

Extensive Systemic Thrombosis in Polymicrobial Lemierre Syndrome Complicated By Diabetic Ketoacidosis: A Case Report and Therapeutic Dilemma

Case Report

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Keywords Key Clinical Message

Polymicrobial Lemierre syndrome in patients with metabolic derangements such as diabetic ketoacidosis may present with extensive systemic thrombosis, including intracranial involvement; early recognition and selective use of anticoagulation are critical despite limited consensus guidelines.

Abstract

Lemierre syndrome is a rare septic thrombophlebitis of the internal jugular vein typically caused by oropharyngeal infection and associated with septic emboli. We report a 50-year-old man with poorly controlled type 2 diabetes who presented with severe oropharyngeal infection complicated by Diabetic Ketoacidosis (DKA), bilateral internal jugular vein thrombosis with extension into the sigmoid and transverse venous sinuses, pulmonary embolism, and bilateral lower-extremity deep venous thromboses. Oropharyngeal cultures grew methicillin-sensitive *Staphylococcus Aureus* (MSSA) and *Serratia marcescens*. Management included broad-spectrum antibiotics, metabolic stabilization, and therapeutic anticoagulation following transient thrombocytopenia. This case highlights the expanding spectrum of Lemierre syndrome and the thromboinflammatory interplay between infection and metabolic crisis.

Keywords

Lemierre Syndrome; Septic Thrombophlebitis; Diabetic Ketoacidosis; Venous Thromboembolism; Anticoagulation

Introduction

Lemierre syndrome is a rare but potentially life-threatening condition characterized by septic thrombophlebitis of the internal jugular vein following an oropharyngeal infection. It is classically associated with *Fusobacterium necrophorum* and predominantly affects young, otherwise healthy individuals [1-3]. The clinical course typically involves pharyngitis, bacteremia, internal jugular vein thrombosis, and septic embolization, most commonly to the lungs [4].

In recent years, the epidemiology and clinical presentation of Lemierre syndrome have evolved [1-4]. Increasing reports describe atypical pathogens, polymicrobial infections, older patient populations, and significant comorbidities [2-4]. These factors may contribute to more severe disease manifestations, including widespread thromboembolism and involvement of nontraditional vascular territories.

Diabetes mellitus, particularly when complicated by metabolic derangements such as Diabetic Ketoacidosis

(DKA), is associated with impaired innate immunity, endothelial dysfunction, and a prothrombotic state [5,6]. These mechanisms may predispose patients to severe infections and exaggerated thromboinflammatory responses.

We describe a case of polymicrobial Lemierre syndrome in a patient with DKA complicated by extensive systemic thrombosis, including intracranial venous sinus involvement. This case underscores the interplay between infection, metabolic dysregulation, and thrombosis, and highlights ongoing uncertainty regarding the role of anticoagulation in management.

Case Presentation and Examination

A 50-year-old man with poorly controlled type 2 diabetes mellitus presented with a two-week history of progressive dysphagia, neck pain, oral discomfort, and generalized weakness. His symptoms evolved to include muffled voice, neck stiffness, and severe odynophagia, resulting in significantly reduced oral intake and inability to take prescribed medications. The patient reports no history of drug allergies, tobacco use, alcohol consumption or illicit drug use. Social and psychological functioning are intact. On examination, the patient appeared ill and dehydrated. Vital signs revealed tachycardia (135 beats/min), tachypnea (23 breaths/min), and hypertension (158/102 mmHg). He was afebrile with oxygen saturation of 98% on room air. Neurologic examination was nonfocal, with negative Kernig and Brudzinski signs.

Differential Diagnosis, Investigations, and Management

Laboratory evaluation (Table 1) demonstrated leukocytosis ($26.8 \times 10^3/\mu\text{L}$) with neutrophilic predominance, elevated D dimer (41.9 $\mu\text{g/mL}$), platelets ($375 \times 10^3/\mu\text{L}$), hemoglobin (14.1 g/dL). Metabolic studies were notable for severe hyperglycemia ($>1100 \text{ mg/dL}$), metabolic acidosis (pH 7.28), elevated anion gap (20), bicarbonate 19 mmol/L, and HbA1c of 12.7%, consistent with diabetic ketoacidosis complicated by dehydration and acute kidney injury which was managed with *intravenous fluids* and insulin in the ICU.

