An unusual case of diphtheria and its complications

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The clinical syndrome of diphtheria is usually caused by toxigenic *Corynebacterium diphtheriae*. However, this paper describes a case of *C. ulcerans* infection, which rarely occurs in humans and can present with a clinical picture indistinguishable from *C. diphtheriae* infection.

DISCUSSION

Diphtheria is now a rare disease, immunization programmes having had a major impact. Only four cases were reported in England and Wales between 1990 and 1997 (CDR Weekly, 1997). Most cases occur in non-immunized people and are acquired abroad (CDR Weekly, 1997). Most cases of diphtheria result from infection with toxin-producing strains of *C. diphtheriae*. However, cases of diphtheria caused by *C. ulcerans* have been reported. Incidents of human infection by this organism are rare. Five cases of *C. ulcerans* infection were recorded between 1951 and 1980 in England and Wales (Meers, 1979). In 1983, a further four cases were described in rural communities in Devon, occurring over a 2-year period (Hart, 1984).

C. ulcerans toxin is indistinguishable from the toxin produced by *C. diphtheriae*. Usually a commensal organism in horses and cattle, *C. ulcerans* causes mastitis in cows and has been isolated from cow's milk. Infection in humans is associated with contact with farm animals or consumption of unpasteurised dairy products. Human infection is said to occur in the summer months among rural populations. There is no evidence of person-to-person transmission.

This patient was unable to recall her vaccination history and records were unavailable. She did not have any of the recognized risk factors, living in an urban area with no contact with farm animals, and her illness occurring in December. The source of her infection is not known. It may be that cattle are not the only important host of *C. ulcerans*.

Infection with *C. ulcerans* generally only causes mild disease, characterized by a sore throat. Of the cases mentioned above, only one presented with severe diphtheria. Infection with *C. ulcerans* can cause severe diphtheria-like illness including exudative pharyngitis, pseudo-membrane formation, and cardiac and neurological complications. These systemic effects are caused by the production of exotoxin, which has its most damaging effects on the heart and nervous system.

The risk of developing these complications is proportional to the severity of the local disease. The case described here fitted closely the classical description of diphtheria with palatal paralysis in the third week and car-

CASE REPORT

A 76-year-old woman presented complaining of a 3-day history of sore throat, blocked nose and of difficulty swallowing. There was no significant past medical history. On examination, she was apyrexial and drooling saliva. A firm white exudate was seen on the soft palate and posterior pharyngeal wall. The uvula was swollen, but the epiglottis was not involved. Both nostrils were occluded with white exudate, preventing the passing of a nasendoscope. At this time, cardiac, chest and neurological examination were unremarkable. The white cell count was 17.6×10^9/litre (15.0 granulocytes), but other blood tests were normal. Electrocardiography showed normal sinus rhythm and the chest X-ray was unremarkable. Nose and throat swabs were taken. A provisional clinical diagnosis of diphtheria was made, despite the lack of any obvious risk factors. Broad-spectrum antibiotics were started.

Over 24 hours her condition deteriorated and she required a tracheotomy to maintain her airway. A coryneform bacterium was isolated from one of the swabs. Diphtheria antitoxin was therefore administered and intravenous penicillin commenced. *Corynebacterium ulcerans* was subsequently confirmed by Central Public Health Laboratory, London. Intravenous erythromycin was later added. The patient became hypoxic as a result of sloughing off of the pseudo-membrane causing obstruction of the tracheotomy and this precipitated admission to the intensive therapy unit for ventilatory support. Bronchoscopy revealed extensive pseudo-membrane extending from both nostrils into both lungs.

Fast atrial fibrillation and pulmonary oedema complicated her 12-day stay on the intensive therapy unit, although she had no history of previous cardiac problems. On transfer to the ward, she complained of difficulty swallowing and of choking while eating. Her voice had developed a nasal quality. Examination revealed palatal weakness. Laryngeal and pharyngeal weaknesses were confirmed on videofluoroscopy and nasogastric feeding was commenced. Insertion of a percutaneous gastrostomy feeding tube was ultimately required. Her rehabilitation was complicated by episodes of unstable angina, pulmonary oedema and poor exercise tolerance. Echocardiography demonstrated reduction in left ventricular function. Nine months following discharge she had made an almost complete recovery, living independently and having regained normal speech and swallowing.

