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Main Article

Lemierre's and Lemierre's-like syndromes in association with infectious mononucleosis

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Abstract

Objective: This study aimed to review cases of Lemierre's and Lemierre's-like syndromes in paediatric patients, to examine a possible association with Epstein–Barr virus as a predisposing factor, and to assess the impact of this virus on the severity of illness.

Methods: We performed a retrospective analysis of data from the in-patient database at Winthrop University Hospital, from January 2001 to October 2007. We reviewed clinical and laboratory findings as well as the outcome of infection in patients aged 21 years or less with a diagnosis of Lemierre's syndrome. An additional case of Lemierre's-like syndrome was also included. The illness severity and duration of in-patient management of those testing positive for heterophile antibody were then compared with the same parameters in patients who tested negative.

Results: Of the five patients diagnosed with Lemierre's syndrome, two had concomitant acute infection with Epstein–Barr virus. Additionally, a 19-year-old adolescent was admitted during this period with acute infectious mononucleosis, *Fusobacterium necrophorum* sepsis, sinusitis, frontal lobe abscess and ophthalmic vein thrombosis. The clinical presentation of all patients included fever, sore throat, and ear or neck pain. The duration of symptoms ranged from two days to three weeks prior to admission. The patients with acute Epstein–Barr virus infection had been diagnosed with infectious mononucleosis prior to admission, and tested positive for heterophile antibody. These patients subsequently underwent more extensive in-patient treatment, including intensive care management and ventilator support. The patients who tested negative for heterophile antibody experienced a milder course of illness, with a shorter duration of in-patient management.

Conclusion: Two patients diagnosed with Lemierre's syndrome, and a third with *Fusobacterium necrophorum* sepsis, had coexisting acute Epstein–Barr virus infection. Patients who tested positive for heterophile antibody experienced a more severe course of illness. These observations suggest a possible association between Epstein–Barr virus infection and the severity of concomitant Lemierre's syndrome.

Key words: Fusobacterium Necrophorum; Infectious Mononucleosis; Lemierre's Syndrome

Introduction

Lemierre's syndrome (also termed postanginal sepsis and necrobacillosis) is a rare condition consisting of suppurative infection of the lateral pharyngeal space accompanied by septic jugular thrombophlebitis. The syndrome was first reported in 1936,[1] when the French bacteriologist and infectious disease physician Andre Lemierre (1875–1956) described in *The Lancet* 20 cases of anaerobic thrombophlebitis of the internal jugular vein with metastatic infection.

The condition is usually caused by an anaerobic, Gram-negative bacillus, *Fusobacterium necrophorum*. Other causative organisms reported include other fusobacterium species, streptococcal species, staphylococci, *Bacteroides melaninogenicus*, *Eikenella corrodens* and peptostreptococcus.[2] The initial symptoms are usually nonspecific and include sore throat, fever, rigor and lateral neck tenderness. The disease usually begins as pharyngitis or tonsillitis. Subsequently, septic clots can dislodge from an internal jugular vein thrombus, causing pulmonary infarcts. Other common complications include sepsis, jaundice, abnormal liver function, pleural effusion and empyema. In addition, septic arthritis, meningitis, endocarditis or soft tissue infection can result from haematogenous seeding. The infection responds to antibiotic therapy with β-lactamase-resistant compounds that exert adequate anaerobic coverage. Anticoagulation and surgical intervention may be helpful in advanced cases.

In recent years, an increasing number of case reports of Lemierre's syndrome in children have been published. These publications[3,4] have focused

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on the clinical and laboratory findings at presentation, and the outcomes of infection.

Epstein–Barr virus (EBV), or human herpes virus 4, is a γ-herpes virus which occurs worldwide. By the age of five years, approximately 50 per cent of the US population has been infected.[5] Epstein–Barr virus infection acquired during adolescence is either asymptomatic or associated with acute infectious mononucleosis. Approximately 90 per cent of the US population has been affected with EBV by 25 years of age. The virus occurs with the same frequency and symptomatology in the developed nations of the world as it does in the US.[5]

The clinical diagnosis of infectious mononucleosis is suggested on the basis of the symptoms of fever, sore throat, pharyngitis and lymphadenopathy, and the patient's age. The three classic criteria for laboratory confirmation of acute infectious mononucleosis are (1) lymphocytosis, (2) the presence of at least 10 per cent atypical lymphocytes on a peripheral blood smear, and (3) a positive serological test for heterophile antibody.[5,6]

The National Center for Infectious Disease (at the Centers for Disease Control and Prevention) has described the laboratory diagnosis of infectious mononucleosis, which can be made based on a single acute-phase serum sample.[6] This is done by testing for antibodies to several EBV-associated antigens. Serological tests for EBV are useful for evaluating patients who have heterophile-negative infectious mononucleosis. These tests can determine whether the patient has had a recent EBV infection, is susceptible to EBV, has had a past infection, or has reactivated EBV infection.

