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Streptococcus constellatus–Associated Lemierre Syndrome Presenting With Cavitating Septic Pulmonary Emboli Managed by Video-Assisted Thoracoscopy

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

ABCDEF 1 Andre Fierro-Mosquera
BDEF 1 Camila Perez-Guerron
BDEF 1,2 Jordan Llerena-Velastegui
DF 1,2 Ruth Jimbo-Sotomayor
G 1,2 Xavier Sanchez

1 Pontificia Universidad Católica del Ecuador, Quito, Ecuador
2 Centro de Investigación para la Salud en América Latina (CISeAL), Quito, Ecuador

Corresponding Author: Ruth Jimbo-Sotomayor, Pontificia Universidad Católica del Ecuador, 12 de Octubre 1076 and Roca, Quito, Ecuador
Phone: +593 99 500 5177, e-mail: rejimbo@puce.edu.ec

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Patient: Male, 25-year-old
Final Diagnosis: *Streptococcus constellatus*-associated Lemierre syndrome with left internal jugular vein thrombosis, cavitating septic pulmonary emboli, empyema, and pulmonary abscess
Symptoms: Dyspnea • fever • hemoptysis • productive cough
Clinical Procedure: —
Specialty: General and Internal Medicine
Objective: Rare disease
Background: Lemierre syndrome is a rare septic thromboembolic disorder. Although historically associated with *Fusobacterium necrophorum*, non-fusobacterial cases caused by *Streptococcus constellatus* can present with dominant thoracic sepsis and delayed recognition.
Case Report: A 25-year-old previously healthy man from northern Ecuador presented with 7 days of dyspnea, productive cough, fever, and hemoptysis, with profound hypoxemia and respiratory distress. Chest computed tomography showed bilateral cavitating septic pulmonary emboli and necrotizing pulmonary infection, and cervical vascular imaging confirmed left internal jugular vein thrombosis. One blood culture set grew *Streptococcus constellatus* susceptible to penicillin and ceftriaxone; sputum and pleural fluid cultures were sterile. He required intensive care, endotracheal intubation, empiric piperacillin-tazobactam plus vancomycin, escalation to meropenem plus vancomycin during clinical deterioration while resistant or polymicrobial infection was considered, therapeutic anticoagulation, and 2 video-assisted thoracoscopic procedures for empyema and a subsequent pulmonary abscess. After 4 weeks of intravenous antibiotics, he improved and was discharged on a direct oral anticoagulant for 1 additional month, remaining asymptomatic at short-term follow-up.
Conclusions: Lemierre syndrome should be considered in young adults with severe sepsis and multifocal cavitating lung lesions, even without pharyngeal symptoms. Empiric therapy should initially cover oral aerobes and anaerobes; once penicillin-susceptible *Streptococcus constellatus* is confirmed and no co-pathogen is recovered, de-escalation to targeted beta-lactam therapy, such as penicillin G or ceftriaxone, should be considered together with drainage of empyema or abscess when present.

Keywords: Embolism • Empyema • Jugular Veins • Thrombophlebitis

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Introduction

Lemierre syndrome is a rare, often fulminant clinicopathologic entity characterized by septic thrombophlebitis of the internal jugular vein (or its tributaries), bacteremia, and metastatic septic embolization, most commonly to the lungs [1,2]. First delineated in 1936 by André Lemierre as an anaerobic postanginal septicemia associated with cervical thrombophlebitis and distant septic foci, the syndrome remains clinically relevant in the contemporary era because early manifestations can be nonspecific and diagnostic suspicion is frequently delayed [1]. Contemporary population-based data indicate a non-negligible incidence in adolescents and young adults, with clinically meaningful morbidity and mortality [3,4].

In a prospective survey from Denmark (1998-2001), the incidence of Lemierre syndrome was 3.6 cases per million population per year overall, increasing to 14.4 cases per million per year among individuals aged 15-24 years, and the overall mortality was 9% [3]. Similarly, a nationwide Swedish study of invasive *Fusobacterium necrophorum* infections (2010-2017) demonstrated an increase in incidence from 2.9 to 5.0 cases per million per year; Lemierre syndrome represented 35% of infections and was frequently complicated by sepsis, septic shock (18%), and intensive care unit admission (43%), even though 30-day mortality was 2% [4]. Beyond fatality, the clinical burden is driven by thrombotic progression, metastatic suppuration, and persistent sequelae; in recent individual patient-level analyses, early complications (new thrombosis or thrombosis extension, new peripheral septic lesions, and bleeding) occurred in approximately 13% of cases, and late sequelae were reported in about 10% of patients [5].

