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Case Report

Atypical Presentation of Lemierre's Syndrome Masquerading as Gastroenteritis Lemierre's Syndrome Mimicking Gastroenteritis

Aslı Haykır Solay [1*], Dilek Bulut [1], Pelin Beyza Ünal [1], Semanur Kuzi [1]

[1] Infection Disease and Clinical Microbiology, Etlik City Hospital, Ankara, Turkey

*Correspondence: Aslı Haykır Solay (aahaykir@hotmail.com)

Abstract: Lemierre's syndrome (LS) is a rare clinical condition characterized by septic thrombophlebitis of the internal or external jugular vein, usually following an oropharyngeal infection. Here, we present a 20-year-old male patient who developed diarrhea, nausea, and vomiting after receiving clarithromycin for an upper respiratory tract infection. On admission, he had fever, hypotension, and elevation in acute phase reactants (WBC: 20,410/μL, CRP: 197 mg/L). Empirical treatment with ceftriaxone and metronidazole was initiated. Stool and throat cultures were negative. On the second day, abdominal tenderness developed; direct abdominal radiograph showed dilated bowel loops, but toxic megacolon was excluded during follow-up. Thoracic CT revealed septic emboli in the lungs. Due to persistent fever despite ceftriaxone and metronidazole therapy, treatment was escalated to meropenem on the fifth day. On the same day, blood cultures grew *Fusobacterium necrophorum*, raising suspicion of LS. Doppler ultrasound detected a thrombus in the left external jugular vein. Anticoagulant therapy with low-molecular-weight heparin and clopidogrel was initiated. The fever resolved by the seventh day of full antibiotherapy After three weeks of intravenous therapy, follow-up imaging showed regression of the thrombus. The patient completed a four-week course of antibiotics and anticoagulants and was discharged with full recovery. This case highlights the diagnostic challenge of LS presenting with gastrointestinal symptoms and emphasizes the importance of early blood cultures and imaging. External jugular vein involvement due to F. *necrophorum* is rare and should be considered in patients presenting with septic emboli.

Keywords: Lemierre Syndrome; *Fusobacterium necrophorum*; Septic Thrombophlebitis; External Jugular Vein Thrombosis; Septic Pulmonary Embolism

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1. Introduction

Lemierre's syndrome (LS) is a rare and potentially life-threatening condition characterized by septic thrombophlebitis of the internal or external jugular vein, typically occurring after an oropharyngeal infection [1]. First described by André Lemierre in 1936, this syndrome is classically associated with *Fusobacterium necrophorum*, an obligate anaerobic, non-spore-forming, non-motile, filamentous gram-negative bacillus that colonizes the oropharynx, gastrointestinal tract, and female genital tract [2]. It has a high virulence potential due to its ability to invade vascular endothelium and promote thrombosis [3]. Notably, *F. necrophorum* grows slowly in culture media, often taking five days or more, which can delay microbiological confirmation and diagnosis [4].

The clinical course usually begins with pharyngitis, followed by local invasion, bacteremia, and metastatic septic emboli, most often to the lungs. Although internal jugular vein thrombosis is typical, external jugular vein (EJV) involvement is exceedingly

rare, with only a few cases reported in the literature. In addition, initial presentation with gastrointestinal symptoms such as diarrhea or abdominal pain is uncommon and may obscure the diagnosis.

Here, we report a case of Lemierre's syndrome with two unusual features: initial manifestation as acute gastroenteritis, and thrombosis of the external jugular vein. To our knowledge, this is the first case documented in the literature of EJV thrombosis caused by *F. necrophorum* presenting primarily with gastrointestinal symptoms. The case highlights the importance of clinical suspicion, early blood cultures, and appropriate imaging in patients with persistent fever and signs of systemic infection.

2. Case Report

A 20-year-old male patient presented to the emergency department with complaints of diarrhea, nausea, and vomiting. Due to moderate dehydration and poor oral intake, he was admitted to our ward. His medical history revealed that a week earlier, he had presented with a sore throat and fever, for which he was started on clarithromycin, after which diarrhea began. He had been experiencing diarrhea five times a day for three days, which was non-bloody and without mucus.

On examination, he was conscious, mucous membranes were dry, abdomen was soft, blood pressure was 83/48 mmHg, heart rate was 78 bpm, and respiratory rate was 20 breaths per minute. Laboratory results showed white blood cell count (WBC): 20,410/µL, polymorphonuclear leukocytes (PMNL): 91.6%, C-reactive protein (CRP): 197 mg/L, erythrocyte sedimentation rate (ESR): 35 mm/h, urea: 39.8 mg/dL, creatinine: 0.85 mg/dL, alanine aminotransferase (ALT): 26 U/L, and aspartate aminotransferase (AST): 24 U/L. Blood, throat, and stool cultures were obtained, and the patient was started on intravenous (IV) ceftriaxone 1 g every 12 hours and metronidazole 500 mg every 8 hours for suspected antibiotic-associated diarrhea and salmonellosis.

Throat culture grew normal flora, and stool cultures were negative for *Salmonella spp.* and *Shigella spp.* On the second day of follow-up, abdominal tenderness and guarding developed, and a plain abdominal X-ray showed dilation of bowel loops. A diagnosis of toxic megacolon was considered. Oral intake was discontinued, a nasogastric tube was inserted, and a computed tomography (CT) scan of the abdomen was performed. Both clinical follow-up and abdominal CT excluded toxic megacolon, but septic emboli were seen in the pulmonary sections of the CT scan.

