From a sore throat to the intensive care unit: the Lemierre syndrome

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Summary. Lemierre syndrome is characterized by an acute oropharyngeal infection, suppurative thrombophlebitis of the internal jugular vein and metastatic infections. The infection is usually caused by Fusobacterium necrophorum. We report on a 19-year-old male patient who was admitted with a five-day history of fever, sore throat and progressive dyspnea. Computed tomography of the neck and chest revealed a parapharyngeal abscess, jugular vein thrombosis, descending necrotizing mediastinitis and multiple areas of bilateral consolidation and cavitations within the lungs. Fusobacterium necrophorum was identified in the blood culture. Early combined abscess drainage with neck and chest incisions, together with broad spectrum intravenous antibiotic treatment and medical management in an intensive care unit resulted in a good clinical outcome.

Key words: Lemierre syndrome, descending necrotizing mediastinitis, fusobacterial sepsis.

Introduction

Symptoms of pharyngitis, such as sore throat, are frequently reported complaints of patients seeking medical care [1]. Treatment of pharyngitis will vary according to the cause: symptomatic therapy is usually sufficient to treat viral infections but pharyngitis in association with bacteria should be treated with appropriate antibiotic therapy. If untreated, pharyngitis may progress to peritonsillar or parapharyngeal abscess formation. Because of regional anatomy, the infection could extend from the parapharyngeal to the retropharyngeal space, carotid sheath and mediastinum [2]. Although the incidence of soft-tissue abscesses of the neck has markedly declined with the widespread use of antibiotics, peritonsillar and/or parapharyngeal abscesses are feared complications. This is of particular importance as recent reports suggest that anthroposophic treatment of primary care patients with sore throat had a better outcome than conventional treatment including antibiotic therapy [3].

We report on the case of a patient who was treated symptomatically for a sore throat and who developed septic multorgan failure in association with a peritonsillar abscess complicated by jugular vein thrombophlebitis. Clinical, microbiological and radiographic findings suggested that the patient was suffering from Lemierre syndrome.

Case report

A 19-year-old previously healthy young man was admitted for progressive shortness of breath, dysphagia, bilateral chest pain and shaking chills. He had an unremarkable past medical history and a smoking history of 20 cigarettes/day. Five days before admission he sought medical attention for a sore throat, where he was diagnosed with acute pharyngitis. He was advised to rest, increase liquids and take non-steroidal anti-inflammatory drugs as required; however, the treatment was without response.
Physical examination on admission disclosed an acutely ill young man with a body mass index of 17.9 kg/m². The patient appeared pale, exsiccated and fatigued. His temperature was 38.5°C, blood pressure 80/50 mmHg and pulse rate 120 beats/min. His breathing rate was markedly increased at 60 breaths/min and he was using his accessory respiratory muscles. Inspection of the mouth revealed pustular exudates on both tonsils and pharyngitis. The right side of the patient’s neck along the anterior border of the sternocleidomastoid muscle was tender but not swollen. There was right-sided submandibular and cervical lymphadenopathy and bilateral rales on chest auscultation. The findings on cardiovascular examination were normal except for tachycardia. Notable laboratory test results were as follows: hemoglobin 14.3 g/dl, platelet count 35,000 cells/µl, serum urea nitrogen 69.0 mg/dl, serum creatinine 1.8 mg/dl and serum cholinesterase 1033 U/l. The blood lactate level was elevated at 6.2 mmol/l. Arterial blood gas analysis showed type I respiratory failure with a PaO₂ of 52 mmHg, PaCO₂ of 30 mmHg, pH 7.418 and an arterial oxygen saturation of 87% SaO₂ on room air. The HIV status was negative.

