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Lemierre's Syndrome: A Systematic Review

Peter D. Karkos, AFRCS, MPhil; Sheetal Asrani, MBBS; Christos D. Karkos, FRCS, PhD; Samuel C. Leong, MRCSEd, DO-HNS; Evangelia G. Theochari, MD; Thalia D. Alexopoulou, MD; Assimakis D. Assimakopoulos

Objectives/Hypothesis: Lemierre's syndrome is characterized by a history of recent oropharyngeal infection, clinical or radiological evidence of internal jugular vein thrombosis, and isolation of anaerobic pathogens, mainly *Fusobacterium necrophorum.* It was once called the forgotten disease because of its rarity, but it may not be that uncommon after all. This review aims to provide physicians with an update on the etiology, management, and prognosis of Lemierre's syndrome.

Methods: Systematic review using the terms: Lemierre's syndrome, postanginal septicemia, fusobacterium, internal jugular vein thrombosis. Inclusion criteria: English literature; reviews, case reports, and case series. Exclusion criteria: variants or atypical Lemierre's syndrome cases, negative fusobacteria cultures, and papers without radiological evidence of thrombophlebitis.

Results: Eighty-four studies fulfilled our inclusion criteria. The male to female ratio was 1:1, 2, and the ages ranged from 2 months to 78 years (median, 22 years). Main sources of infection were tonsil, pharynx, and chest. Most common first clinical presentation was a sore throat, followed by a neck mass and neck pain. The most common offending micro-organism was *F. necrophorum*. Treatment modalities used were antimicrobial, anticoagulant, and surgical treatment. Morbidity was significant with prolonged hospitalization in the majority of patients. The overall mortality rate was 5%.

Conclusions: Lemierre's syndrome may not be as rare as previously thought. This apparent increase in the incidence may be due to antibiotic resistance or changes in antibiotic prescription patterns. Successful management rests on the awareness of the condition, a high index of suspicion, and a multidisciplinary team approach.

Key Words: Lemierre's syndrome, postanginal septicemia, internal jugularvein thrombosis, fusobacterium, necrobacillosis.

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INTRODUCTION

Lemierre's syndrome is characterized by a history of recent oropharyngeal infection, clinical or radiological evidence of internal jugular vein thrombosis, and isolation of anaerobic pathogens, mainly *Fusobacterium necrophorum*.[1] There has been an increase in reporting of Lemierre's syndrome over the last 10 years.[2] Whether this reflects a true increase in the incidence of the syndrome or just a literature/publishing trend remains to be seen. The aim of this study was to assess the incidence, etiology, management, and prognosis of Lemierre's syndrome, and identify possible causes behind this rapid recent rise in the incidence of a previously rare entity.

MATERIALS AND METHODS

A systematic review of the literature by a MEDLINE search was performed using the terms: Lemierre's syndrome, postanginal septicemia, fusobacterium, and internal jugular vein thrombosis. Inclusion criteria was English language literature only. Reviews, case reports, and case series of both adult and the pediatric population were included. Exclusion criteria were variants or atypical Lemierre's syndrome cases and negative cultures for any of the fusobacteria types. Papers without radiological evidence of thrombophlebitis, that is, studies where evidence of thrombophlebitis was based on clinical criteria alone, were also excluded.

RESULTS

One hundred two studies were published from 1950 to 2007. Eighty-four fulfilled our inclusion criteria, totaling 114 patients for review.[3–86] Selection and information bias, lack of uniform reporting, and inclusion of low methodological quality studies prevented a formal meta-analysis. The male to female ratio was 1:1, 2, and the ages ranged from 2 months to 78 years (median, 22 years). Most cases presented in the 2nd decade of life (51%), followed by the 3rd decade (20%), and then the 1st decade (8%). The main sources of infection were

From the Department of Otolaryngology, Liverpool University Hospitals, Liverpool, United Kingdom (P.D.K., S.A., S.C.L.); The 5th Department of Surgery, Aristotle University of Thessaloniki, Hippocrateio Hospital, Thessaloniki, Greece (C.D.K., T.D.A.); the Department of Medicine, Royal Liverpool University Hospital, Liverpool, United Kingdom (E.G.T.); and Alexandroupoli, Greece (A.D.A.).