Because the initial upper-aerodigestive focus can be mild, transient, or clinically unapparent at the time of hospital presentation, diagnosis is commonly prompted by pulmonary manifestations of septic embolic disease and subsequently confirmed by directed evaluation of cervical vasculature [2,5]. Contrast-enhanced computed tomography (CT) of the neck is particularly useful for demonstrating internal jugular vein thrombophlebitis and perivascular inflammatory changes and may be the principal radiographic clue in patients lacking overt oropharyngeal symptoms [6]. Early recognition is clinically consequential, given that untreated or incompletely treated disease can rapidly progress to severe sepsis, respiratory failure, and pleuropulmonary suppuration [3-5].

Management is inherently multidisciplinary, focused on prompt antimicrobial therapy, timely source control, and individualized assessment of thrombotic and bleeding risks [3,5]. Prolonged broad-spectrum antimicrobial coverage with reliable anaerobic activity is generally required, with adjustment guided by cultures and susceptibility testing, and abscess drainage or pleural space interventions are frequently necessary as part of definitive source control when metastatic suppuration develops [3,5]. The role of therapeutic anticoagulation remains controversial, given the limited evidence and the potential need for concurrent invasive procedures. In a post hoc population-based observational study of 82 patients, jugular vein thrombosis was identified in 62% of those radiologically investigated, and those with jugular thrombosis had more severe thrombocytopenia and longer time to defervescence; however, thrombosis progression, septic complications, major bleeding, and 30-day mortality were similarly rare across groups receiving therapeutic-dose, prophylactic-dose, or no anticoagulation, supporting individualized decision-making rather than a uniform antithrombotic strategy [7].

Although *Fusobacterium necrophorum* remains the prototypical pathogen associated with Lemierre syndrome, modern analyses underscore that empiric management cannot presume uniform microbiology or antibiotic susceptibility [3-5]. A large-scale susceptibility and genomic analysis of contemporary *F. necrophorum* isolates identified resistance determinants and documented phenotypic resistance to agents commonly considered for treatment, emphasizing the importance of obtaining appropriate cultures and tailoring antimicrobial therapy to microbiologic results whenever feasible [8]. Together, these considerations reinforce the need for early broad empiric coverage at presentation, followed by rational de-escalation and targeted source control based on evolving clinical and radiologic findings [5,8].

Current evidence supports a broader conceptualization of Lemierre syndrome as a thrombogenic and septic embolic phenotype that can be produced by multiple bacterial species. In a 2025 individual patient-level analysis, bacterial growth was detected in 574 of 712 reported cases; *Fusobacterium* spp. were isolated in 415 patients, whereas *Streptococcus* spp. or *Staphylococcus* spp. were isolated in 108, and other bacteria in 51, indicating that non-fusobacterial etiologies comprise a clinically meaningful subset [5]. Notably, internal jugular vein thrombosis at presentation was highly prevalent among cases involving *Streptococcus* spp. or *Staphylococcus* spp. (94%), consistent with a syndrome in which thrombophlebitis and embolic disease can arise even when *Fusobacterium* is absent or not recovered [5]. In the 394-case meta-analysis cited later in this report, *Streptococcus constellatus* was explicitly reported in 6 patients (1.5%), underscoring the rarity of this specific organism among published Lemierre syndrome cases [19].

Within this spectrum, *Streptococcus constellatus* merits particular attention because it is a member of the *Streptococcus anginosus* group (SAG), a group of oral facultative anaerobes increasingly recognized as invasive pyogenic pathogens and increasingly implicated in pleuropulmonary infections, including

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pneumonia complicated by empyema [9,10]. Invasive SAG infections are characterized by a strong association with abscess formation and frequent polymicrobial growth; in a tertiary-care cohort of sterile-site SAG infections, abscesses accounted for 68% of cases, polymicrobial infection was common, and mortality was 6%, despite a high overall susceptibility to penicillin (91%) [10]. In a larger contemporary cohort of 390 hospitalized patients with confirmed *S. constellatus* infection, short-term mortality remained substantial (30-day 13%), with outcomes strongly influenced by polymicrobial infection and infection site, highlighting that severe, invasive disease can occur even when in vitro susceptibility is preserved [11]. *Streptococcus constellatus*–associated Lemierre syndrome has also been documented in the literature, supporting the clinical validity of Lemierre syndrome diagnoses in which *Fusobacterium* is not isolated and reinforcing the need to maintain broad microbiologic consideration in empiric management strategies [12]. For this reason, the present report is framed primarily around *Streptococcus constellatus* and *Streptococcus anginosus* group biology rather than classic fusobacterial postanginal sepsis, because the clinical course was driven by abscess-forming pleuropulmonary disease.

