Case Report

# Lemierre's syndrome presenting with septic shock

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**Abstract**

Lemierre's syndrome is a rare condition characterized by septic thrombophlebitis of the internal jugular vein and metastatic abscesses following oropharyngeal infection. Though classically caused by *Fusobacterium necrophorum*, a number of other causative organisms have been reported in literature. We report a case of Lemierre's syndrome following parapharyngeal abscess due to *staphylococcus aureus* which progressed to septic shock.

**Keywords:** Internal jugular vein thrombosis, Lemierre's syndrome, oropharyngeal infection

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## Introduction

Lemierre's syndrome (necrobacillosis or postanginal septicaemia) was first reported in 1900 by Courmont and Cade[1] and later described by Andre Lemierre in 1936 in a review of 20 cases.[2] It is characterized by oropharyngeal infection, thrombophlebitis of the internal jugular vein (IJV) and metastatic abscesses. It is classically due to anaerobic infection and *Fusobacterium necrophorum* is the most common pathogen.[3] In the pre-antibiotic era this syndrome was common and usually fatal. With the advent and widespread use of antibiotics this is rarely reported and became the "forgotten disease."[4] We report an unusual variant of the syndrome wherein the primary source of oropharyngeal infection was *staphylococcus aureus* as the causative organism.

## Case Report

A 24-year-old young male weighing 80 Kg, with no co-morbidities presented with history of sore throat of 10 days duration, associated with difficulty in swallowing. 4 days before presenting to the hospital, he developed pain and swelling on the left side of the neck and had a 2 day history of severe throbbing headache with vomiting and restlessness. Examination of his oral cavity showed a bulge in the posterior pharyngeal wall, more towards left side and the uvula was deviated to the right. He was febrile, tachycardic, and had tender swelling on the left side of the neck involving the angle of mandible and upper third of the sternomastoid. Further systemic examination was unremarkable and his Hb 13.3 gm%, total count of 9800 cells/cumm, platelet count 282,210 cells/cumm, ESR 37 mm/h and was HIV negative. Magnetic resonance imaging of neck revealed a left parapharyngeal abscess and magnetic resonance (MR) venogram [Figure 1] showed thrombosis of the left IJV and reduced flow or stasis in left transverse and sigmoid sinuses. Other investigations were unremarkable. A diagnosis of Lemierre's syndrome was made. The parapharyngeal abscess was drained under general anesthesia and was empirically started on imepenem awaiting culture reports of aspirated pus and devitalized tissue. Fondaparinux was started in view of thrombosis of left IJV.

On the 2nd post-operative day, patient had persistent hypotension with systolic pressures between 70 and 90 mmHg, not responding to repeated fluid boluses. The total count decreased to 3720 cell/cu mm, platelet count dropped to 35,000/cu mm, Activated partial thromboplastin time was prolonged (84 s), and serum procalcitonin 1.4 ng/mL. A diagnosis of septic shock

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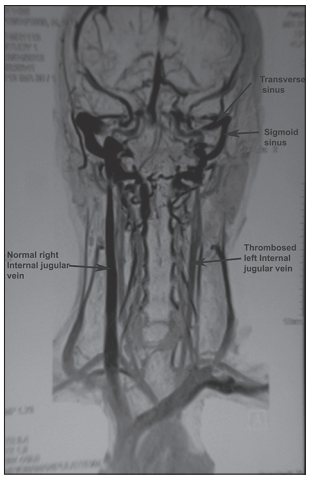


**Figure 1:** MR venogram demonstrating thrombosed left internal jugular vein and reduced flow or stasis in left transverse and sigmoid sinuses

was entertained and patient was shifted to intensive care unit. Imipenem was stopped and antibiotics vancomycin & clindamycin were started for Gram-positive coverage and metronidazole for anaerobic coverage as suggested by clinical microbiologist. Single donor platelet units were transfused for thrombocytopenia.

Central venous access was obtained via the right femoral vein. Neck veins and subclavian vein cannulation was not attempted in view of thrombosis of the left IJV. Fondaparinux was stopped in view of deranged coagulation and dropping platelet count. Inotrope in the form of noradrenaline infusion was started to maintain MAP > 65 mmHg.