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Send correspondence to Peter D. Karkos, 36 Hopkinsons Court, Walls Avenue, Chester, CH1 4LN, UK. E-mail: pkarkos@aol.com

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TABLE I.
Source of Infection for Lemierre's Syndrome.

Source of Infection	%
Tonsil	37
Pharynx/URTI	30
Chest/LRTI	25
Middle ear/mastoid	2
Larynx	2
Dental	1
Paranasal sinuses	1
Orbit	1
Metastatic disease	0.5
Gastrointestinal	0.4
Lip piercing	0.1

URTI = upper respiratory tract infection; LRTI = lower respiratory tract infection.

tonsil, pharynx, and chest, followed by larynx and middle ear (Table I). The most common first clinical presentation was a sore throat, followed by a neck mass and neck pain (Table II). The main offending microorganisms were *F. necrophorum* (57%), *Fusobacterium species* (30%) and *Fusobacterium nucleatum* (3%), followed by anaerobic streptococci and other miscellaneous Gram-negative anaerobes (10%).

Chest x-ray was the first-line investigation in the vast majority of patients (92%). This was reported as showing some form of consolidation in 75% of cases and as normal in approximately 10% of patients. The most commonly requested scan was computed tomography of neck/chest (55% of cases), followed by ultrasound (26%), magnetic resonance imaging (6%), or a combination of scans, mainly ultrasound and magnetic resonance imaging (6%). Magnetic resonance venogram and gallium scans were requested in 4% of cases. The hospital stay ranged from 4 to 112 days (median, 25 days), with 58% of patients requiring admission to the intensive care unit for a median of 21 days (Table III). Treatment modalities used were antimicrobial, anticoagulant, and surgical treatment, or a combination of the above. Anticoagulation was administered in 30% of the patients. Morbidity (Tables IV and V) was significant, resulting in prolonged hospital and/or intensive care unit stay in more than one half of the cases (52%). Mortality was encountered in 6 out of 114 patients (5%).

DISCUSSION

In 1936, Andre Lemierre published a series of 20 cases of throat infections with anaerobic septicemia, of whom 18 died.[1] Following the introduction of antibiotics in the 1940s, and its widespread use for streptococcal pharyngitis, the incidence of Lemierre's syndrome has fallen dramatically to a degree that it had been called the forgotten disease. Bartlett et al. were unable to identify a single case of Lemierre's syndrome in the 1950s and 1960s.[2] More recently though, there has been a rapid increase in the reporting of Lemierre's syndrome. A MEDLINE search, using the term Lemierre's syndrome as the sole key word, identified only six relevant articles between 1980 and 1990, 50 articles from 1991 to 2000, and 121 articles in the last 8 years (2001–2008).

Whether this represents a true increase in the incidence of this rare entity or just a literature/publishing trend remains to be seen. One could assume that this rapid increase may be due to increased antibiotic resistance or changes in antibiotic prescription patterns. Ramirez et al., in their well-designed pediatric review, speculated that the recent increase in the number of serious infections caused by *F. necrophorum* could be attributed to regional alterations in antibiotic usage patterns.[3] As awareness about antibiotic resistance is increasing, both in primary care and in the general population, family physicians are more reluctant in prescribing penicillin for uncomplicated sore throats, and parents are less demanding in requesting antibiotics for their children's pharyngitis. As a result, a few seemingly uncomplicated cases of sore throat will progress into fusobacteria infections. Additionally, shifting away in prescription practices from antibiotics requiring multiple doses to those requiring single or twice-daily dose uses, such as various second and third generation cephalosporins, has meant treating pharyngitis with antibiotics that often lack activity against fusobacteria.[3] Finally, higher resolution scanning means that internal jugular vein thrombosis is more easily diagnosed, and may provide the first and often the only clue for suspecting possible Lemierre's syndrome.