Chest x-ray showed bilateral pleural effusions and several ill-defined intrapulmonary opacities on both sides. Ultrasound examination of the neck revealed a large right-sided parapharyngeal soft-tissue mass. Contrast enhanced computed tomography (CT) of the neck confirmed hypodensities in the right parapharyngeal space, indicative of a parapharyngeal abscess (Fig. 1). Furthermore there was evidence of thrombosis of the right internal jugular vein on CT. The contrast CT scan of the thorax revealed massive, bilateral multiloculated pleural effusions with consecutive compression atelectases, a large air-space consolidation of the right lower lobe with central necrosis, and a necrotizing soft-tissue mass in the anterior mediastinum indicating a mediastinal abscess (Fig. 2).

The patient had severe respiratory distress and hemodynamic instability and was immediately transferred to the intensive care unit. A trial of non-invasive pressure support ventilation was unsuccessful and he was intubated and mechanically ventilated. His hemodynamic status required aggressive rehydration and catecholamine substitution. After the respiratory and hemodynamic status was stabilized he was urgently taken to the operating room. The parapharyngeal abscess was drained and bilateral tonsillectomy performed. In order to drain the pleural and mediastinal abscess formations, bilateral thoracotomy was performed with the removal of 600 ml of pus on the right side and 1800 ml of pus on the left, followed by bilateral decortication. Bilateral pleural drains were placed in addition to a drain in the anterior mediastinum.

The patient was postoperatively transferred to the intensive care unit. Blood, urinary and sputum cultures were collected before starting antibiotic therapy. Empirical intravenous therapy was started with metronidazole (1.5 g/day), fosfomycin (16 g/day) and piperacillin/tazobactam (18 g/day). Anticoagulation was commenced with low-dose subcutaneous low-molecular-weight heparin. There was no evidence for acid-fast bacilli, fungi or bacteria in pleural aspirates obtained intraoperatively. Blood cultures (BacT Alert 3D, BioMerieux, France) obtained on admission were positive for anaerobic Gram-negative bacilli. Further analysis with the Rapid ID 32 A system (BioMerieux, France) identified the strain as Fusobacterium necrophorum. On the basis of these findings together with the clinical and radiological results, a diagnosis of Lemierre syndrome was made.

Minimal inhibitory concentrations determined with the E-Test (Biodisk, Sweden) were less than 1 for all tested antibiotics (0.064 µg/ml for penicillin, 0.047 µg/ml for ampicillin/BLI, 0.064 µg/ml for imipenem, 0.032 µg/ml for clindamycin, 0.094 µg/ml for cefoxitin and 0.064 µg/ml for metronidazole).

The patient remained in a critical condition postoperatively with laboratory and clinical signs of sepsis and multiorgan failure for about two weeks despite high-dose antibiotic therapy. On day 12 serological inflammation parameters increased again and chest x-ray showed new infiltrates on both sides, indicating ventilator-associated pneumonia. In addition, a multiresistant coagulase-negative staphylococcus was identified from samples of postoperatively obtained pleural fluid. The antibiotic regimen was changed to clindamycin (3.6 g/day), levofloxacin (1 g/day) and vancomycin (2 g/day), which were given for a total of 21 days. The patient recovered slowly and was weaned off mechanical ventilation after 20 days. He was discharged from the intensive care unit on day 35. He continued supportive medical therapy on the normal ward and was discharged from the hospital on day 63. Follow-up visits at our outpatient clinic showed persisting recovery.

Discussion

Courmont and Cade were the first to describe postanginal sepsis in association with an anaerobic infection in 1900 [4]. It was in 1936, however, when André Lemierre, a Parisian bacteriologist, published a case series on 20 young adults who suffered from sepsis in association with anaerobic organisms following infection of the tonsils [5]. Lemierre identified that most of these patients had a peritonsillar abscess, jugular venous thrombophlebitis, and metastatic abscess formations with a predilection to the lung. Only two of the 20 patients survived. Although the widespread use of antibiotics in the management of acute oropharyngeal infections has led to a rapid decline in the incidence of Lemierre syndrome, mortality remains high in those cases where appropriate antimicrobial therapy is not initiated promptly [6].

Lemierre syndrome is usually caused by F. necrophorum, a strictly anaerobic, nonmotile, non-spore-forming Gram-negative rod. The bacterium is a normal inhabitant...