MI has been rarely reported.

The immediate precipitating event causing MI in our patient may have been due to several factors. Hypotension can precipitate myocardial injury (indicated by increased cardiac troponin T) both in patients of hypovolemic shock as well as those with septic shock.⁸ Our patient had prolonged hypotension, which probably contributed to the myocardial injury. Infection itself may predispose to a prothrombotic state and MI by interacting with the endothelium to produce changes in the functioning and regulation of coagulation and fibrinolysis.⁹

We faced several difficult therapeutic decisions at various points during this patient's admission. The first point was when he developed an acute MI during SLEDD. This was probably a result of coronary hypoperfusion due to hypotension in a patient with

risk factors for coronary artery disease. The hypercatabolic state and increased cardiac work due to the underlying infective process may have contributed to cardiac ischaemia. The first decision that we had to make was whether to administer antiplatelet agents in a patient with acute MI with thrombocytopenia. Thrombocytopenia can occur as a complication in patients admitted for ACS. In this setting, even mild thrombocytopenia may be associated with increased risk of haemorrhage and with increased mortality.¹⁰ Case reports have been published where patients admitted with haematological cancers and thrombocytopenia developed acute myocardial infarction (MI) during the course of treatment, and have undergone percutaneous coronary intervention (figure 1).^{11,12} A case series has been published where 30 cases of preexisting thrombocytopenia who developed ACS underwent PCI as per guidelines, and none had any complications of major bleeding. However, this study excluded patients with active bleeding and thrombocytopenia due to sepsis.¹³ Patients with preexisting cancer and thrombocytopenia who develop ACS have been treated with aspirin and there were no complications of severe bleeding.¹⁴ Published data regarding the management of ACS in a patient with thrombocytopenia due to a preexisting infectious disease could not be found. In view of the active haemoptysis and the fact that sepsis was the underlying cause of thrombocytopenia we decided to withhold aspirin. Further management options included thrombolysis and percutaneous coronary intervention. Thrombolysis would have been risky in view of hypotension and active bleeding and the patient refused PCI due to financial constraints. Anticoagulants were contraindicated. So, in effect, our hands were tied, and we could only give our patient pain relief and support him haemodynamically. Pulmonary haemorrhage in patients with leptospirosis is a complication associated with increased mortality.¹⁵ Methylprednisolone has been tried in several case series, with some encouraging results.¹⁶

We present this case as an unusual cardiac complication of leptospirosis and also to illustrate the therapeutic dilemma we faced while treating the patient.

Learning points

- Infectious diseases like leptospirosis can be complicated by acute coronary syndrome.
- Appropriate treatment requires the clinician to walk on a thin line navigating between the interconnected complications and treatment of each condition.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned, externally peer reviewed.

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