Due to persistence of fever on the fifth day of ceftriaxone, metronidazole, the regimen was switched to meropenem 1 g every 8 hours IV. On the first day of meropenem treatment, blood cultures obtained during hospitalization revealed *Fusobacterium necrophorum*. Due to the limited laboratory facilities in our hospital, susceptibility testing could not be performed. The treatment was continued witf ceftriaxone ande metronidazole in accordance with guideline recommendations. Based on this finding, Lemierre's syndrome was suspected, and a Doppler ultrasound of the neck was performed, revealing a thrombus in the left external jugular vein (EJV) extending from the proximal to distal one-third of the vein, filling 50% of the lumen. A thoracic CT scan revealed widespread septic emboli in both lungs.

Since the diagnosis of Lemierre's syndrome was confirmed, a search for the primary infection source was conducted. An otolaryngology consultation ruled out deep neck infection, and dental examination showed no evidence of odontogenic infection. Because the patient presented with gastroenteritis, an intra-abdominal infection was investigated with abdominal ultrasound, contrast-enhanced abdominal CT, and colonoscopy, all of which yielded no evidence of localized infection. A transthoracic echocardiogram was performed, revealing no vegetations or any findings suggestive of infective endocarditis.

Due to the thrombosis, the patient was started on subcutaneous low-molecular-weight heparin 0.6 mL twice daily and oral clopidogrel 75 mg once daily, as recommended by the cardiovascular surgery specialist. By the seventh day of total antibiotic therapy, a

clinical response to fever was observed, along with a downward trend in inflammatory markers (WBC: 13,250/μL; neutrophils: 85%; CRP: 87 mg/L). After three weeks of IV treatment, clinical improvement was noted, and a follow-up Doppler ultrasound showed a regression in the thrombus.

The patient was discharged on oral metronidazole 500 mg three times daily and intramuscular ceftriaxone 2 g once daily. Both antibiotic and anticoagulant/antiplatelet therapies were continued for a total of four weeks, resulting in complete recovery without sequelae.

3. Discussion

LS is an uncommon but potentially fatal condition, with a significantly increased risk of mortality when diagnosis is delayed or treatment is not promptly initiated. Although *Fusobacterium necrophorum* is the most commonly implicated pathogen in Lemierre syndrome, other organisms such as *Streptococcal* species, *Eikenella corrodens*, and *Staphylococcus aureus* have also been reported as causative agents [1].

LS often follows oropharyngeal infections. Physical examination of the oropharynx may reveal exudative, hyperemic, ulcerated, or normal tonsils. However, by the time septic thrombophlebitis or metastatic complications arise, significant oropharyngeal findings may already have resolved. Cervical lymphadenopathy may be present, whereas thrombosed jugular veins are rarely palpable. Gastrointestinal symptoms and signs are observed in approximately half of the patients [5]. In our case, head and neck examinations were normal at presentation and during follow-up. The patient initially presented with diarrhea but later developed abdominal pain and tenderness. Liver function tests remained within normal limits.

One to three weeks after the primary infection, local invasion and thrombosis of the internal jugular vein (IJV) may occur due to the spread of infection within the pharyngeal cavity [6]. Since venous drainage of the oropharynx primarily occurs through the IJV, thrombosis most commonly affects this vein. External jugular vein (EJV) thrombosis is rare, accounting for approximately 4% of cases [1]. Reported cases of EJV thrombosis are frequently associated with LS following streptococcal infections [7,8]. To our knowledge, this is the first documented case of LS with EJV thrombosis caused by *Fusobacterium necrophorum*.

During follow-up, careful monitoring is necessary for the development of local suppurative infections and possible need for surgical debridement. Approximately 70% of cases in the literature respond to antibiotic treatment alone, while the remaining require surgical intervention [1]. Several cases reported from our country have also required debridement [9-16]. In the present case, there was a history of pharyngitis but no evidence of contiguous spread to the deep neck spaces. The absence of complications requiring surgical intervention may be attributed to the early initiation of effective antimicrobial therapy.

Empirical treatment for LS caused by *F. necrophorum* typically includes piperacillin–tazobactam, imipenem or meropenem, or a combination of ceftriaxone and metronidazole [17]. In our case, treatment was initiated with ceftriaxone and metronidazole. Due to persistent fever, the regimen was escalated to meropenem. Following identification of the pathogen and clinical improvement, the patient was switched back to the initial regimen. Antibiotic treatment is generally recommended for 3–6 weeks, with at least two weeks of parenteral therapy [18,19]. In our case, ceftriaxone was administered parenterally for four weeks, and metronidazole was given parenterally for three weeks and orally for an additional week, resulting in full recovery.

Although there is no consensus on anticoagulation therapy, a 2020 meta-analysis of 394 cases reported no significant benefit in terms of vascular recanalization or mortality [1,19]. In our case, we opted not to administer anticoagulant or antiplatelet therapy due to the absence of coagulation abnormalities, and no complications were encountered.

However, the contribution of this decision to the overall outcome could not be fully assessed.

LS is a rare and diagnostically challenging disease. It should be considered in young adults presenting with recent pharyngitis, fever, neck tenderness or swelling, and/or pulmonary symptoms suggestive of septic emboli. A through history and physical examination, followed by blood cultures and appropriate imaging, are essential. During treatment, attention should be paid to the risk of metastatic infections and potential need for surgical drainage.

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