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From sore throat to sepsis: unmasking life-threatening Lemierre syndrome

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SUMMARY

Lemierre syndrome is a serious condition that typically affects young, otherwise healthy individuals. It typically begins with a bacterial oropharyngeal infection that progresses to septic thrombophlebitis of the internal jugular vein, frequently accompanied by septic emboli. We present a case involving a previously healthy young male who developed Lemierre syndrome following a sore throat and fever, later complicated by sepsis. Imaging confirmed internal jugular vein thrombosis, and blood cultures grew *Fusobacterium necrophorum*. Further investigation revealed underlying thrombophilia, which likely contributed to the severity of his thrombotic complications. The patient was successfully treated with broad-spectrum antibiotics and anticoagulation. Although incidence has declined with widespread antibiotic use, this case emphasises the importance of early recognition and prompt treatment to reduce the risk of severe complications and mortality.

BACKGROUND

Lemierre syndrome is a rare (1/1 000 000) but potentially life-threatening condition characterised by oropharyngeal infection leading to septic thrombosis of the internal jugular vein. Most commonly caused by *Fusobacterium necrophorum*, an anaerobic gram-negative bacillus, the syndrome progresses rapidly from localised infection to systemic sepsis. It is often overlooked due to its non-specific clinical presentation in the early stages, leading to frequent delays in diagnosis and treatment.

CASE PRESENTATION

A male patient in his early 20s was hospitalised with a 3-day history of fever and sore throat.

His medical history was unremarkable with no chronic illnesses, neurological symptoms or systemic complaints in the months preceding this episode. Family history was also unremarkable, with no known genetic, autoimmune or vascular disorders associated with coagulation abnormalities. The patient had no prior dental issues. He engages in physically demanding work and does not smoke.

INVESTIGATIONS

Laboratory evaluation revealed markedly elevated inflammatory markers: C-reactive protein 567 mg/L, procalcitonin 82 ng/mL and leucocytosis of 16.27×10^9/L. Due to the presence of meningeal signs, a lumbar puncture was performed. Cerebrospinal fluid analysis demonstrated neutrophilic pleocytosis, elevated protein and increased lactate levels (table 1).

On admission, the patient was in a severe, agitated state. Neurological examination revealed positive meningeal signs. Cranial nerve function was intact. There was no evidence of motor deficits in the upper or lower extremities; muscle strength was 5/5 bilaterally according to the Medical Research Council score. Tendon reflexes were symmetrical and graded 2+. Pathological reflexes were absent. Sensory testing, including touch, temperature and pinprick, was normal in all extremities. Notably, tenderness was elicited over the C2–C7 spinous processes. Coordination tests, including finger-to-nose and heel-to-shin, were normal.

A CT scan of the chest revealed pulmonary embolism in the right lower lobe, bilateral pulmonary infiltrates and thrombosis of the left internal jugular vein. Cervical spine MRI showed spinal epiduritis. Despite ongoing treatment, the patient's clinical condition deteriorated and, on day 4, he was intubated for sepsis-related acute respiratory distress syndrome. Blood cultures were positive for *F. necrophorum*. The combination of positive blood culture, imaging findings and clinical presentation confirmed the diagnosis of Lemierre syndrome. Hypercoagulation with secondary hyperfibrinolysis was discovered based on rotational thromboelastometry results.

TREATMENT

Therapeutic-dose enoxaparin was initiated; however, therapeutic anticoagulation was not achieved (anti-factor Xa (aFXa) level: 0.42 IU/mL). Follow-up CT imaging revealed progression of thrombosis to the left brachiocephalic vein, left vertebral vein and left sigmoid sinus. Consequently, continuous intravenous unfractionated heparin infusion was started and titrated to 2300 IU/hour to achieve the target activated partial thromboplastin time (aPTT). Despite increased dosing, D-dimer levels continued to increase, and achieving the target aPTT was difficult despite escalating doses of heparin, suggesting resistance to heparin. Antithrombin III levels were within the normal range, while protein C and protein S levels were found to be low. Heparin therapy was subsequently replaced with fondaparinux 7.5 mg once a day, which led to the achievement of therapeutic anticoagulation (aFXa levels: 1.15–1.2 IU/mL). Antiplatelet therapy with 100 mg of acetylsalicylic acid once a day and 75 mg of clopidogrel once a day was also introduced. Follow-up CT imaging demonstrated

Table 1 Analysis of cerebrospinal fluid

Parameter	Result
Total leucocyte count	0.279×10^9/L
Red blood cells (RBC)	0×10^{12}/L
Glucose	3.52 mmol/L
Protein	0.96 g/L
Lactate	4.41 mmol/L
Albumin	561 mg/L
Colour	Clear

partial resolution of the thrombosis. Fondaparinux was later transitioned to rivaroxaban 20 mg once a day. The patient was eventually discharged in stable condition, without any residual neurological deficits.

OUTCOME AND FOLLOW-UP

Follow-up MRI of the cervical spine performed 1 month after initiation of treatment demonstrated positive progression, with complete resolution of epiduritis and the epidural abscess. On discharge, the patient was prescribed aspirin 100 mg once a day and rivaroxaban 15 mg once a day for a duration of 3 months. Thereafter, rivaroxaban monotherapy at the same dose was continued for an additional 3 months.