Thus, this case report shows that severe cavitating pulmonary septic emboli with fulminant pleuropulmonary sepsis can dominate the presentation, prompting targeted cervical imaging even without oropharyngeal symptoms, and that recovery may require repeated thoracic source-control procedures and tailored therapy.

Case Report

A 25-year-old man born and residing in northern Ecuador, who worked as a veterinarian and had no known allergies, no prior medical history, and no tobacco, alcohol, or intravenous drug use, presented to the emergency department after 7 days of progressive dyspnea, productive cough with purulent sputum, fever, and hemoptysis. On arrival, he appeared acutely ill, with marked tachypnea, tachycardia, oxygen saturation 60% on room air, and borderline blood pressure. He remained alert and conscious; however, he exhibited evident respiratory distress. He denied any recent sore throat, neck pain, or dental procedures, and examination of the oropharynx and neck was unremarkable. Physical examination disclosed several pustular lesions on the anterior chest, absent breath sounds at both lung bases, diffuse crackles throughout both lung fields, and scleral icterus. Given the severity of his respiratory compromise and the hemodynamic risk suggested by his initial presentation, he was stabilized in the emergency setting and transferred to the intensive care unit. The anterior chest pustules were documented clinically; however, no culture or biopsy of these lesions was documented in the clinical records reviewed for this report. Therefore, their relationship to the invasive infection cannot be definitively established, and a cutaneous portal of entry remains possible but unproven.

Initial laboratory evaluation demonstrated leukocytosis with a left shift, abnormal liver function tests, elevated creatinine, and respiratory alkalosis, consistent with severe systemic infection accompanied by early multiorgan dysfunction. Noncontrast chest CT showed findings highly suggestive of septic pulmonary embolization and necrotizing pulmonary infection (**Figure 1**). As this constellation raised concern for septic thrombophlebitis as the source of metastatic pulmonary disease, vascular imaging of the neck was performed. Doppler ultrasonography and contrast-enhanced neck CT confirmed thrombosis of the left internal jugular vein, along with absence of flow in the left external jugular vein (**Figure 2**). Complete numerical laboratory values for the assessed 3 time points (initial admission, post-treatment/post-video-assisted thoracoscopic surgery [VATS], and outpatient follow-up) were unavailable in the clinical records reviewed for this report; consequently, the laboratory course is reported qualitatively, and this limitation is acknowledged below.

Two sets of blood cultures were obtained during the diagnostic evaluation prior to antibiotic therapy. One set grew *Streptococcus constellatus* (susceptible to penicillin and ceftriaxone), whereas the other was sterile. Sputum culture showed no growth of pathogenic microorganisms. Pleural fluid collected during thoracoscopic drainage was cultured and remained sterile. In view of his occupational exposure as a veterinarian, brucellosis serology was also requested and proved negative. Taken together, the clinical, radiologic, vascular, and microbiologic findings supported a diagnosis of severe septic thrombophlebitis of the left internal jugular vein complicated by bilateral cavitating septic pulmonary emboli, pleuropulmonary sepsis, and acute hypoxemic respiratory failure. The available microbiology report identified the organism to species level and provided penicillin and ceftriaxone susceptibility results, but it did not specify the identification platform (eg, MALDI-TOF mass spectrometry versus automated biochemical identification) or whether the positive blood-culture signal occurred in the aerobic or anaerobic bottle. Right-sided infective endocarditis and occult immunodeficiency were considered relevant differential/contextual issues in this presentation; however, the clinical records reviewed for this report did not include echocardiography results, HIV serology, or quantitative immunoglobulin levels, so definitive statements about these evaluations cannot be made.