It is possible to measure antibodies to several antigen complexes. These include the viral capsid antigen, the early antigen and the EBV nuclear antigen. Immunoglobulin (Ig) G and M subclasses to the viral capsid antigen can also aid confirmation. When the heterophile antibody test is negative, the optimal combination of EBV serological testing consists of the antibody titration of four markers: IgM and IgG to viral capsid antigen, IgM to early antigen, and antibody to EBV nuclear antigen.

The diagnosis of EBV infection is summarised as follows. A patient is susceptible to EBV infection if antibodies to viral capsid antigen are not detected. Primary EBV infection is indicated by the presence of IgM antibody to the viral capsid antigen and the absence of antibody to EBV nuclear antigen. Also suggestive of primary infection is a high titre of IgG antibody to viral capsid antigen and no antibody to EBV nuclear antigen after at least four weeks of illness. Antibody to early antigen is also seen in 80 per cent of patients with active EBV infection. Past EBV infection (i.e. ranging from four to six months earlier to several years earlier) is indicated by the presence of antibodies to both viral capsid antigen and EBV nuclear antigen. Reactivation is suggested by elevated titres of antibodies to early antigen in the presence of antibodies to EBV nuclear antigen. A number of healthy, asymptomatic people can have antibodies to early antigen for years after their initial EBV infection. It is reported that reactivation can often occur subclinically.[6]

Evidence has been published supporting a role for EBV infection either as a predisposing element or a risk factor for the manifestation of Lemierre's syndrome or Lemierre's-like syndrome. Riordan and Wilson[7] suggested mechanisms by which EBV could predispose to Lemierre's syndrome. They reported several cases of Lemierre's syndrome in patients with serological evidence of recent EBV infection, which may have induced immunosuppression via a transient decrease in T-cell mediated immunity, facilitating secondary bacterial infection. They described a pathogenic mechanism whereby the inflammatory response to *F necrophorum* infection is dependent on the production of endotoxin and extracellular leucocidin. Riordan and Wilson also reported that *F necrophorum* aggregates human platelets, and that the resulting intravascular coagulation may contribute to an anaerobic environment. They believed that this was a key virulence factor in generating septic thrombophlebitis in the tonsillar veins, which then propagates to involve the internal jugular vein. Subsequently, septic emboli can disseminate to various sites to cause metastatic abscesses. Riordan[8] has proposed several other mechanisms by which infectious mononucleosis may predispose to invasive *F necrophorum* infection. Potential factors reported include lymphatic obstruction, impairment of leukocyte migration, and defective Ig production secondary to T-cell suppression of B-cells.[8]

In the current study, we reviewed five paediatric cases of Lemierre's syndrome managed at a university hospital over a six-year period. A sixth patient with clinical features of Lemierre's syndrome and radiographic evidence of jugular venous thrombosis was also included in this study. We present our observations on the course of these six patients, and the association of more severe illness with concomitant EBV infection.

Methods

We reviewed the paediatric in-patient database at Winthrop University Hospital to identify patients diagnosed with Lemierre's syndrome. The data reported in this study are based on hospital admissions of patients diagnosed with infectious mononucleosis, thrombophlebitis or Lemierre's syndrome. We conducted a retrospective medical chart review using the International Classification of Diseases nine code 451.89, designated for phlebitis and thrombophlebitis of other sites. A thorough review of patients' primary and secondary diagnoses excluded non-jugular vein thrombophlebitis. We also reviewed medical charts with an International Classification of Diseases nine coding of 075, designated for infectious mononucleosis.