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diomyopathy in the fourth week (Meers, 1979).

Cardiac complications

Clinically, congestive cardiac failure, progressive dyspnoea, cardiac dilatation and gallop rhythm may be apparent. The electrocardiogram may show ST segment changes, first-degree heart block, more severe forms of atrioventricular block and arrhythmias, including atrial fibrillation. Serum levels of aspartate-amino transferase reflect the severity of myocardial damage and can be used to monitor its course.

This patient had no previous cardiac history, but during her illness developed fast atrial fibrillation, pulmonary oedema and unstable angina. An echocardiogram demonstrated reduced left ventricular function. Despite the symptoms there was no significant elevation of serum cardiac enzymes and apart from the episode of atrial fibrillation, electrocardiography showed lateral ischaemic changes only.

Neurological complications

The symptoms that this patient demonstrated (difficulty swallowing, palatal paralysis and nasal speech) are typical. Other cranial neuropathies may develop including facial and oculomotor paralysis. Later, a peripheral motor neuropathy, primarily affecting the proximal muscles, may develop. Resolution of all neurological symptoms is usual.

CONCLUSIONS

Patients with toxigenic *C. ulcerans* infection should be managed as cases of clinical diphtheria. The decision to administer diphtheria antitoxin should be made on clinical grounds and should not be delayed by laboratory confirmation in cases where there is high suspicion of clinical diphtheria. Delay is associated with increased mortality. Antibiotic treatment aims to reduce toxin production and prevent the spread of the organism. Penicillin or erythromycin are the drugs of choice. **HM**

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Hart RJC (1984) *C. ulcerans* in humans and cattle in North Devon. *J Hyg Camb* **92:** 161–4

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IN THE PUBLIC'S VIEW...

Modernizing the NHS in 3 months and other Labour 'visions'

Another week, another story, another political opportunity. I reckon Tony Blair and Alan Milburn have a list of NHS visions. They move one down the list with each media moan. Each vision is nuttier and less well thought-out than the last.

A man's throat cancer is missed. Forget the difficulties of diagnosis; the media declare it a scandal. Professor Mike Richards, the cancer tsar, tells us that improving communication skills for doctors is 'of critical importance'. He would have got more doctors on his side by saying just how much communication is possible in the average 7-minute GP consultation, but instead he gave Mr Milburn the chance to move one down the list. Problem: doctors can't communicate. Solution: train all health workers together.

Six teams are to provide modernization plans for the NHS: Tony Blair's famous six Ps. Beveridge took 6 years to design the original NHS. These teams were given just 3 months: the deadline is July. But Mr Blair and Mr Milburn pre-empt everything by moving down their list. They say they are consulting widely with the professions, so why don't they shut up until the plans appear?

Milburn is obsessed with the idea of breaking down professional barriers. Sharing or delegation of tasks, especially in well coordinated units, already happens. By all means build on this idea, but there are dangers in making it the universal model, and difficult to see why there is any need for staff to train together. People who want to be doctors, nurses and physiotherapists have different educational backgrounds and different academic aspirations. Because some nurses later want to become doctors (rarely the other way round) is not a good reason for tearing up the whole structure of medical and paramedical training and, at a time when it is acknowledged that the NHS is in difficulty, imposing a radically different model that is used nowhere else in the world.

There are indeed schisms within the NHS caused by differing salaries and responsibilities, but the new model will not remove the schisms. They will simply appear somewhere else, between different grades of nurses earning different salaries, for example. Schisms occur because the NHS is staffed by human beings with human feelings, and some people don't get on with one another. The best way of getting people to work well together is to give them decent working conditions, not ask them to work too hard with stretched resources, and then to dole out a fair amount of praise. Not much of that is currently to the fore.

Messrs Blair and Milburn might also ponder that while they are pressing for flexibility between doctors, nurses, and everyone else in the NHS, an undue emphasis on consumerism and a misunderstanding of the variability inherent in medicine is forcing doctors' practice to become more and more narrow. All doctors of whatever specialty already share a large amount of their training, but that does not allow a foot surgeon easily to become a bowel surgeon, let alone a forensic psychiatrist. **HM**

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