Contrast-enhanced CT of the neck revealed extensive pharyngeal inflammation with bilateral parapharyngeal fat stranding and thrombosis of both internal jugular veins, with extension into the left sigmoid and transverse dural venous sinuses (Figure 1). These findings were consistent with Lemierre syndrome. Microbiologic evaluation showed negative blood cultures; however, oropharyngeal cultures grew MSSA and *Serratia marcescens*.

Further imaging demonstrated systemic thromboembolic complications. CTA of the chest identified subsegmental pulmonary emboli in the left lower lobe with associated bibasilar opacities suggestive of pulmonary infarction (Figure 2). Duplex ultrasonography of the lower extremities revealed bilateral deep vein thromboses and superficial venous thrombosis (Figure 3 and 4). Transthoracic and transesophageal echocardiography did not demonstrate valvular vegetations, intracardiac thrombus, or intracardiac shunting.

During hospitalization, the patient developed acute thrombocytopenia (nadir $65 \times 10^3/\mu\text{L}$) and worsening anemia (hemoglobin 7.6 g/dL). Additional laboratory evaluation (Table 1) revealed iron 99 $\mu\text{g/dL}$, ferritin 3226 ng/mL, vitamin B12 1593 pg/mL, folate 10.3 ng/mL, mean corpuscular volume 94.9 fL, haptoglobin 360 mg/

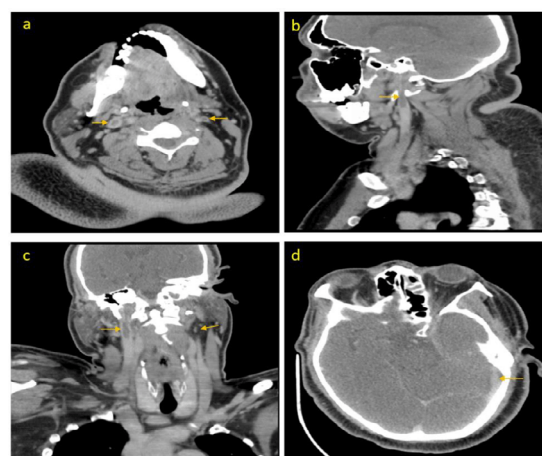


Figure 1: Contrast-enhanced CT neck demonstrating bilateral internal jugular vein thrombosis (arrows) on axial (a), sagittal (b), and coronal (c) views, with additional left transverse sinus thrombosis on axial intracranial imaging (d)

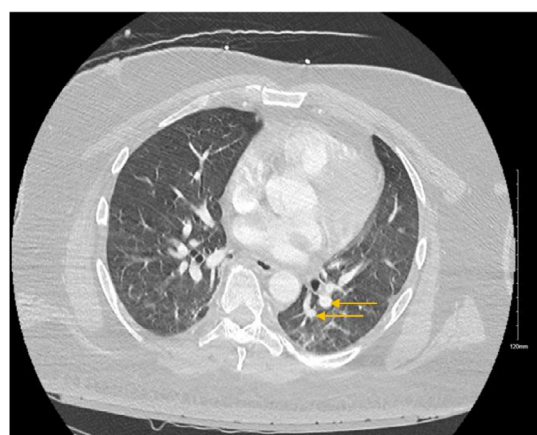


Figure 2: CT pulmonary angiography demonstrating left-sided subsegmental pulmonary embolism (arrows)

Table 1: Pertinent laboratory investigations during ED, during hospitalization, and upon discharge.