Our inclusion criteria comprised children aged zero to 21 years who had received a diagnosis of jugular venous thrombosis or Lemierre's syndrome at Winthrop University Hospital (Mineola, New York) between January 2001 and October 2007. Designation

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of a case as Lemierre's syndrome required all of the following: (1) clinical diagnosis of oropharyngeal infection or other febrile illness; (2) radiographic or clinical evidence of jugular venous thrombosis; (3) isolation of a causative organism by microbiological culture of blood, tissue and/or purulent fluid, in patients who had not been treated with antibiotics before culture; and (4) at least one distal focus of infection (e.g. pneumonia or arthritis).[4]

We identified a total of five patients admitted and treated at a single university hospital for Lemierre's syndrome. A case of Lemierre's-like syndrome (subject three) was also included in the study, based on isolation of *F necrophorum* from blood and epidural abscess material, in association with ophthalmic vein thrombophlebitis.

We further analysed the clinical presentation and laboratory findings of these six patients. Heterophile monospot screening had been performed on subjects one, two and three, prior to hospital admission. In addition, Epstein–Barr virus (EBV) serology testing had been performed on subjects one and three, both prior to hospital admission. None of the patients had received Ig or blood products prior to serology assessment. Three of the six patients had coexisting acute EBV infection, as suggested by either positive heterophile antibody testing, positive EBV serology or both.

Results

We identified a total of five adolescents with a diagnosis of Lemierre's syndrome, plus one adolescent with clinical features of Lemierre's syndrome, all of whom had been admitted to the in-patient unit. Two of the five patients with diagnosed Lemierre's syndrome, and a third with *F necrophorum* sepsis, had laboratory evidence of acute, simultaneous Epstein–Barr virus (EBV) infection. In-patient data were collected as shown in Table I.

In the first subject studied, tests for heterophile antibody, EBV capsid antibody IgM and EBV capsid antibody IgG were positive. In the second subject with Lemierre's syndrome, the heterophile mononucleosis screen test was positive. This subject had a white blood cell (WBC) count of 11.5×10^3/μl and a differential with 15 per cent atypical lymphocytes. Epstein–Barr virus titres were not tested in this second subject. In the third subject studied, tests for heterophile antibody, EBV capsid antibody IgM and EBV capsid antibody IgG were positive. Three days prior to admission, this subject had had an out-patient WBC count of 12.1×10^3/μl and a differential with 10 per cent atypical lymphocytes. The remaining three subjects tested negative for acute EBV infection. According to the American Academy of Pediatrics *Red Book*, diagnosis of acute EBV infection is supported by a finding of >10 per cent atypical lymphocytes in combination with a positive heterophile antibody test.[9]

Table I details all six patients' clinical course, laboratory and radiographic findings, and complications.

Patients with acute EBV infection required paediatric intensive care unit admission and ventilator support. Subject one underwent video-assisted thoracic surgery for a loculated pleural effusion, and required 24 days' hospital admission 10 days of which were spent in the paediatric intensive care unit. Subject two required chest tube placement together with thrombolytic and vasopressor support, and had a 28 day hospital stay with 15 days in the paediatric intensive care unit. Subject three required a frontal craniotomy and endoscopic sinus surgery with drainage, and had a 22 day hospital stay with 8 days in the paediatric intensive care unit. Subjects one and two received anticoagulants. Subject three did not receive anticoagulants due to the risk of post-operative bleeding.

The remaining three subjects (four, five and six), who tested negative for heterophile antibody, had a milder course of illness. Subjects five and six spent three to four days in the paediatric intensive care unit, without the need for ventilator support. Subject four had a one week hospital stay as a standard in-patient. Subjects four and five received anticoagulants, while subject six did not.

All the subjects clinically presented with a history of sore throat and fever for a period ranging from two days to three weeks prior to admission. Infectious mononucleosis was clinically diagnosed one month, six days and three days prior to admission in subjects one, two and three, respectively. Subjects two and three also received an out-patient course of corticosteroids (5–7 days of prednisone prior to hospital admission).

Five of the six patients (subjects one, two, three, four and six) had blood cultures positive for anaerobic Gram-negative rods, identified as *F necrophorum*. Subject five, who had clinical features of Lemierre's syndrome, had a negative blood culture, presumably as a result of previous intravenous antibiotic therapy for painful cervical swelling and pneumonia. However, this patient did have a Doppler study finding of jugular venous thrombosis.

Regarding distal foci of infection, five of the six patients had pulmonary involvement while one subject had renal failure. Subject three also exhibited ophthalmic vein thrombosis.

Discussion

These study findings indicate that patients who develop Lemierre's syndrome in the presence of acute Epstein–Barr virus (EBV) infection may suffer a more severe course of illness.

