''My back is killing me''

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Emerg Med J 2005;**22**:824–825. doi: 10.1136/emj.2003.008177

This is a case of a rare cause of back pain that presented to several clinicians before the diagnosis became obvious and the correct management was initiated. Lemierre's syndrome was confirmed at post mortem examination. We conclude that thoracic back pain should not be assumed to have a simple mechanical cause and that efforts should be made to discover the underlying aetiology.

A 30 year old white woman presented to a GP complaining of thoracic back pain. The pain had been present from waking the previous day and had not resolved with ibuprofen. She had recently been coryzal but had no other past medical history of note. On examination, the GP found bilateral paravertebral muscle spasm in the mid to lower thoracic region, for which she was prescribed diclofenac.

The following day the patient presented to the Chelsea and Westminster Hospital, complaining that her back pain had worsened. She reported that it was exacerbated by movement and deep inspiration. Further history revealed that she had suffered from a severe sore throat and a dry cough accompanied by sweating a week previously. On examination, it was noted that she had difficulty standing and was tender adjacent to L2. A neurological examination was performed, which was normal, and she was discharged with diazepam and paracetamol. The respiratory system and neck were not examined, no baseline observations were recorded, and no investigations were performed.

The next morning the patient was seen by a physiotherapist who felt the pain was not mechanical and referred her back to the accident and emergency (A&E) department. As she booked back into A&E she appeared pale, so was put on a trolley to lie down. She immediately felt much better, her colour returned to normal, and she returned to a chair in the minor injury waiting room. She declined analgesia, was triaged as standard, and waited to be seen by a doctor. Approximately 5 hours later (22.50) it was noticed that she had become pale and uncomfortable. The first set of observations were performed (temperature 37°C, heart rate (HR) 132 beats/min, blood pressure (BP) 96/58 mmHg, respiration rate (RR) 20 breaths/min, oxygen saturation 85% on air), the emergency medicine middle grade and senior house officer were called, and the patient was moved into the resuscitation room.

Table 1 Arterial blood gases

	On air, at 23.07	On 60% O_2 at 01.13
pH	7.298	7.17
pCO_2 (kPa)	5.49	6.77
pO_2 (kPa)	5.75	10.73a
HCO_3 (mmol/l)	18.9	16.3
BE (mmol/l)	−6.7	−10.3

Further history revealed that the patient had developed nausea, diarrhoea, and a cough productive of green sputum overnight, and she had been successfully treated for cervical malignancy 10 years previously.

On examination, there was a clear airway, with nothing abnormal detected in the oropharynx/neck. High flow oxygen was applied via a Hudson mask. RR was 26 breaths/min, trachea central, and percussion was dull at both bases. There was reduced air entry bilaterally with crepitations throughout both lungs and oxygen saturation of 97% on 15 litres of oxygen. HR was 132 beats/min, BP 96/58 mmHg, jugular venous pressure unseen, and she had normal heart sounds. An intravenous infusion of 0.9% saline was started. The patient was drowsy but has Glasgow Coma Score of 5/15, blood glucose 5.5 mmol/l. The abdomen was distended but soft, with mild tenderness centrally but no signs of peritonism. There was bilateral leg swelling to the knees with a slightly tender left calf, which felt hot and tense. There was no rash and otherwise the examination was normal.

A working diagnosis of pneumonia was made and antibiotics (cefuroxime and erythromycin) were immediately administered.

ECG showed sinus tachycardia. Arterial blood gases are shown in table 1. Chest *x* ray showed a left sided pleural effusion, multifocal consolidation (most marked in the lingula and right base), and patchy infiltrate in the right upper lobe, and the left hilum appeared enlarged (fig 1). Full blood count showed haemoglobin 134 g/l, white blood cells 9×10^9/l, platelets 34×10^9/l. Urine and electrolyte analysis showed sodium 33 mmol/l, potassium 3.7 mmol/l, urea 33.3 mmol/l, and creatinine 432 μmol/l, while liver function tests were: bilirubin 31 μmol/l, albumin 20 g/l, otherwise normal. Prothrombin time was13.4 seconds, activated partial thromboplastin time 354 seconds, thrombin time 13 seconds, and urine was positive for blood and protein.

The patient was reviewed by the medical team and transferred directly to the intensive thereapy unit. Despite maximum therapy with crystalloids, colloids, and platelets, she continued to deteriorate. At 04.30 she was intubated and ventilated, and inotropes were started. Unfortunately, she died later that morning at 10.38.

Blood cultures later grew *Fusobacterium necrophorum*. Postmortem revealed fibrinopurulent exudates in the pleural cavities and multiple necrotic areas. Microscopy revealed numerous microabscesses. There was no evidence of deep venous thrombosis or pulmonary thromboembolism. A diagnosis of Lemierre's syndrome was made.