The present study confirms that Lemierre's syndrome is mainly a disease of previously healthy young adults, although it spares no age group. The pooled mortality rate was 5%, slightly lower than the previously reported rates of 6% to 22%.[84] *F. necrophorum* appears to be responsible for 10% of all acute sore throats and 21% of all recurring sore throats, with the remainder being caused by group A streptococci or viruses.[85] *F. necrophorum* is a Gram-negative non-spore forming obligate anaerobe, and although older resources have stated that *Fusobacterium* is a common occurrence in the human oropharynx, the current consensus is that *Fusobacterium* should always be treated as a pathogen. *F. necrophorum* is usually susceptible to penicillin, clindamycin, metronidazole, and chloramphenicol, but there is

TABLE II.
Usual First Clinical Presentation of Lemierre's Syndrome.

Presentation	%
Sore throat	33
Neck mass	23
Neck pain	20
Bone/joint pain	8
Otalgia and/or otorrhea	8
Dental pain	5
Orbital pain	1
Gastrointestinal symptoms	1
Limb weakness	1

TABLE III.
Case Reports and Series of Patients with Lemierre's Syndrome.

Study Reference No.	No. of Patients	Culture	Hospital Stay*
3	14	FN	
4	1	FN	2 w
5	3	FN	4–7 d
6	1	FN, *Staphylococcus epidermidis*	>10 d
7	1	FN	
8	3	FNu, FN	6 w
9	1	FN	13 d
10	1	FN	15 d
11	1	FN	4 w
12	1	FN	
13	1	FN	
14	1	FN	11 w
15	1	FN	
16	3	FN in all	9–14 d, 11 d, >2 w
17	1	FN	44 d
18	1	FN	6 w
19	1	FN	4 w
20	1	FN	4 w
21	1	FN	2 w
22	1	FN	3 w
23	1	FN	5 w
24	1	FN	
25	1	FN	3 w
26	1	NEG	2 w
27	1	FN	3 w
28	1	FN	
29	1	FN	8 w
30	1	FN	2.5 w
31	2	NEG	3 w
		FN	2 w (8th postoperative day)
		Peptostreptococcus, group C strep	
32	1	FN, group C strep	3 w
33	1	FN, group C strep	5 w
34	3	FN	6 w
		FN	5 w
		FN	7 w
35	1	FNu	
36	1	*Fusobacterium* sp	4 w
37	1	*Klebsiella pneumoniae*	6 w
38	1	*Peptostreptococcus anaerobius, Bacteroides fragilis, Eikenella corrodens*	3 w
39	1	*Staphylococcus haemolyticus, Staphylococcus hominis*	8 w
40	1	*Escherichia coli*, proteus, FN, *Peptostreptococcus anaerobius*	5 w
41	1	NEG	2 w
42	1	*Bacteroides uniformis, Proteus*, diptheroids, *Peptostreptococci*	2.5 w
43	1	FN	8 w

(*Continued*)

TABLE III.
(*Continued*).

Study Reference No.	No. of Patients	Culture	Hospital Stay*
44	1	NEG	2 w
45	1	NEG	
46	1		2–3 w
47	1	FN; *Staphylococcus epidermidis*	10 d
48	1	*Bacteroides melaninogenicus*	2 w
49	1	*Fusobacterium* sp	2 w
50	1	FN	6 w
51	2	FN	4 w
52	3	FN	2 w
		FN	1 w
		FN; *Staphylococcus epidermidis*	2 d
53	2	*Fusobacterium* sp	1–2 w
			4 w
54	2	FNu	3 w
		Fusobacterium sp	4 w
55	1	FN	2 w
56	1	FN	6 w
57	1	FN	3 w
58	1	FN	3–4 w
59	1	*Fusobacterium* sp, *Propionibacterium*	
60	1	*Fusobacterium* sp, *Bacteroides fragilis, Proteus, Peptostreptococcus*	8 d
61	1	FN	2 w
62	1	*Fusobacterium* sp	
63	1	FN	6 w
64	2	FN	3 w
65	1	FN	
66	1	*Fusobacterium* sp	>4 w
67	1	FN	
68	1	FN	
69	1	FN	7 w
70	1	FN	
71	1	FN	5 w
72	2	FN	2 w
73	1	FN	12–16 w
74	1	FN	10 d
75	1	FN	12 d
76	1	NEG	30 d
77	1	FN	23 d
78	1	FNu	4 w
79	1		4 w
80	1	*Streptococcus viridans*	3 w
81	1	*Fusobacterium* sp	16 d
82	1	FN	4 w
83	1	FN	6 w
84	1	NEG	4 w
85	1	FN	2 w
86	2	FN	4 w
		FN	3 w

*Hospital stay is expressed as days (d) or weeks (w).