The patient was admitted to the intensive care unit, underwent endotracheal intubation, and was started on empiric intravenous piperacillin-tazobactam plus vancomycin. During the first 5 days of critical care, he improved sufficiently to permit transfer to the hospital ward, with normalization of vital

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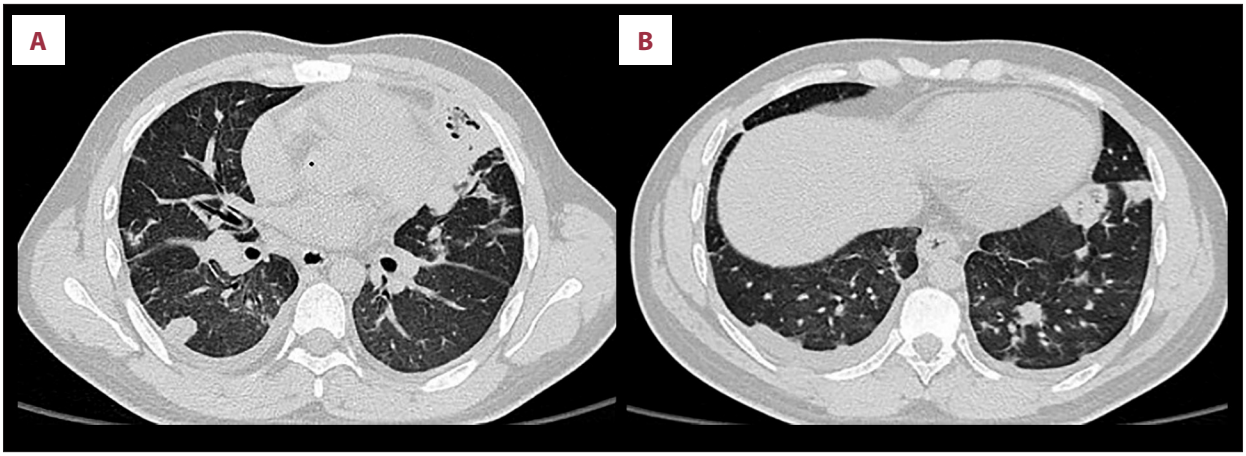

Figure 1. (**A, B**) Non-contrast chest computed tomography demonstrates bilateral heterogeneous pulmonary opacities with interlobular septal thickening, associated with multiple randomly distributed nodular and pseudonodular lesions in both central and peripheral locations, as well as air bronchograms in both lung fields. Bilateral pleural effusions with associated compressive atelectatic changes are also observed.

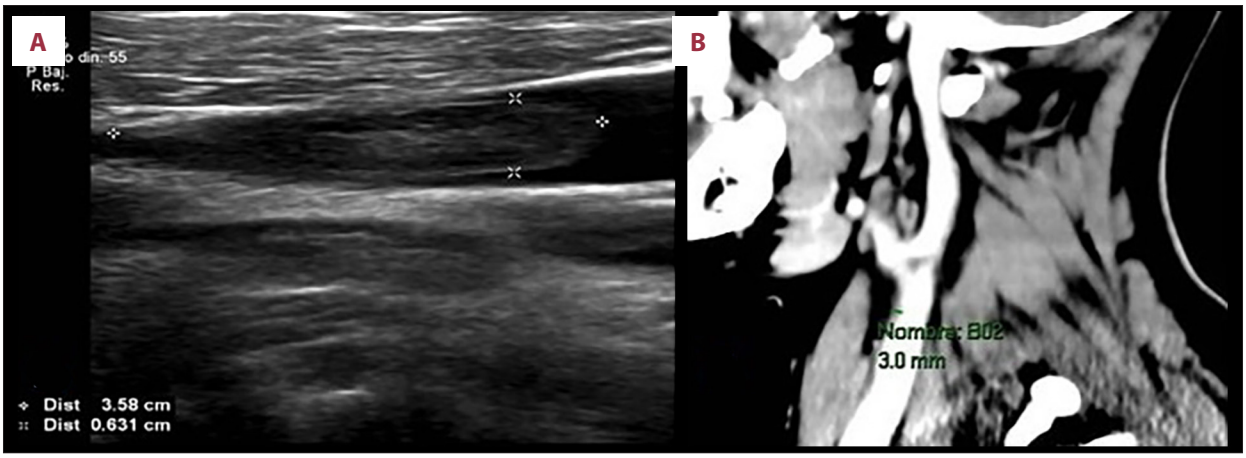

Figure 2. Doppler ultrasound and contrast-enhanced neck computed tomography demonstrate (**A**) an intraluminal thrombus within the left internal jugular vein on Doppler ultrasound, measuring 3.58 cm in longitudinal diameter, 0.63 cm in anteroposterior diameter, and 0.77 cm in transverse diameter, adherent to the lateral wall and compromising approximately 66% of the vascular lumen; and (**B**) a thrombus in the left internal jugular vein on contrast-enhanced neck computed tomography, causing approximately 25% luminal stenosis, with absent flow in the left external jugular vein.