Cultures of pus and devitalized tissue collected during surgery grew *S. aureus* sensitive to antibiotics vancomycin and clindamycin, which the patient was already receiving and blood culture was negative. No anaerobes were isolated in the specimens. Patient's condition remained stable for next 2 days after which he became progressively tachypneic and hypoxemic, with chest X-ray showing bilateral lower zone opacities. He required to be intubated and ventilated in view of persistent hypoxaemia. Over the next 3 days he made a gradual recovery. He was weaned off the inotrope and ventilator support. His platelet count improved to 379,000/cu mm, coagulation and chest X-ray normalized. After a further 3 days stay in intensive care unit, patient was discharged to ward. Throughout his stay in the hospital patient had a Glasgow coma scale 15/15 and demonstrated no neurological deficits. His further hospital course in the ward was uneventful and was discharged 3 weeks after his admission to the hospital. Oral linezolid was continued for 3wks in order to treat residual infective thrombus in the IJV.

## Discussion

Lemierre's syndrome or post-anginal septicemia is a potentially fatal complication of oropharyngeal infection that is characterized by IJV thrombosis and septic emboli, frequently involving the lungs. Classically it is caused by the organism *F. necrophorum*.[3] However, various other causative organisms described in literature include *streptococcus, staphylo*cocci, *Klebsiella, Eikenella* and *bacteroides* species. Also, mixed bacterial infection along with *F. necrophorum* has been reported.[3] Our patient grew *S. aureus* from the parapharyngeal abscess which is not very common.

*F. necrophorum* is a normal inhabitant of the oral cavity and is an obligate anaerobic non-motile Gram negative bacillus. Oropharynx is the primary source of infection in majority of cases. Otitis media, sinusitis, odontogenic infection and mastoiditis can also cause the syndrome. Infection of the lateral parapharyngeal space can result from any of these sources. The lateral parapharyngeal space is divided into an anterior muscular compartment and a posterior neurovascular compartment by the styloid process. Carotid sheath is located in the posterior compartment and infection of this compartment can result in thrombophlebitis of the IJV.[1] Factors that facilitate invasive infection are not clearly understood. Mucosal damage by prior bacterial or viral oropharyngeal infection may act as a precipitating factor. In contrast to other anaerobic bacteria, *F. necrophorum* possesses a lipopolysaccharide endotoxin. It aggregates human platelets without lysing them. The resulting intravascular coagulation creates an anaerobic environment. The septic thrombophlebitis in the tonsillar veins propagates to the IJV.[1] The exact pathogenesis of Lemierre's syndrome is not known, but lymphatic or direct spread of infection from thrombophlebitis of the tonsillar veins causing thrombosis of the IJV due to direct involvement of the alveolar tissue of the neck has been suggested as a possible mechanism.[1] The organisms can spread hematological via septic emboli to cause metastatic abscesses; most commonly the lungs and spread to the joints, liver and kidney have also been described.[4]

Though metastatic abscesses involving various organ systems are common, sepsis and septic shock requiring inotropic and ventilator support in patients with Lemierre's syndrome is not commonly described in literature.[5] Our patient was unique in this respect. Use of anticoagulants is controversial,[6] as no controlled trials exist. There are no clear recommendations on the use of anticoagulation. However, anticoagulation is frequently advocated in cases where there is involvement of sigmoid or cavernous sinuses. The concern with using systemic

anticoagulation is the risk of hemorrhage. We had to stop the anti-coagulant that we started initially in our patient as he developed deranged coagulation and low platelet count as part of sepsis. Anti-coagulant was restarted once his platelet count improved and coagulation normalized. The thrombosis of the IJV thrombus may or may not show resolution.[3,4] Our patient did not demonstrate resolution of the thrombus. If signs of sepsis persist with propagation of infection despite optimal treatment, ligation or excision of the IJV thrombus may be required.[7] Our patient, though he developed septic shock, did not require this extreme therapy.

The advent and widespread use of antibiotics has made this syndrome a rarity and is frequently overlooked. Critical care providers should be aware of the syndrome in patients with oropharyngeal infection who later present with signs of systemic illness or pulmonary involvement. A high degree of clinical suspicion is necessary for diagnosis. It is important to recognize it early and initiate adequate treatment to reduce morbidity and prevent mortality.

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