Test Parameters	Lab Value on Admission	Hospitalization	Discharge	Reference Range
White blood cells	26.8	19.30	6.4	4.50-11.0×10 ³ /μL
Reb blood cells	5.0	2.75	2.69	3.98-5.70
Platelets	375	65	399	150-450×10 ³ /μL
Hemoglobin	14.1g/dL	7.6	7.9	12-16.3 g/dL
Mean Corpuscular Volume (MCV)	94.8	94.9	95.9	81.0-96.0 fL
Glucose	>1100	142	110	74-106 mg/dL
Bicarbonate	19	25	26.4	22-29 mmol/L
pH	7.28			7.32-7.43
Hemoglobin A1c	12.7%			<5.7%
Potassium	6.1	4.2	3.6	3.5-5.1 mmol/L
D-dimer	41.9			<0.500 ug/mL
Iron		99		45-160 μg/dL
Ferritin		3226		30-400 ng/mL
Vitamin B12		1593		232-1245 pg/mL
Folate		10.3		>4.6 ng/mL
Haptoglobin		360		30-200 mg/dL
Fibrinogen		261		200-393 mg/dL
Absolute reticulocyte count		0.037 ×10 ⁶ /μL		
Prothrombin time (PT)	17.2	12.4	11.9	10.2-13.5 seconds
Activated partial thromboplastin time (aPTT)	22.9	25.7	33.5	26-40 seconds
INR		1.50	1.04	0.88-1.18 seconds

dL, fibrinogen 261 mg/dL, absolute reticulocyte count 0.037 ×10⁶/μL, prothrombin time 17.2 seconds, activated partial thromboplastin time 25.7 seconds, and INR 1.50. Evaluation for hemolysis and consumptive coagulopathy was unrevealing, suggesting an inflammatory etiology.

Patient was started on broad-spectrum intravenous antibiotics; vancomycin and meropenem and later de-escalated to ertapenem based on culture results. The patient completed a two-week course of antimicrobial therapy. Given the extensive thrombotic burden, therapeutic anticoagulation was considered; however, initiation was deferred due to thrombocytopenia and anemia. Following platelet recovery and clinical stabilization, anticoagulation was initiated with intravenous heparin and subsequently transitioned to apixaban.

The differential diagnosis included deep neck space infections (peritonsillar, parapharyngeal, or retropharyngeal abscess), mucormycosis in the setting of uncontrolled diabetes, disseminated intravascular coagulation, and infective endocarditis with septic embolization. Lemierre syndrome was favored based on characteristic imaging findings of internal jugular vein

thrombosis with intracranial extension in the context of oropharyngeal infection. Peritonsillar, parapharyngeal, or retropharyngeal abscess was ruled out based on physical examination and imaging. Disseminated intravascular coagulation and thrombotic microangiopathy were excluded based on preserved fibrinogen levels and absence of hemolysis. Infective endocarditis was ruled out by negative blood cultures and absence of valvular vegetations on echocardiography. Mucormycosis was excluded based on imaging findings, negative biopsy of the nasal turbinates, and negative fungal cultures.

Outcome and Follow Up

Following resolution of DKA, the patient was transitioned to subcutaneous insulin therapy with basal insulin glargine and prandial insulin. He was transferred to the general medical floor and demonstrated significant clinical improvement, including resolution of pain, improved oral intake, and glycemic control. Leukocytosis and thrombocytopenia normalized, and hemoglobin improved to 11.7 g/dL with a decrease in ferritin to 1093 ng/mL Patient was discharged home to complete a total three-month course of apixaban and outpatient follow up.

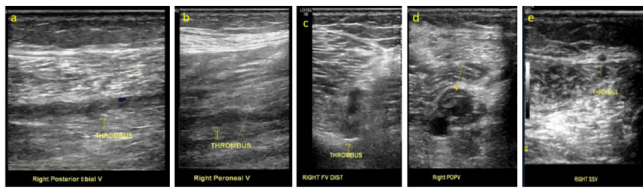


Figure 3: Right lower extremity duplex ultrasound demonstrating acute deep vein thrombosis of the (a) tibial, (b) peroneal, (c) femoral, and (d) popliteal veins, with associated superficial venous thrombosis of the small saphenous vein (e)

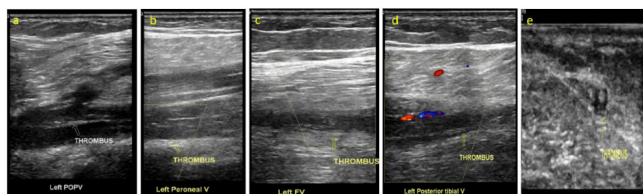


Figure 4: Left lower extremity duplex ultrasound demonstrating acute deep vein thrombosis of the (a) popliteal, (b) peroneal, (c) femoral, and (d) tibial veins, with associated superficial venous thrombosis of the small saphenous vein (e)

Discussion

This case illustrates a complex and atypical presentation of Lemierre syndrome characterized by polymicrobial infection, metabolic derangement, and extensive systemic thrombosis. While *Fusobacterium necrophorum* remains the most commonly implicated pathogen, increasing reports describe non-classical and polymicrobial etiologies, particularly in patients with underlying comorbidities [1-3].

