The Royal College of Surgeons of England

Ann R Coll Surg Engl 2003; **85**: 242–244

Case report

Serious consequences of a sore throat

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Lemierre's syndrome, caused by *Fusobacterium necrophorum*, is a potentially fatal sequelae of a sore throat characterised by septicaemia, internal jugular vein thrombophlebitis and metastatic abscesses. The Chief Medical Officer reported in February 2001 that the incidence is increasing. Two cases seen in one year, with different presentations, are reported. The first patient presented with sepsis, jaundice, hepatic abscesses and portal vein/superior mesenteric vein thrombosis, whilst the second presented with sepsis, sore throat and internal jugular vein thrombophlebitis. Both patients were treated with antibiotics and anticoagulants with a favourable outcome.

Key words: Lemierre's syndrome – Portal vein thrombosis – Internal jugular vein thrombosis – *Fusobacterium necrophorum*

In 1936, André Lemierre reported in *The Lancet* 20 cases of anaerobic septicaemia caused by an organism now known as *Fusobacterium necrophorum*.[1] These cases shared similar features, namely septicaemia in young adults preceded by a sore throat, with internal jugular vein thrombophlebitis and metastatic abscesses which, when present, are now referred to as Lemierre's syndrome. Sixty-five years later, after a significant decline in the incidence of severe illness caused by this organism (following the introduction of antibiotics), the Chief Medical Officer in his February 2001 Update brought this organism to our attention and reminded us of both the increasing incidence and potential severity of infection.[2]

Case reports

Case 1

A previously healthy 19-year-old girl was admitted to hospital with abdominal pain, pyrexia, jaundice and 11 kg-weight loss. Initial blood tests revealed a leukocytosis of 18.6 x 10^9/l, a C-reactive protein of 222 mg/l and abnormal liver function tests (bilirubin, 98 µmol/l; alkaline phosphatase, 331 IU/l; alanine aminotransferase, 52 IU/l). Chest radiography revealed bilateral pleural effusions. Abdominal ultrasound scan (USS) showed both portal vein and superior mesenteric venous thrombosis as well as hepatosplenomegaly. Computerised tomography (CT) scan confirmed this, and additionally revealed multiple hepatic abscesses, the largest measuring 4.5 cm in diameter (Figs 1&2).

Two months previously, she had undergone a minor gynaecological procedure, followed by a 1-week course of doxycycline. One month later, having had a normal menstrual period, she presented to her GP with a cough, sore throat and intermittent vaginal discharge. A 1-week course of amoxycillin had been prescribed.

A drain was inserted percutaneously under CT guidance into the largest hepatic abscess cavity and 50 ml of pus was aspirated. *F. necrophorum* was isolated, by the reference laboratory, from the blood cultures and later confirmed by the abscess aspirate. Other investigations

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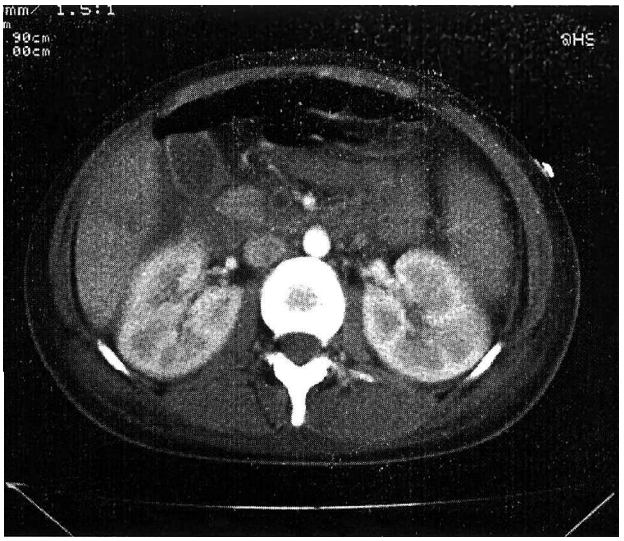

Figure 1 Computed tomography scan demonstrating thrombus within the superior mesenteric vein.

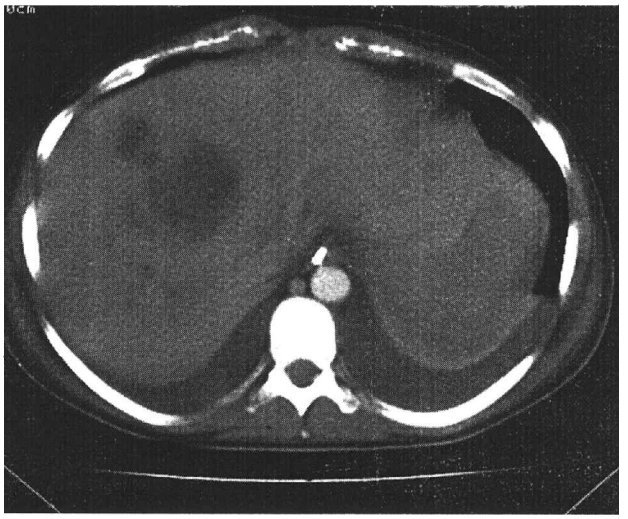

Figure 2 Large hepatic abscess with smaller associated abscesses are seen on this computed tomography scan.

included a positive Paul Bunnell test confirming infectious mononucleosis and positive serology for influenza B.