signs and oxygen requirements reduced to low-flow supplementation. On hospital day 5, persistent fever and new imaging findings consistent with an empyema prompted video-assisted thoracoscopic decortication with pleural drainage and chest-tube placement for source control. The empyema was characterized radiologically by a right-sided loculated, high-attenuation pleural collection with pleural thickening and compressive atelectatic change; pleural fluid obtained during thoracoscopic drainage remained sterile, supporting a treated or culture-negative empyema. The Infectious Diseases service was consulted at that time, and antimicrobial therapy was escalated to intravenous meropenem (continuing vancomycin) to cover potential resistant pathogens given the complicated course. This escalation was made because of persistent fever, new empyema, critical illness, and concern for secondary polymicrobial or hospital-acquired infection, not because of resistance in the *Streptococcus constellatus* isolate, which was reported as susceptible to penicillin and ceftriaxone. Despite partial postoperative improvement, follow-up CT 5 days later demonstrated a new right-sided pulmonary abscess (**Figure 3**). Because of this new suppurative complication, a second video-assisted thoracoscopic procedure was performed. At that point, Vascular Surgery was consulted; after assessing his clinical status and post-procedural bleeding risk, they recommended initiating therapeutic anticoagulation with low-molecular-weight heparin. Thereafter, the patient completed 4 weeks of intravenous antibiotic therapy, with progressive clinical recovery, resolution of fever, and sustained respiratory improvement.

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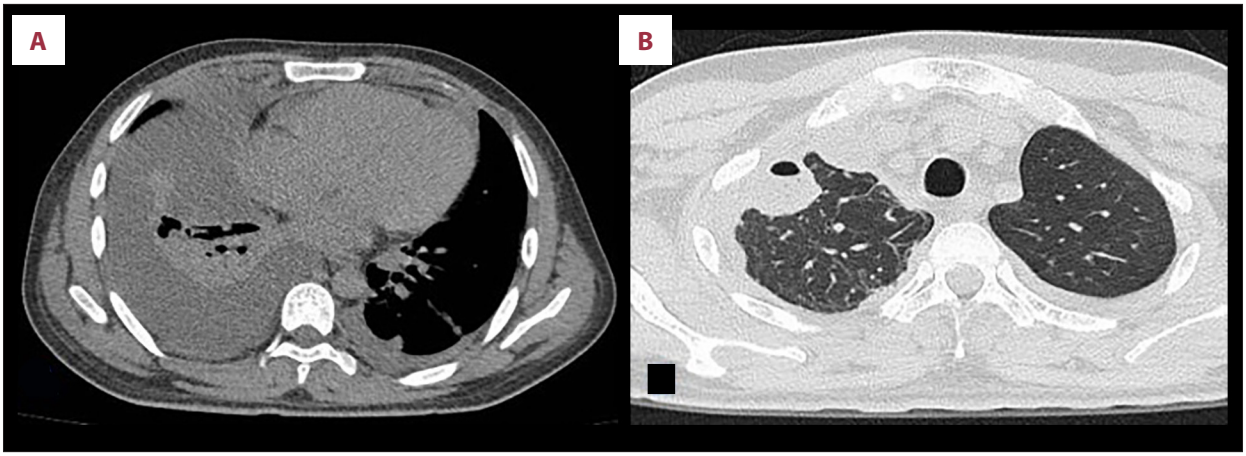

Figure 3. Non-contrast chest computed tomography demonstrates (**A**) a right-sided pleural effusion with high-attenuation fluid and loculated collections extending toward the apical and basal regions, and (**B**) a rounded pleural-based fluid collection in the right pulmonary apex containing internal gas bubbles, measuring 68 × 31 mm, with associated pleural thickening, consistent with a complex pleuropulmonary suppurative process.

At discharge, he was clinically stable and was prescribed a direct oral anticoagulant for 1 additional month.

Twelve days after hospital discharge, he was re-evaluated in the outpatient clinic and reported complete resolution of symptoms. Pulmonary auscultation was normal, routine laboratory testing showed no abnormalities, and chest radiography demonstrated no new infiltrates, thereby supporting a favorable short-term outcome after combined antimicrobial, surgical, and anticoagulant management. The admission and follow-up chest radiograph images were not available as separate figure files for inclusion; therefore, the follow-up radiographic finding is reported narratively.

