Lemierre’s syndrome: cavitary lung disease caused by uncommon bacteria

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DESCRIPTION
A 32-year-old Asian man with a history of gastritis presented to the emergency room with abdominal pain, fever, cough and dyspnoea, one episode of haemoptysis. He also reported a sore throat 3 days ago. The patient was a sushi chef and an active smoker. Early lab work was remarkable for leucocytosis of 21 x 10^9/L, lactic acidosis of 3 mmol/L, bandaemia of 19% with a procalcitonin of >100 and platelets of 74 x 10^9/L. CT of the lungs was performed without contrast and was significant for multiple cavity lesions throughout the lungs (figures 1 and 2). The patient was intubated due to respiratory failure. He was empirically started on vancomycin, cefepime and azithromycin. A right internal jugular central line was attempted for triple lumen catheter placement but the vessel was not compressible. A CT scan of the neck revealed a right internal jugular vein 9 mm thrombosis (figure 3). Blood culture grew Fusobacterium necrophorum. The antibiotics were switched to ampicillin-sulbactam. Bronchoscopy and bronchoalveolar lavage returned negative for fungal infection, Legionella, Pneumocystis jiroveci and acid fast bacilli as well as negative for HIV, urine Streptococcus and Legionella test and the nasal influenza. A transthoracic echo did not reveal vegetation to suggest endocarditis. The patient did receive short course of anticoagulation, but as repeat CT neck was not showing any further extension of clot, anticoagulation was discontinued. Eventually, the patient improved and discharged home on amoxicillin-clavulanic acid for 2 months. Lemierre’s syndrome is defined as suppurative thrombophlebitis of the internal jugular vein often preceding an upper respiratory infection such as pharyngitis. The bacterial culprit is primarily F. necrophorum, affecting young healthy individuals.1 The causative bacteria can penetrate, either through the lymphatic system or along the fascial planes, into the adjacent blood vessels causing thrombosis and subsequent thrombophlebitis of the internal jugular vein, often forming parapharyngeal inflammation and a peritonsillar abscess along the way. The presence of haemagglutinin promotes the fulminant nature of the disease by augmenting platelet aggregation and septic thrombus formation.2 The propagation of the septic emboli throughout the body causes a cascade
of disastrous effects, most commonly lodging into the lungs and creating cavitary lesions. Considering the overall severity of the septicemic illness, acute respiratory distress syndrome occurs in a relatively small proportion of cases. The location of the primary infection is an important prognostic factor: infection in the oropharyngeal location (compared with other sites) was associated with a higher risk of longer intensive care unit stays due to complications such as respiratory problems. Studies from the modern era have reported mortality rates from 0% to 18%. Disseminated intravascular coagulation has been reported in 3%–9% of cases. Mild elevation of blood urea nitrogen and creatinine with haematuria is not uncommon but acute renal failure is rare and the possible aetiology includes either septic emboli to kidneys or acute septic tubular necrosis. Meningitis may also complicate up to 3% of cases. Blood cultures, chest X-ray and CT chest should be enough to provide a diagnosis. Antibiotics should be started as early as possible. The role of anticoagulation is still controversial.3

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