simple glaucoma can be managed in general practice, preferably in conjunction with an ophthalmology clinic as regular measurements of intraocular pressure and visual field charting are necessary to assess adequacy of control. The drugs most commonly used in chronic simple glaucoma are timolol, adrenaline, pilocarpine, and guanethidine (with or without adrenaline) eye drops and oral acetazolamide (roughly in descending order of popularity). Surgery should be considered if these fail to control intraocular pressure.

Vision may be saved after central retinal arterial occlusion if effective treatment is given within three or four hours of onset and especially if the occlusion is embolic. Lie the patient flat and attempt to lower intraocular pressure by brief intermittent massage of the globe. Using both index fingers apply firm pressure—ask the patient to look down—for about 20 seconds and repeat this after an interval. (More prolonged compression may increase intraocular pressure and further impair retinal perfusion). If giant cell arteritis is a possibility systemic steroids should be given in high dose. Unfortunately vision is often lost, particularly because most patients present late, and emphasis should therefore be placed on preventing blindness in the other eye. Patients should be warned to report immediately if any symptoms develop in the "good" eye.

[1] Crombie AL. Eye disorders. In: Davies DM, ed. *Textbook of adverse drug reactions*. 2nd edition. Oxford: Oxford University Press, 1981:449-61.

Lesson of the Week

Necrobacillosis: a forgotten disease

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Abstract

Over four years five previously healthy young adults developed necrobacillosis, a severe septicaemic illness caused by Fusobacterium necrophorum. The infections were characterised by sore throat followed by rigors and the formation of metastatic abscesses and all caused considerable diagnostic confusion.

Necrobacillosis should be considered in previously healthy patients who develop severe septicaemia and pulmonary symptoms after an initial sore throat

Introduction

Necrobacillosis—infection with *Fusobacterium necrophorum*—is a severe suppurative illness that before the advent of antibiotics was often fatal. The presenting features are usually characteristic and were described in detail in 1936 by Lemierre,[1] who suggested that the diagnosis may be strongly suspected on clinical grounds alone. Despite the intense interest in anaerobic infections over the past decade *F necrophorum* is seldom specifically mentioned in reports and necrobacillosis is now rarely, if ever, diagnosed. Over four years we saw five patients with this illness, all of whom showed the classic features of the disease.

Case reports

Case 1—An 18 year old man developed a severe sore throat and over three days became very ill with rigors, pain in the back and shoulders, pleuritic chest pain, haemoptysis, and dyspnoea. On admission he had cervical lymphadenopathy and jaundice. A chest *x* ray film showed bilateral abscesses and a pleural effusion. The white cell count was $21{\cdot}4 \times 10^9$/l, blood urea concentration was raised at 13·9 mmol/l (83 mg/100 ml), and bilirubin concentration was 38 μmol/l (2·2 mg/100 ml). Initial treatment with intravenous erythromycin and cefuroxime produced no clinical improvement, and a week later shadowing was visible in the chest *x* ray film, which gallium scanning and computed tomography suggested was a paravertebral abscess. Metronidazole was added empirically to his treatment, and a few days later *F necrophorum* was isolated from two of the eight blood cultures taken on admission. Metronidazole was continued for two months, during which time he made a slow but complete recovery.

Case 2—A 20 year old woman developed a severe sore throat and over one week became very ill with fever, cough, abdominal pain, and dyspnoea. On admission she had bilateral cervical lymphadenopathy and jaundice. A chest *x* ray film showed extensive bilateral consolidation, which subsequently cavitated. The white cell count was raised at $17{\cdot}0 \times 10^9$/l, blood urea concentration raised at 38·3 mmol/l (230 mg/100 ml), and serum bilirubin concentration raised at 178 μmol/l (10·4 mg/100 ml). Treatment was initially with intravenous gentamicin, metronidazole, and ampicillin. Her abdominal pain worsened and the results of ultrasound scanning and computed tomography were highly suggestive of a perinephric abscess, but the result of laparotomy was entirely normal. Cultures of blood and pleural fluid taken on admission subsequently grew *F necrophorum* and a microaerophilic streptococcus. Metronidazole and benzylpenicillin, and then oral metronidazole and amoxycillin, were administered during her lengthy and severe illness, which was further complicated by joint effusions in hip and knee and an episode of obstruction of the small bowel. She eventually recovered completely.

Case 3—A 21 year old man developed a sore throat and over one week became very ill with high fever, pleuritic chest pain, haemoptysis, and severe dyspnoea. On admission cervical lymphadenopathy and haematuria were noted. A chest *x* ray film showed extensive bilateral consolidation and effusions. The white cell count was raised at $23{\cdot}7 \times 10^9$/l, blood urea concentration was normal, and serum bilirubin concentration was raised at 50 μmol/l (2·9 mg/100 ml). He improved after initial treatment with intravenous erythromycin but after a further five days again developed a fever. At this stage *F necrophorum* was

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isolated from blood cultures taken on admission and treatment was changed to oral amoxycillin and metronidazole. He made a complete recovery, and oral treatment was continued until complete four weeks after admission.