Discussion

Lemierre syndrome is a rare but potentially catastrophic septic thromboembolic disorder classically defined by an antecedent oropharyngeal infection, septic thrombophlebitis of the internal jugular vein (or its tributaries), and metastatic septic embolization, most frequently to the lungs [1,2]. Although the archetypal clinical narrative begins with pharyngotonsillitis followed by unilateral neck pain or swelling and subsequent systemic toxicity, contemporary evidence emphasizes substantial heterogeneity in early manifestations and in microbiology, which can delay diagnostic recognition and contribute to severe organ dysfunction at first presentation [3-6,13-15]. The present case is clinically instructive because the dominant initial syndrome was fulminant pleuropulmonary sepsis with cavitating pulmonary lesions and acute hypoxemic respiratory failure, and the diagnosis of Lemierre syndrome was established only after thoracic imaging and directed evaluation of the cervical venous system demonstrated internal jugular vein thrombosis, thereby unifying the pulmonary findings as septic embolic disease [6].

From an internal medicine perspective, an important diagnostic implication is that septic pulmonary emboli may represent the principal clinical "signal" prompting recognition of an otherwise occult septic thrombophlebitis. CT patterns consistent with septic embolization include multiple peripheral nodules, frequent cavitation, wedge-shaped pleural-based lesions, and pleural extension; critically, CT can suggest the diagnosis even when chest radiography is nonspecific, and can reveal a greater extent of pleural involvement than plain radiographs [16]. In an appropriate clinical context, recognition of this pattern should trigger a systematic search for an intravascular septic source, including deep neck space infection with internal jugular thrombophlebitis, right-sided endocarditis, and infected venous catheters, among other causes [16]. For Lemierre syndrome specifically, contrast-enhanced CT of the neck is particularly valuable to demonstrate venous thrombosis and perivascular inflammatory changes and to evaluate for deep neck space infection when the oropharyngeal examination is unrevealing or when the patient presents primarily with pulmonary disease [6]. These considerations are directly applicable to the present case because pulmonary imaging preceded and enabled targeted identification of the cervical thrombotic focus. In the present report, the radiologic sequence follows this diagnostic pathway from initial septic embolic chest CT (**Figure 1**), to confirmation of jugular thrombosis (**Figure 2**), and to pleural and abscess complications requiring VATS source control (**Figure 3**).

Microbiologically, Lemierre syndrome was historically described as "postanginal septicemia" caused by anaerobic organisms, with *Fusobacterium necrophorum* regarded as the prototypical

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pathogen because of its virulence and its association with severe monomicrobial disease in otherwise healthy hosts [1,17]. Nonetheless, it is increasingly evident that restricting diagnostic consideration to *Fusobacterium* is clinically unsafe. Contemporary conceptual frameworks and empirical data support a broader view of Lemierre syndrome as a clinical phenotype that can be produced by diverse organisms, including streptococci and staphylococci, and that may be culture-negative when antibiotic exposure precedes specimen collection or when fastidious organisms are involved [5,15,18,19]. This broader microbiologic reality has immediate therapeutic consequences: empiric therapy at presentation must provide reliable anaerobic coverage while also targeting likely aerobic pathogens, with subsequent de-escalation, when feasible, based on microbiologic results and evolving clinical and radiologic findings [8,15,19].

Within the non-fusobacterial spectrum, *Streptococcus constellatus* is a biologically plausible cause of fulminant Lemierre syndrome. *Streptococcus constellatus* is a member of the *Streptococcus anginosus* group, organisms that commonly colonize the oral cavity and upper-aerodigestive tract but demonstrate a notable propensity for invasive pyogenic infection and abscess formation, including pleuropulmonary suppuration [9-11,20]. Moreover, invasive *S. constellatus* infection can carry clinically meaningful mortality, and outcomes appear strongly influenced by infection site and polymicrobial infection, showing that severe disease can occur even when beta-lactam susceptibility is preserved in vitro [11]. *S. constellatus*–associated Lemierre syndrome has been reported in the literature, reinforcing that internal jugular septic thrombophlebitis with embolic lung disease can occur in the absence of *Fusobacterium* recovery and supporting the diagnostic validity of cases such as the present one [12]. In this context, the development of empyema and subsequent pulmonary abscess requiring repeated operative source control is concordant with the known tropism of *Streptococcus anginosus* group pathogens for suppurative complications and with the broader Lemierre syndrome literature in which metastatic thoracic suppuration frequently drives morbidity [9,10,20]. Published Lemierre syndrome experience with *Streptococcus constellatus* remains limited: in the 394-case meta-analysis, *Streptococcus constellatus* accounted for 6 reported cases (1.5%), while broader *Streptococcus anginosus/intermedius/milleri*-group isolates were also uncommon [19]. This supports our observation that the case should be interpreted principally through the lens of this unusual SAG pathogen.