The presence of methicillin-sensitive *Staphylococcus aureus* and *Serratia marcescens* in this case highlights the evolving microbiologic spectrum of Lemierre syndrome. These organisms are known to possess virulence factors that promote endothelial injury and coagulation activation, potentially contributing to thrombus formation [4].

The coexistence of DKA likely played a critical role in disease severity. Hyperglycemia and insulin deficiency impair neutrophil function and host immune responses, increasing susceptibility to invasive infections [5,6]. Additionally, DKA promotes a hypercoagulable state through dehydration, increased blood viscosity, endothelial dysfunction, and activation of inflammatory and coagulation pathways [6]. These mechanisms likely amplified the thromboinflammatory cascade characteristic of Lemierre syndrome, resulting in widespread thrombosis, including intracranial venous sinus involvement.

An important consideration in patients presenting with extensive, multi-territory venous thrombosis is the

possibility of underlying malignancy. Cancer-associated thrombosis may precede the diagnosis of occult malignancy [11-14]. Although the temporal association with infection and diabetic ketoacidosis supports a reactive process, the magnitude of thrombosis warrants evaluation for malignancy. No evidence of malignancy was identified during hospitalization; however, continued outpatient surveillance was recommended.

The thrombocytopenia in this patient further reflects the complex interplay between infection and coagulation. Severe bacterial infections, particularly those involving *Staphylococcus aureus*, can induce platelet activation, aggregation, and consumption through toxin-mediated and immune-mediated pathways [7]. The resolution of thrombocytopenia following antimicrobial therapy supports an infection-driven process.

Management of Lemierre syndrome relies on early recognition, prompt broad-spectrum antimicrobial therapy, and source control. The role of anticoagulation remains controversial due to the lack of randomized trials. However, observational data suggest potential benefit in selected patients, particularly those with extensive thrombosis or intracranial venous sinus involvement [3,8-10]. In this case, the presence of widespread thrombosis and cerebral venous sinus extension supported anticoagulation once thrombocytopenia improved, reflecting an individualized risk-benefit approach.

This case highlights several important clinical implications. First, Lemierre syndrome should remain a diagnostic consideration in patients presenting with severe oropharyngeal infection and thrombosis, even in atypical populations and in the absence of classic pathogens. Second, metabolic derangements such as DKA may significantly potentiate infection-associated hypercoagulability and should be recognized as contributors to disease severity. Finally, in patients with extensive or intracranial thrombosis, anticoagulation in addition to antimicrobial therapy may be a reasonable strategy, although further prospective data are needed to guide standardized management.

Conclusion

This case illustrates an atypical and severe presentation of Lemierre syndrome characterized by polymicrobial infection, diabetic ketoacidosis, and extensive systemic thrombosis. Metabolic derangements and acute infection likely amplify a thromboinflammatory cascade, contributing to disease severity and multi-territory thrombotic involvement. Clinicians should maintain a high index of suspicion for Lemierre syndrome in patients presenting with oropharyngeal infection and thrombotic complications, even in non-classical populations. The

presence of extensive thrombosis should also prompt evaluation for underlying malignancy in addition to infection-related causes. Anticoagulation may be considered in selected patients, particularly those with extensive or intracranial thrombosis, although further studies are needed to guide optimal management.

Patient Consent Statement

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Author Contributions

Albert Owusu-Ansah: Conceptualization, Data curation, Investigation, Project administration, Supervision, Writing original draft, Writing – review & editing

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Vanessa Osei-Bonsu: Investigation, Writing – review & editing

Jude Odinakachukwu Ossai: Investigation, Writing – review & editing

Hari Chandanaa Kistipati: Data curation, Investigation, Writing – review & editing

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