Strep = *Streptococcus*; FN: *Fusobacterium necrophorum*; FNu = *Fusobacterium nucleatum*; NEG = negative; *Fusobacterium* sp = *Fusobacterium* species.

TABLE IV.
Case Reports and Series of Patients with Lemierre's Syndrome— Complications/Morbidity.

Study Reference No.	Complications/Morbidity
3	NC
4	Sigmoid sinus thrombosis, vocal cord palsy, XI nerve palsy
5	NC
6	Pansinusitis, mastoiditis, cavernous sinus thrombosis, CVA
7	Cerebral/spleen/kidney lesions
8	Acute cerebellar event, gastrointestinal bleed, empyema
9	NC
10	Mediastinitis
11	Cavernous sinus thrombosis, parapharyngeal abscess, temporal lobe infarct
12	Epidural & psoas abscess
13	Post mortem: pulmonary exudates
14	Suppurative hip arthritis, empyema
15	Anterior uveitis, endogenous ophthalmitis
16	Mastoid abscess, sigmoid sinus thrombosis, extradural collection, subdural collection, temporal bone osteomyelitis
17	Ascites, myocarditis, pericarditis, parapharyngeal abscess, bronchopleural fistula
18	NC
19	NC
20	Spondylodiscitis, gluteal abscess
21	NC
22	Vitreous hemorrhage, septic emboli
23	Osteomyelitis (tibia, humerus), hepato-splenomegaly, mastoiditis
24	NC
25	Transverse sinus thrombosis
26	NC
27	NC
28	ARDS
29	Obturator internus abscess
30	Splenic abscess
31	DIC, Hepato-, splenomegaly, thoracocentesis, surgical excision of involved IJV
32	NC
33	Sigmoid sinus thrombosis
34	NC
35	Discharged himself against medical advice on day 7
36	Sigmoid sinus thrombosis, superior ophthalmic vein elongation
37	NC
38	Sigmoid sinus thrombosis, superior ophthalmic vein elongation
39	ICA thrombosis, retrobulbar inflammatory mass
40	Submandibular abscess, hip dislocation, DIC
41	Subclavian vein thrombosis
42	Cholesteatoma, lateral sinus thrombosis
43	Piriform muscle abscess, osteomyelitis (ilium)
44	NC
45	Thrombosis of both IJVs
46	IJV and subclavian vein thrombosis
47	NC
48	SIADH, hemolytic anemia, transverse myelitis, and transient areflexia
49	DIC, lung abscess, hepato-, splenomegaly
50	N C
51	Metastatic abscess of humerus/wrist
52	Multiple trunk and arm pustular lesions
53	NC
54	Cavernous sinus thrombosis, ARDS
55	IJV thrombosis, carotid sheath abscess
56	Hepato-, splenomegaly
57	VI N palsy, mastoiditis, IJV, ICA, and cavernous sinus thrombosis
58	Mastoiditis, transverse and sigmoid sinus thrombosis, osteomyelitis (fibula)
59	Hepato-, splenomegaly
60	Hydropneumothorax, acute renal failure, DIC
61	NC
62	Infraspinatus muscle abscess
63	Osteomyelitis (fibula, knee)
64	NC
65	Empyema, parapharyngeal abscess, ARDS, shoulder septic arthritis, pneumothorax
66	NC
67	NC
68	Septic arthritis (hip)
69	Empyema, hemothorax, foot gangrene
70	NC
71	Parapharyngeal abscess, EJV thrombosis
72	Pulmonary consolidation, pneumothorax, ARDS
73	Gluteal abscess, hepato-, splenomegaly
74	NC
75	Hepato-, splenomegaly, ARDS
76	Peritonsillar abscess, ascites
77	NC
78	NC
79	NC
80	Parapharyngeal abscess
81	NC
82	Splenic lesions, gluteal abscess, pulmonary infarct
83	Hepatic abscess, acute renal failure
84	NC
85	Mastoiditis
86	Multiple pneumatoceles, inflammatory lesions shoulder/hip/knee, diffuse encephalopathy