Therapeutically, the present case underscores 2 principles that are central to contemporary Lemierre syndrome management: prolonged antimicrobial therapy and decisive source control. Reviews emphasize that mortality and morbidity are largely preventable with prompt, appropriate antibiotics, but also highlight persistent variability in antibiotic selection and duration, reflecting the absence of randomized data and the need to individualize therapy to metastatic foci and clinical response [13-15]. In a meta-analysis of 394 published Lemierre syndrome cases, the average antibiotic duration was approximately 34 days (intravenous plus oral), and a substantial subset of patients required surgical interventions in addition to antibiotics, consistent with a syndrome in which drainage of abscesses or pleural space infection is often decisive for cure when suppurative complications develop [19]. Pragmatically, empiric regimens frequently incorporate beta-lactam/beta-lactamase inhibitor combinations, carbapenems, and/or metronidazole, reflecting the need for dependable anaerobic coverage and the real-world occurrence of polymicrobial infection [15,19]. Even when a non-fusobacterial organism is isolated, early broad coverage remains rational until clinical stability is achieved and co-infection has been reasonably excluded, particularly in critically ill patients with cavitating pulmonary disease and pleural sepsis [15,19]. In addition, modern susceptibility surveillance has identified resistance determinants among contemporary *Fusobacterium necrophorum* isolates, underscoring that empiric choices should not assume uniform susceptibility and reinforcing the broader imperative for culture acquisition and targeted therapy whenever feasible [8].

In the 394-case meta-analysis, Gore reported that 120 of 394 patients (30.5%) received antibiotics plus a surgical procedure, whereas 274 (69.5%) received antibiotics alone; the analysis did not provide a separate pooled percentage limited specifically to abscess or empyema drainage. Mortality in that series was 16 of 394 patients (4.1%), and septic pulmonary emboli were reported in 148 of 394 patients (37.6%) [19]. For antimicrobial therapy, empiric treatment of suspected Lemierre syndrome should cover oral anaerobes and aerobic streptococci/staphylococci, commonly with a beta-lactam/beta-lactamase inhibitor, a carbapenem, or ceftriaxone/penicillin plus metronidazole when anaerobic co-infection is plausible [15,19]. Once *Streptococcus constellatus* is identified as penicillin- and ceftriaxone-susceptible and no co-pathogen is recovered, targeted beta-lactam therapy such as penicillin G or ceftriaxone is stewardship-concordant, while duration should be individualized to bacteremia clearance, thrombus stability, pulmonary/pleural source control, and clinical response [10,11,15,19].

In retrospect, the emergence of empyema and a lung abscess in this case is consistent with *Streptococcus anginosus* group pyogenic biology and, by itself, does not prove antimicrobial spectrum failure [9,10,20]. The continued meropenem plus vancomycin approach was a conservative decision during critical illness and concern for undetected polymicrobial or hospital-acquired infection; however, because pleural fluid culture was sterile and no co-pathogen was isolated, earlier de-escalation to a targeted beta-lactam regimen could also have

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been justified and is acknowledged as the more stewardship-aligned strategy.