DISCUSSION

Lemierre syndrome is a rare but serious condition, often associated with severe complications. It is characterised by septic thrombophlebitis of the internal jugular vein, typically following an oropharyngeal infection, and is frequently accompanied by septicaemia and metastatic septic emboli. In approximately 86% of cases, the causative pathogen is *F. necrophorum*, a Gram-negative anaerobic bacillus.[1]

The lungs represent the most common site of septic embolisation in Lemierre syndrome, making the combination of timely drainage of purulent collections and appropriate antimicrobial therapy critical for improving clinical outcomes.[1] Empirical treatment with broad-spectrum antibiotics that include beta-lactamase inhibitors is recommended.[2] Metronidazole is frequently included in treatment regimens due to its excellent anaerobic coverage and its ability to penetrate the blood−brain barrier, making it effective in cases with central nervous system involvement.[3] The duration of antimicrobial therapy generally ranges from 4 to 6 weeks, depending on disease severity and clinical response.

In addition to antimicrobial therapy, anticoagulation may play an important role in the management of Lemierre syndrome. Patients with Lemierre syndrome have an increased risk of thromboembolic complications and mortality.[4] Anticoagulants are commonly used to prevent further propagation of thrombus; however, the decision to initiate anticoagulation must be carefully weighed against the bleeding risk, extent of thrombosis and overall clinical status of the patient.[5]

There are currently no established coagulation management guidelines for Lemierre syndrome. A comprehensive systematic review published in 2020 identified 216 articles addressing anticoagulation strategies in Lemierre syndrome. Notably, in most cases, thrombus resolution occurred regardless of the choice of anticoagulant.[6] The authors suggested that established The American College of Chest Physicians (CHEST) guidelines for venous thromboembolism (VTE) could theoretically be applied to patients with Lemierre syndrome.[6]

Given the absence of disease-specific guidelines, anticoagulation management in this case was guided by general VTE and critical care anticoagulation principles. Initial treatment with low-molecular-weight heparin (LMWH) was selected in accordance with standard VTE management. Previous studies have demonstrated that monitoring aFXa activity during LMWH therapy allows for individualised dose adjustment, thereby optimising anticoagulant efficacy while minimising bleeding risk and lowering risk of subsequent thrombotic events compared with delayed or unmodified dosing.[7 8] Applying this strategy in our patient enabled careful titration of LMWH to achieve effective anticoagulation without increasing haemorrhagic complications. The suggested therapeutic aFXa levels for LMWH are 0.5–1.2 U/mL for two times a day enoxaparin and 1–2 U/mL for once a day enoxaparin.[9] In our case, due to subtherapeutic aFXa activity and progression of thrombosis, the LMWH dose was escalated with serial monitoring, reflecting evidence that critically ill patients often require individualised dosing because of altered pharmacokinetics.[10] Persistent failure to achieve therapeutic anticoagulation prompted conversion to unfractionated heparin, allowing continuous infusion and closer titration. Intravenous unfractionated heparin treatment is typically monitored by the aPTT, with a therapeutic target defined as the range that corresponds to an aFXa level of 0.3–0.7 U/mL. Some studies found aFXa assays better for achieving laboratory end points, but data regarding relevant clinical outcomes are more limited.[11–13] Despite a normal level of antithrombin III, the clinical patient exhibited resistance to heparin therapy due to persistent coagulopathy which can be partially related to a decreased level of protein C and protein S. The anticoagulation regimen was therefore transitioned to fondaparinux, a synthetic factor Xa inhibitor which was selected following the development of heparin resistance, as it provides predictable factor Xa inhibition and has antithrombin-independent activity.[14] Periodic monitoring of aFXa subsequently allowed one to achieve a therapeutic concentration of fondaparinux in patient plasma. The addition of antiplatelet agents (aspirin and clopidogrel) was considered in the context of extensive thrombotic burden and laboratory evidence of thrombocytosis.

Due to the limited number of reported Lemierre syndrome cases, consensus on the optimal duration and type of anticoagulation therapy remains lacking. Nevertheless, anticoagulation for a duration of 6–12 weeks is commonly considered reasonable and effective, particularly in patients with extensive thrombosis or ongoing thrombotic risk.

Learning points

- Despite being rare, Lemierre syndrome should be considered in young patients presenting with oropharyngeal infection, septicaemia and signs of internal jugular vein thrombosis, to avoid delays in life-saving treatment.
- Early targeted antibiotic therapy remains the cornerstone of the management of Lemierre syndrome and is vital to reducing mortality and progression to multi-organ dysfunction.
- The syndrome can present with fulminant sepsis and metastatic septic emboli; timely intensive care support and source control are essential for survival.
- In patients with underlying thrombophilic disorders, managing Lemierre syndrome becomes more complex. Anticoagulation must be individualised, balancing the risks of bleeding against thrombotic progression.
- Effective management of Lemierre syndrome requires close collaboration between infectious disease specialists, haematologists, intensivists and radiologists to guide timely diagnostics, antibiotic strategy, anticoagulation decisions and supportive care.

This case highlights the complexity of managing Lemierre syndrome, particularly in the setting of critical illness, acquired coagulopathy and resistance to standard anticoagulation strategies. It underscores the importance of individualised, multidisciplinary management addressing both the infectious and thrombotic components of this rare disease.

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Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

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