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Tetanus presenting as dysphagia

M. KASANZEW*, F.R.C.S., B. BROWNE†, D.A. and P. DAWES‡, F.R.C.S.(Ed) (Bristol)

Introduction

Dysphagia, as one of the multitude of symptoms of tetanus is common (Macrae, 1974; Beaty, 1983). However, it is rare for it to be the initial and sole presenting symptom of the disease. We report a case in which none of the classical signs were evident in the early stages of the disease.

Case report

A 48-year-old woman was admitted with a two week history of gradually progressive dysphagia. Three days prior to her admission she suddenly developed total dysphagia for solids and liquids, including her own saliva. On careful questioning, her only other symptoms were a feeling of generalized weakness and a complaint that her voice was a little faint. She was a non-smoker, drank alcohol in moderation and was a keen gardener.

An initial examination confirmed her inability to swallow. She was apyrexial and all systems including her neurological system and neck movements were found to be normal. Routine blood tests were normal apart from a raised white cell count ($17\,000 \times 10^9$ cells/l) with a neurotphilia (82 per cent). Chest and lateral neck X-rays were normal, but a barium swallow performed two days after her admission showed an occlusive spasm in the cricopharyngeal region with overspill and aspiration into her trachea. She was referred to the Ear, Nose and Throat Department, where the findings were confirmed.

Indirect laryngoscopy showed that there was pooling of her saliva in the piriform fossae. That evening, direct laryngoscopy and oesophagoscopy were performed under general anaesthesia, to exclude the possibility of a mass obstructing the oesophagus. Once again, pooling of the saliva was noted. The larynx, pharynx and oesophagus were normal and in particular there was no spasm of the cricopharyngeus muscle. A nasogastric tube was inserted at the end of the procedure to allow feeding and rehydration via the enteral route.

The following day, she developed several distressing bouts of pharyngeal spasm, each lasting approximately ten minutes. A neurological opinion was sought. A thorough review of her history and a repeated examination suggested a differential diagnosis of motor neurone disease, myasthenia gravis or an hysterical grief reaction based on two recent deaths in her family for which she had suppressed her grief. The first of these was discounted because of insufficient clinical evidence. A normal edraphonium ('Tensilon') test discounted the second. The third seemed to be the most likely diagnosis. At this stage more than ten doctors had been involved with her assessment.

Only on the sixth day of her admission did she develop painful neck stiffness and mild trismus. Further questioning revealed that she had never received tetanus toxoid. There was no evidence of any trauma, but she did admit that she frequently sustained minor injury through her open sandals, and that her nails were often full of soil. Her tetanus toxin antibody level was 0.02 IU/ml., fifty times less than the protective level of 1.00 IU/ml (Pai *et al.*, 1986).

On the strength of this clinical diagnosis, she was given benzylpenicillin 600mg six hourly for ten days, diazepam or intravenous 'Diazemuls' to control the muscle spasms and anti-tetanus human immunoglobulin 500 units intramuscularly. Innoculation with tetanus toxoid was also started.

Spirometry revealed a deterioration of her respiratory function. In view of this and the need for close monitoring to avoid the risk of aspiration or laryngeal spasm, she was transferred to the Intensive Care Unit. She did not require intubation or artificial ventilation. Continuous monitoring of her Peak Flow and Forced Expiratory Volume showed that these did decrease to less than 35 per cent of her normal.

Two weeks after her admission, the pharyngeal spasms stopped, she could swallow her saliva and within a further week was able to manage a soft diet. Four weeks after her discharge she had improved greatly but complained of still feeling a little weak.

Discussion

Tetanus is a lethal, but treatable infectious disease caused by *Clostridium tetani*. Atrakchi and Wilson (1977) estimate that about 22 to 25 cases occur in England and Wales per year. Present mortalities range from 80 to 20 per cent (Adams *et al.*, 1966; Ganendran, 1974). The diagnosis of tetanus is usually easily made from the classical symptoms and signs, although these may be absent during the early stages of the disease and a traumatic lesion is not necessarily found (Macrae, 1974; Scott, 1980; Beaty, 1983; Watanabe *et al.*, 1984). The occupation of our patient allowed for ample exposure to Tetanus spores, an important aspect of her history to which insufficient attention was given.

This case demonstrates that although dysphagia is rarely the initial and major symptom of tetanus (Weider and Tingwald, 1970; Wang and Karmody, 1985), it should be remembered as an unusual cause in patients whose symptoms and signs do not fit a clear clinical picture.

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* Senior House Officer in Ear Nose and Throat Surgery, † Registrar in Anaesthetics and ‡ Registrar in Ear Nose and Throat Surgery, Southmead Hospital, Bristol.

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Address for correspondence:
Mr. Patrick Dawes,
37 Eastfield Road,
Westbury-on-Trym,
Bristol BS9 4AE.

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