The role of therapeutic anticoagulation remains the most debated component of Lemierre syndrome management, particularly in patients who require invasive procedures and in those with sepsis-associated thrombocytopenia or coagulopathy. Observational and case-based data show wide practice variation, and available evidence does not support a uniform antithrombotic strategy [7,18,19,21,22]. In the 394-case meta-analysis, anticoagulation was used in approximately two-thirds of patients, yet pooled analyses did not demonstrate a statistically significant association between anticoagulation and vessel recanalization or mortality among those with available outcome and follow-up imaging data [19]. Similarly, a retrospective institutional series did not identify improved thrombosis outcomes attributable to anticoagulation, with thrombus improvement observed over time in both anticoagulated and non-anticoagulated patients [21]. Conversely, in a large individual patient-level analysis, clinically relevant post-diagnosis events (new or recurrent venous thromboembolism and new peripheral septic lesions) occurred despite therapy, and these events were less frequent among anticoagulated patients, while major bleeding occurred in a minority (2.9%), emphasizing the need for individualized assessment of thrombotic burden, embolic risk, bleeding risk, and procedural requirements [18]. Expert syntheses have consequently proposed anticoagulation in selected scenarios such as poor clinical response despite appropriate antibiotics and source control, suspected thrombophilia, and intracranial venous extension, provided that the bleeding risk is acceptable [22]. The present case illustrates how anticoagulation decisions may reasonably evolve over the disease course: once thoracic source control had been achieved and the risk-benefit balance favored thrombosis management, anticoagulation was instituted and continued briefly after discharge; however, high-quality data to guide optimal agent selection and duration remain limited, particularly for direct oral anticoagulants, which have been reported only rarely in published case aggregations [19].

Several limitations warrant acknowledgement. Single-case inference cannot disentangle the relative contributions of antimicrobial selection, timing and completeness of source control, and anticoagulation to the favorable outcome. Additionally, as in many Lemierre syndrome cases, microbiologic ascertainment may be incomplete because cultures may be negative or may not fully capture polymicrobial infection when antibiotic therapy precedes sampling or when deep foci are not sampled; therefore, the possibility of undetected co-pathogens cannot be excluded [5,8,15]. Future research priorities include prospective or registry-based studies to define evidence-based criteria for anticoagulation, to clarify optimal duration and route of antimicrobial therapy in patients with extensive pulmonary and pleural suppuration, and to better characterize non-fusobacterial Lemierre syndrome phenotypes, including Streptococcus anginosus group–associated disease [14,15,18,19]. Notwithstanding these limitations, this case illustrates a high-yield clinical heuristic for internists: in young, previously healthy adults presenting with severe sepsis and cavitating pulmonary lesions consistent with septic emboli, early directed evaluation for jugular venous thrombophlebitis is essential, and definitive management commonly hinges on coordinated antimicrobial therapy, timely pleural or abscess source control, and individualized antithrombotic decision-making [6,15,16,18,19]. Additional limitations relate to incomplete clinical documentation: skin-lesion culture/biopsy, echocardiographic exclusion of right-sided infective endocarditis, HIV or immunoglobulin screening, complete quantitative laboratory values across all assessed time points, the blood-culture bottle type, and the specific microbiological identification platform were unavailable in the clinical records reviewed for this report. Admission and follow-up 2-view chest radiographs were also not available as separate image files for inclusion. These gaps constrain causal interpretation of the anterior chest pustules, definitive exclusion of a cardiac embolic source, and assessment of a predisposing immune condition.

Conclusions

Although Lemierre syndrome is classically linked to antecedent oropharyngeal infection and *Fusobacterium necrophorum*, this case illustrates that it should also be considered in previously healthy young adults who present primarily with fulminant pleuropulmonary sepsis, cavitating septic pulmonary emboli, and acute hypoxemic respiratory failure, even in the absence of prominent pharyngeal symptoms and even when a non-fusobacterial pathogen such as *Streptococcus constellatus* is isolated. Thus, when evaluating severe sepsis with multifocal cavitating pulmonary lesions, internists should maintain a high index of suspicion for septic thrombophlebitis of the internal jugular vein and pursue early directed imaging of the cervical venous system to establish or exclude this diagnosis. In addition, this case highlights that achieving a favorable outcome may require a multidisciplinary approach: early broad-spectrum antibiotics tailored by culture results, prompt surgical source control of thoracic suppurative foci, and individualized anticoagulation decisions [15,18]. For invasive *Streptococcus constellatus* Lemierre syndrome, empiric therapy should initially cover oral aerobic and anaerobic flora; after susceptibility confirmation, de-escalation to targeted beta-lactam therapy such as penicillin G or ceftriaxone should be considered when no co-pathogen is recovered, with continued anaerobic coverage reserved for situations in which polymicrobial infection remains plausible and with prompt drainage of empyema or abscess when present.

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Institution Where Work Was Done

Hospital Axxis, Quito, Ecuador.

Patient Permission/Consent Declaration

Written informed consent was obtained from the patient for publication of this case report and accompanying images. No identifiable patient information is included in the manuscript.

Declaration

The authors declare no relevant disclosures and further affirm that the study was conducted entirely independently, without any external assistance.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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