There is increasing evidence supporting a link between EBV infection and Lemierre's syndrome. In 1983, Adams *et al.*[10] described the first case with a possible link between these two entities: a 16-year-old adolescent diagnosed with infectious mononucleosis, confirmed by a positive heterophile antibody test five weeks prior to onset of illness, who developed Lemierre's syndrome. Since then, Riordan has described 23 cases in which infectious mononucleosis was linked to Lemierre's syndrome or invasive *F necrophorum* infection.[8]

Venglarcik *et al.*[11] had previously reviewed the literature and noted that EBV had been reported in association with fusobacterium infections. These

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TABLE I

IN-PATIENT DATA

Pt no/age (y)/sex	BC isolate	Htphl Ab	EBV titre	Atyp lymphocytes (%)	Distal infection	Hospital stay	
						Duration	Clinical course
1/19/M	*F necrophorum*	+ve	EBV capsid Ab IgM +ve EBV capsid Ab IgG +ve EBNA Ab IgG slightly +ve	0	Pulmonary	24 days (PICU 10 days)	Intubation (8 days), VATS, chest tube, anticoagulants, antibiotics Complications: loculated pleural effusion, hepatosplenomegaly, decubitus ulcer Chest CT: loculated right pleural effusion, RML infiltrate & atelectasis
2/17/F	*F necrophorum*	+ ve	Not tested	15	Pulmonary	28 days (PICU 15 days)	Intubation (7 days), chest tube, vasopressors, anticoagulants, thrombolytics, PPN, antibiotics, prednisone Complications: pleural effusions, myocarditis, CHF, IJV thrombosis Radiology: chest CT – R lung loculations, bilateral effusions; neck CT – R IJV branch with 2–3 cm clot
3/19/M	*F necrophorum*	+ve	EBV capsid Ab IgM +ve EBV capsid Ab IgG +ve	10	Pulmonary Ophthalmic	22 days (PICU 8 days)	Intubation (3 days), endoscopic sinus surgery & drainage, frontal craniotomy, antibiotics, prednisone Complications: bibasal airspace disease, atelectasis, ophthalmic v thrombosis, frontal lobe empyema, pan-sinusitis Radiology: head CT – left superior ophthalmic v thrombosis, frontal lobe empyema; chest X-ray – bilateral bibasal infiltrates, atelectasis
4/18/M	*F necrophorum*	–ve	EBV IgM –ve EBV IgG +ve EBV early Ag +ve EBNA Ag +ve	0	Pulmonary	7 days (PICU 0 days)	Anticoagulants, antibiotics Complications: IJV thrombosis, pleural effusions, cavitations Radiology: neck CT – R IJV thrombosis; chest X-ray – bilateral pleural effusions & cavitations
5/17/F	No growth	–ve	–ve	0	Pulmonary	10 days (PICU 3 days)	Thoracentesis, anticoagulants, antibiotics Complications: pneumonia, cervical adenitis, pleural effusions, thrombosed venous channels, cholecystitis Radiology: neck CT – EJV thrombosis; chest X-ray – bilateral pleural effusions
6/13/M	*F necrophorum*	–ve	Not tested	0	Renal	9 days (PICU 4 days)	Antibiotics, vasopressors Complications: pleural effusion, borderline splenomegaly, septic shock, acute renal failure Chest X-ray: pleural effusion

Pt no = patient number; y = years; BC = blood culture; htphl ab = heterophile antibody; EBV = Epstein–Barr virus; atyp = atypical; M = male; F = female; +ve = positive; –ve = negative; Ig = immunoglobulin; EBNA = EBV nuclear antigen; PICU = paediatric intensive care unit; VATS = video-assisted thoracoscopic surgery; CT = computed tomography; RML = right middle lobe; PPN = peripheral parenteral nutrition; CHF = congestive heart failure; IJV = internal jugular vein; R = right; v = vein; Ag = antigen; EJV = external jugular v

authors suggested that EBV-related pharyngitis was responsible for the production of an environment favourable to the growth of *F necrophorum*. They suggested that the invasiveness of *F necrophorum* could be explained by the production of proteolytic enzymes, endotoxins, leukocidin and haemagglutinin. They further stated that disruption of the oropharyngeal mucosal barrier could lead to hypoxia and tissue destruction, creating the oxygen-free environment necessary for anaerobic bacteria proliferation.