DISCUSSION

Lemierre was a Professor of Microbiology in Paris who described a group of 20 patients with very similar symptoms to our patient, who developed ''postanginal sepsis''.[1] The group consisted of young adults who had suffered from an infection that spread to cause local thrombophlebitis and distant septic emboli. The main causative organism was *F necrophorum*.

Abbreviations: A&E, accident and emergency; BP, blood pressure; HR, heart rate; RR, respiration rate

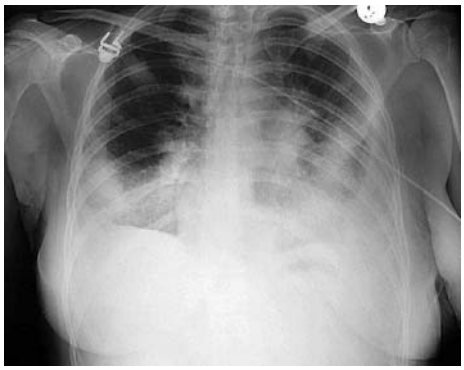

Figure 1 Chest *x* ray showing left sided pleural effusion, multifocal consolidation, patchy infiltrate in right upper lobe and enlarged left hilum.

F necrophorum is a gram negative, non-spore forming, anaerobic bacillus that occasionally proliferates in a cavity to produce local infection followed by distant spread via the blood (infection with anaerobic streptococci or other fusobacterium can cause a similar picture).[2] The most usual history is of infection of the tonsils or peritonsillar region, which spreads to the nearby veins causing a thrombophlebitis (patients often complain of pain along the sternocleidomastoid because of involvement of the internal jugular vein). Approximately 7–15 days later, they develop a febrile illness and symptoms of septic spread. The most commonly affected organs are the lungs (85%), but joints (26%), the peritoneum, liver, kidney, brain, bones, heart, and meninges (more common if the primary focus is otitis media) can all be involved.[2–4] There have also been some reports of the presenting complaint being a gluteal or psoas abscess or a picture of haemolytic uraemic syndrome.[5] Less commonly, the problem can originate from otitis media, mastoiditis, purulent endometritis, appendicitis (especially gangrenous), or infections of the urinary tract.

Many patients (50–75%)[1 2 4] have a raised bilirubin at presentation, often with hepatomegaly. The white blood cell count is usually in the range of $13–30\times10^9$/l and red blood cell count is often low with a haemolytic picture.

F necrophorium can be isolated from blood cultures in 2–4 days, which in a condition that has a mortality rate of >80%[2] if left untreated may cause a problem. The most recent microbiology paper that we found was a retrospective analysis of all reports of *F necrophorium* bacteraemia received by the UK Communicable Disease Surveillance Centre between 1990 and 2000.[6] There were 208 reported cases in England and Wales over the 10 year period with a peak incidence in late winter and a peak age range of 16–23 years. All isolates were susceptible to metronidazole, 15% were resistant to erythromycin, and 2% were resistant to penicillin.

Treatment consists of antibiotics and is supportive in most cases (reducing mortality to approximately 15%), with ligation of the internal jugular vein only needed very rarely in cases with recurrent septic emboli.[2]

There are several important lessons to be learnt from this case. Patients who return with the same complaint should be seen promptly by a senior doctor, and a safety mechanism should be in place for occasions when the senior doctors are unexpectedly busy (on this occasion with patients in the resuscitation room). Any patient who appears unwell at any time during their attendance should have their observations promptly performed/repeated. It also demonstrates the problems encountered in diagnosing Lemierre's syndrome, and that failure to diagnose the syndrome in its early phase may lead to a fatal outcome. Lastly, we believe this case highlights the important message that thoracic back pain often has an underlying aetiology, other than a simple mechanical cause, which should be actively sought with a thorough history, examination, and relevant investigations.

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Competing interests: none declared

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Accepted for publication 8 March 2004

REFERENCES

1 **Lemierre A**. On certain septicaemias due to anaerobic organisms. *Lancet* 1936;**1**:701–3.
2 **Barker J**, Winer-Muram HT, Grey SW. Lemierre syndrome. *South Med J* 1996;**89**:1021–3.
3 **Abele-Horn M**, Emmerling P, Mann JF. Lemierre's syndrome with spondylitis and pulmonary and gluteal abscesses associated with Mycoplasma pneumoniae pneumonia. *Eur J Clin Microbiol Infect Dis* 2001;**20**:263–6.
4 **Hagelskjaer K,** Prag J. Lemierre's syndrome. *Clin Infect Dis* 2000;**31**:524–32.
5 **Chand DH**, Brady RC, Bissler JJ. Hemolytic uremic syndrome in an adolescent with Fusobacterium necrophorium bacteraemia. *Am J Kidney Dis* 2001;**37**:e22.
6 **Brazier JS**, Hall V, Yusuf E, Duerden BI. Fusobacterium necrophorium infections in England and Wales 1990–2000. *J Med Microbiol* 2002;**51**:269–72.