Case 4—A 24 year old woman developed a severe sore throat, becoming increasingly ill over five days with rigors, pleuritic chest pain, haemoptysis, and dyspnoea. On admission bilateral submandibular lymphadenopathy and haematuria were noted. A chest *x* ray film showed bilateral basal shadowing. Her white cell count was raised at $21{\cdot}9 \times 10^9$/l, blood urea concentration raised at 19·8 mmol/l (119 mg/100 ml), and serum bilirubin concentration raised at 38 μmol/l (2·2 mg/100 ml). Initial treatment with intravenous ampicillin and flucloxacillin and oral metronidazole resulted in clinical improvement within 24 hours. After a further two days *F necrophorum* was isolated from blood cultures taken on admission. Treatment was then changed to benzylpenicillin and metronidazole and continued for two weeks, followed by a further week of oral treatment with phenylmethylpenicillin and metronidazole. Recovery was uneventful.

Case 5—A 25 year old man developed a severe sore throat and over two days became ill with rigors, back pain, pleuritic chest pain, and haemoptysis. On admission a peritonsillar abscess and submandibular lymphadenopathy were noted. A chest *x* ray film showed shadowing around the left hilum. The white cell count was slightly raised at $11{\cdot}2 \times 10^9$/l, blood urea concentration was normal, and serum bilirubin concentration was slightly raised at 25 μmol/l (1·5 mg/100 ml). Initial treatment was with intravenous benzylpenicillin, and intravenous metronidazole was added two days later when a Gram negative anaerobe, subsequently identified as *F necrophorum*, was isolated from blood cultures taken on admission. His condition improved over the next few days, and treatment was changed to oral amoxycillin and metronidazole. After three weeks he was entirely well, although a left pleural rub persisted; he subsequently failed to attend for follow up.

Discussion

The clinical features of these five cases were remarkably similar. Almost 50 years ago septicaemias caused by the organism now known as *F necrophorum* were described as constituting a syndrome "so characteristic that mistake is almost impossible . . . it becomes relatively easy to make a diagnosis on clinical findings."[1] In 1955 Alston used the term "necrobacillosis" to describe infections with *F necrophorum* and reviewed previous reports and cases that had occurred in Great Britain.[2] Despite renewed, almost obsessive interest in anaerobic infections during the past decade this organism has been largely ignored. This may be partly attributable to its confusing nomenclature; at least 50 synonyms exist for *F necrophorum*. In addition, isolation of *F necrophorum*, whether from blood or other specimens, is technically demanding as it usually requires prolonged incubation under strictly anaerobic conditions. In a routine laboratory, identification may present even greater difficulty than isolation and the organism may be readily mistaken for *Bacteroides*. Its somewhat bizarre morphological appearance on Gram stained smears, with filaments and swellings,[2] should alert the microscopist to its true identity.

The acute septicaemic illness induced by *F necrophorum* in previously healthy people contrasts appreciably with other anaerobic infections, most of which are secondary either to some underlying abnormality or to surgical intervention. Most anaerobic infections are also characterised by the presence of several species of anaerobic bacteria and, often, aerobic bacteria as well. In necrobacillosis *F necrophorum* is usually found as the sole pathogen (though it may be accompanied by microaerophilic streptococci, as in case 2), and this feature is undoubtedly related to its toxins, which are distinct from those of other anaerobes.[3-6] *F necrophorum* is sensitive to most antibiotics in vitro, particularly penicillin, but is resistant to aminoglycosides. It is a commensal of the oropharynx, genitourinary tract, and gastrointestinal tract,[2] but the tonsillar region is the usual portal of entry.

Features of necrobacillosis are sore throat and a high fever, often with rigors and painful submandibular lymphadenopathy. Metastatic abscesses are common and usually involve the lung with associated pleuritic chest pain and haemoptysis.[1] Multisystemic manifestations are usual and compound the diagnostic confusion. Articular and bone lesions are common and may produce problems ranging from simple joint pains to florid osteomyelitis and pyoarthrosis.[7] Jaundice is also common,[8-10] and renal lesions may manifest as haematuria, albuminuria, and a rise in blood urea concentration.[1] There is usually leucocytosis. Although deaths from local infections are rare, complicating septicaemia has in the past had a grave outlook, and 45% of patients died in three series studied before the advent of antibiotics[2 8 10]; mortality was still 33% among patients treated with antibiotics.[2 8] The true mortality, however, may have been lower as only the most severe cases may have been reported, and difficulties in culturing and identifying the organism might have led to some patients recovering without a microbiological diagnosis having been made.

Alston observed that cases of infection with *F necrophorum* occasionally presented as empyema without a history of preceding septicaemia.[2] We have seen a similar presentation, and also two patients with septicaemia due to *F necrophorum* presenting with acute osteomyelitis but without pulmonary lesions. The "characteristic" clinical picture of necrobacillosis described by Lemierre[1] and illustrated by our five strikingly similar cases may be easily misdiagnosed as due to another infection, particularly staphylococcal septicaemia, especially if a history of a sore throat is not elicited. Indeed, the correct diagnosis was not entertained in any of our five cases until the microbiological findings were available, but the failure to isolate other pathogens should perhaps have led us to consider the correct diagnosis. Although *F necrophorum* is sensitive in vitro to most antibiotics, our experience and that of previous workers[11 12] is that the clinical response may be much slower than this sensitivity would suggest. Despite a lack of conclusive evidence, our clinical impression was that definite improvement coincided with the introduction of metronidazole in at least three of our patients.

Necrobacillosis has been described and studied in detail in the past but is now apparently almost forgotten. Our experience, and that of recent North American workers,[11] suggests that it may still occur; it should be suspected in previously healthy adolescents and young adults who after an initial sore throat develop a severe septicaemic illness with prominent pulmonary signs and symptoms.

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