The literature suggests that *F necrophorum* may be found in such anaerobic environments, among other pathogens. Riordan[8] have proposed that synergy between *F necrophorum* and other anaerobic or microaerophilic organisms may lower the oxygen concentration, which can aid bacterial growth within an abscess. Huits *et al.*[12] have reported disseminated anaerobic sepsis followed primary EBV infection. Boz *et al.*[13] reported a case of Lemierre's syndrome developing after EBV infection, and suspected EBV as a predisposing factor. Bliss *et al.*[14] reported that antecedent EBV infection may promote fusobacterium species invasion, possibly by inducing immunosuppression as well as pharyngeal inflammation.

Our investigation, although limited to a small sample size, suggests a correlation between acute EBV infection and more severe Lemierre's syndrome.

Evidence of distal infection indicates that Lemierre's syndrome is frequently complicated by sepsis and septic metastasis, which can affect the lungs, musculoskeletal system, skin, spleen, kidneys, liver and meninges.[15] Chapman and Tully[16] have described a similar case involving metastatic abscess formation.

Our investigation further suggests that patients with Lemierre's or Lemierre's-like syndrome who test positive for heterophile antibody suffer a more serious course of illness. Our subjects one and two had Lemierre's syndrome and were also positive for heterophile antibody, and required paediatric intensive care unit admission together with intubation, chest tubes, anticoagulant therapy and antibiotics. Subject one also required video-assisted thoracic surgery, while subject two also required vasopressor support, thrombolytics and peripheral parenteral nutrition. These two patients had a hospital stay of between 24 and 28 days, with 10–15 days in the paediatric intensive care unit. Subject three suffered Lemierre's-like syndrome and also tested positive for heterophile antibody; this patient was admitted to the paediatric intensive care unit for eight days and required intubation and antibiotics.

Our three patients who tested negative for heterophile antibody (subjects four, five and six) experienced a milder course of illness, without the need for ventilator support. Although two of these three patients were briefly admitted to the paediatric intensive care unit, their course of illness required less intensive management and their duration of hospital stay was markedly shorter.

Two of our three patients with a severe course of illness (subjects two and three) also received a course of corticosteroids. These two subjects had been diagnosed with infectious mononucleosis and had received a short course (5–7 days) of prednisone as out-patients, before the clinical deterioration that prompted their admission to hospital. Matten and Grecu[4] have suggested that the use of glucocorticoids for symptom relief may predispose patients to bacterial superinfection. Of Riordan's 23 patients with Lemierre's syndrome or *F necrophorum* infection associated with infectious mononucleosis, seven (30 per cent) were reported to have received glucocorticoid therapy.[8]

A larger sample size collected from various institutions would be necessary to confirm our observation that Lemierre's syndrome is more severe in the presence of acute infectious mononucleosis.

- **Lemierre's syndrome is a rare but distinctive illness characterised by severe sore throat, suppurative infection of the lateral pharyngeal space, and jugular vein septic thrombophlebitis with embolic spread to the lungs, joints, spleen and kidneys**
- **The condition is most commonly caused by disseminated *Fusobacterium necrophorum* infection**
- **Published evidence supports a role for Epstein–Barr virus (EBV) infection as either a predisposing element or a risk factor for Lemierre's and Lemierre's-like syndromes**
- **The reported case series suggests a correlation between acute EBV infection and more severe Lemierre's syndrome**

Currently, Lemierre's syndrome is a rare condition affecting one in a million.[17] However, Dalal *et al.*[18] have reported an increased incidence in recent years. Most cases of Lemierre's syndrome typically occur in healthy adolescents and young adults. It is important that clinicians suspect Lemierre's syndrome in previously healthy patients with a recent history or evidence of oropharyngeal infection who are especially ill. It is important to be aware of the changing epidemiology of this syndrome, and the potential link between severe manifestations and concomitant EBV infection. The current study suggests the occurrence of EBV co-infection as a potential predisposing factor for more severe Lemierre's syndrome. This potential association should remind clinicians to maintain a high index of clinical suspicion in patients with EBV infection.

Conclusions

Lemierre's syndrome is a rare but serious complication of pharyngeal infections accompanied by septic jugular thrombophlebitis. Lemierre's and Lemierre's-like syndrome have been previously reported in association with infectious mononucleosis. Our investigation further suggests that subjects who tested positive for heterophile antibody experienced a more serious illness. These observations suggest a possible correlation between EBV infection

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and the severity of concomitant Lemierre's Syndrome. It is crucial that clinicians recognize this potential for severe course of illness in patients with Lemierre's syndrome in the face of acute infectious mononucleosis.

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