Antibiotic therapy included intravenous benzylpenicillin 2.4 g qds, metronidazole 500 mg tds and ciprofloxacin 400 mg bd. Full anticoagulation with intravenous heparin was used to maintain an activated partial thromboplastin ratio of 2.0–2.5.

The abscess drain was removed at 3 weeks and the patient discharged home at 4.5 weeks with a remaining 2-week course of antibiotics and long-term warfarin. Despite improved appearances on USS at 7 weeks, there was still evidence of portal hypertension and numerous collaterals replacing the portal vein.

Case 2

A previously healthy 34-year-old man presented with a 5-day history of fever, rigors, myalgia, neck pain and sore throat. He had been unable to swallow fluids for 5 days and had suffered with 48 h of watery diarrhoea. Initial examination revealed signs of severe dehydration, normal tonsils and an erythematous inflamed pharynx. There was tenderness on the right side of the neck but no cervical lymphadenopathy. Initial tests revealed a leukocytosis of 21 x 10^9/l, platelets of 76 x 10^9/l, C-reactive protein of 325 mg/l, urea 59 mmol/l and creatinine 377 µmol/l. Nasendoscopy demonstrated mild generalised swelling in the right lateral pharyngeal wall with no evidence of retropharyngeal swelling on plain lateral neck X-ray. Bilateral consolidation was present on chest X-ray.

He failed to improve after 36 h and was transferred to the high dependency unit for monitoring. His platelet count had decreased to 47 x 10^9/l, with an international normalised ratio (INR) of 1.7 and normal fibrinogen.

CT scan performed the following day demonstrated a multiloculated right parapharyngeal abscess (maximum diameter 1.5 cm) and a thrombosed right internal jugular vein (Fig. 3). He was, therefore, fully anticoagulated with intravenous heparin and managed conservatively. Repeat scan at 48 h showed some resolution of the abscess cavities and an ultrasound scan of the chest and abdomen revealed bilateral pleural effusions and a patent inferior vena cava.

F. necrophorum was isolated on blood cultures at 4 days. He was thus treated with intravenous benzylpenicillin 1.2 g qds, metronidazole 500 mg tds, cefotaxime 1 g tds and

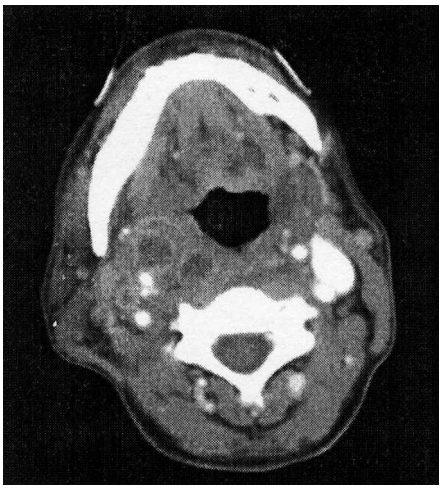

Figure 3 Right parapharyngeal abscess with associated internal jugular vein thrombosis seen on this computed tomography scan.

erythromycin 500 mg qds. Paul Bunnell test was negative and the throat swab revealed no growth.

The patient was discharged after 12 days, with a remaining 3-week course of oral amoxycillin and metronidazole as well as long-term warfarin.

Discussion

Lemierre's syndrome, which accounts for approximately 50% of cases of human *F. necrophorum* infection (necrobacillosis),[3] classically affects young adults, presenting 3–10 days following pharyngotonsillitis, with often life-threatening septicaemia, lateral neck swelling and tenderness, due to internal jugular vein thrombophlebitis, and metastatic septic emboli. These emboli may then cause abscess formation, most commonly in the lungs (up to 85%), but also in joints, bone, muscle, soft tissues, brain, meninges, liver, spleen, kidney, pericardium and endocardium.[4] Hepatic abscesses have been rarely described secondary to *F. necrophorum* septicaemia and where present are often solitary rather than multiple.[5–8]

High-dose intravenous antibiotics for 2–6 weeks form the mainstay of treatment. *F. necrophorum* (a Gram-negative anaerobic organism often mistaken for *Bacteroides* and which commonly can only be isolated in specialist reference laboratories) is normally sensitive to penicillin G. This has been used with success in many of the reported cases, although some resistant strains may now exist.[9] Penicillin G is commonly used in conjunction with other anti-anaerobic antibiotics (*e.g.* metronidazole, clindamycin, and chloramphenicol).[10,11] Any metastatic purulent collections (*e.g.* hepatic, splenic, pulmonary) associated with *F. necrophorum* septicaemia should, where possible, be drained.[12,13]

Anticoagulation in venous thrombophlebitis caused by *F. necrophorum* has been used with varying success,[12,14,15] and there remains no conclusive evidence to support its use in all cases. The decision to initiate such treatment should, therefore, be a clinical decision, based on both the severity of the clinical signs and the degree to which the internal jugular vein or abdominopelvic veins are involved.

These two cases illustrate the varied presentation of this illness and highlight its severity. The recent increase in Lemierre's syndrome is likely to be due to reduced antibiotic prescribing for sore throats. However, since serious *F. necrophorum* infection remains relatively uncommon and the risk of increasing antibiotic resistance exists, it is difficult to advocate change to the current advice regarding the use of antibiotic therapy in sore throat.

Conclusions

In any previously healthy young adult who, following a sore throat, develops signs of septicaemia, Lemierre's syndrome due to *F. necrophorum* infection must be suspected.

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