NC = no complications; CVA = cerebrovascular accident; ARDS = adult respiratory distress syndrome; DIC = disseminated intravascular coagulation; IJV = internal jugular vein; ICA = internal carotid artery; SIADH = syndrome of inappropriate antidiuretic hormone; EJV = external jugular vein.

TABLE V.
Morbidity Encountered in Lemierre's Syndrome.

Morbidity	%
Brain	30
Septic arthritis/osteomyelitis	22
Lung	22
Deed neck space infections	14
Pericardial tamponade	7
Liver	6
Mastoiditis	6
Spleen	6
Eye	5
Lower cranial nerve palsies	3

Brain manifestations include meningitis, epidural/subdural abscess, cavernous/sigmoid/transverse/lateral sinus thrombosis, and stroke. Septic arthritis and osteomyelitis had been reported to involve the humerus, hip, clavicle, tibia, and fibula. Lung manifestations include mediastinitis, empyema, hydro/pneumothorax, and pneumonia. Deep neck space infections refer to parapharyngeal and retropharyngeal abscess. Liver and spleen manifestations include infarcts and abscesses. Eye manifestations include uveitis, vitreous hemorrhage, retrobulbar mass, and VI nerve palsy. Lower cranial nerve palsies refer to XI–XII nerve palsies.

a various response to second- and third-generation cephalosporins. Additionally, penicillin treatment failures due to β- lactamase production of the infecting microorganism have also been reported, especially by *F. nucleatum* and *F. necrophorum*.[86] Most microbiologists would recommend β-lactamase-resistant antibiotics with anaerobic activity, such as metronidazole, clindamycin, and tazocin.[87]

The role of anticoagulation in treating Lemierre's syndrome remains controversial. In a recent review of Lemierre's syndrome, and a review of otology, obstetrics, gynecology, and internal medicine literature looking at both anticoagulation and long-term antibiotic treatment for septic thrombosis, Bondy et al. concluded that, although anticoagulation is commonly used in other specialties for similar septic emboli, its role in Lemierre's syndrome is unclear.[88] The risks and benefits of anticoagulation therapy for internal jugular vein thrombophlebitis have not been properly assessed in controlled studies, as the low incidence of Lemierre's syndrome has not made it possible to set up clinical trials to study the disease. When jugular vein thrombosis occurs, there is inflammation and consequent septic thrombophlebitis, which gives rise to distant emboli that usually migrate to pulmonary capillaries.[89] As a consequence, the most frequently involved site of septic metastases are the lungs, followed by the joints (knee, hip, sternoclavicular joint, shoulder, and elbow).[90] Other sites involved in septic metastasis and abscess formation are the muscles and soft tissues, liver, spleen, kidneys, and central nervous system.[89] Production of bacterial toxins, such as lipopolysaccharide, leads to secretion of cytokines by white blood cells, which then leads to symptoms of sepsis. *F. necrophorum* produces hemagglutinin, which causes platelet aggregation that can lead to diffuse intravascular coagulation and thrombocytopenia.[90,91]

Management of Lemierre's syndrome is often surgical (i.e., draining a neck abscess, intercostal drainage of a pneumothorax), in combination with aggressive intravenous antibiotics based on microbiologists' advice, and targeting fusobacteria when appropriate.[92] Anticoagulation therapy is based more on personal experience and preference and/or departmental protocols rather that robust evidence.

CONCLUSION

There appears to be an increase in Lemierre's syndrome cases, perhaps due to antibiotic resistance or changes in antibiotic prescription patterns. Successful management rests on the awareness of the condition, a high index of suspicion, and a multidisciplinary team approach.

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