Final diagnosis

Cardiovascular dysphagia.

Keywords: cardiovascular dysphagia; mitral valve disease; weight loss

- 1 Cappell MS. Manometric findings in dysphagia secondary to left atrial dilatation. *Dig Dis Sci* 1991;**36**:693–8.
- 2 McNally PR, Rak KM. Dysphagia lusoria caused by persistent right aortic arch with aberrant left subclavian artery and diverticulum of Kommerell. *Dig Dis Sci* 1992;**37**:144–9.
- 3 Berenzweig H, Baue AE, McCallum RW. Dysphagia lusoria: report of a case and review of the diagnostic and surgical approach. *Dig Dis Sci* 1980;**25**:630–6.
- 4 Mittal RK, Siskind BN, Hongo M, *et al.* Dysphagia aortica: clinical, radiological and manometric findings. *Dig Dis Sci* 1986;**31**:379–84.
- 5 Cappell MS. Endoscopic, radiographic and manometric findings associated with cardiovascular dysphagia. *Dig Dis Sci* 1995;**40**:166–76.

Lessons from a case of tetanus in an elderly woman

J Kwan, S Lim, S C Allen

A healthy 84-year-old Caucasian woman fell whilst gardening at home and lacerated her right leg. The wound was deep and heavily contaminated with soil and gravel. She attended an Accident and Emergency (A&E) department for treatment and the wound was cleaned and dressed. She had never received tetanus vaccination in the past and at the A&E department she was given one dose of tetanus toxoid. She was sent home without any follow up. Over the next week she became increasingly immobile and complained of dysphagia, stiff neck and breathlessness. Her general practitioner admitted her into a nursing home with a presumed diagnosis of stroke. Her condition deteriorated over the next week and she was admitted into hospital with a provisional diagnosis of septicaemia from her original leg wound.

On admission she was dehydrated, hypoxic, hypertensive (230/125 mmHg) and tachycardic. She had generalised muscular spasms including trismus and neck stiffness as well as hyperexcitability to touch and noise. Spirometry showed a restrictive lung defect. Initial laboratory results were as follows: leucocytes $15.5 \times 10^9/l$, platelets $653 \times 10^9/l$, urea 15.4 mmol/l, creatinine 227 $\mu\text{mol/l}$ and corrected calcium 2.48 mmol/l. A diagnosis of tetanus was made and she was transferred to the Intensive Care Unit where she was managed according to recommended guidelines for the treatment of tetanus. Her pulse and blood pressure observations were monitored closely (table).

Table Blood pressure and pulse observations on day 7

Time	Blood pressure (mmHg)	Pulse (beats/min)
01:00	122 / 50	80
04:00	90 / 40	70
07:00	125 / 60	80
12:00	150 / 50	100
20:00	160 / 70	120
22:00	130 / 70	88

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Questions

- 1 Comment on the original A&E department management of this patient.
- 2 What caused the abnormally unstable blood pressure and pulse?
- 3 What are the most important aspects of the management of tetanus?
- 4 What is the differential diagnosis and how may the initial diagnosis be confirmed?

Answers

QUESTION 1

The patient's wound was heavily contaminated with soil and gravel and, by definition,¹ the wound was tetanus prone. The patient had never received vaccination against tetanus. According to the Department of Health guidelines,¹ patients who have never been vaccinated and have a tetanus-prone wound should receive an immediate dose of human tetanus immunoglobulin followed by a complete primary course (three doses) of tetanus toxoid. The patient in our case, however, had not been given a dose of human tetanus immunoglobulin and was discharged without follow-up.

QUESTION 2

The labile blood pressure and tachycardia were most likely a result of a combination of pain (from both the wound and muscular spasms), anxiety, sepsis and autonomic dysfunction. Autonomic dysfunction is common in patients with tetanus and it may also present as pyrexia, dry mouth, profuse sweating, urinary retention and cardiac arrhythmia, which is associated with a mortality of over 50%.³ The unstable blood pressure of autonomic dysfunction is difficult to control, but some benefit has been demonstrated with the use of propranolol and clonidine.^{2,4}

QUESTION 3

The immediate management for severe tetanus should include surgical debridement of the wound, intravenous metronidazole and human tetanus immunoglobulin, antispasmodic therapy, early mechanical ventilation and tracheostomy. Nursing the patient in a quiet room prevents muscular spasms from hyperexcitability. Metronidazole has been shown to be more effective than penicillin in preventing death when used in the treatment of tetanus. It has been postulated that penicillin is a centrally acting GABA antagonist, and may therefore act synergistically with tetanospasmin in producing muscular spasms.² Early enteral or parenteral feeding is also recommended because of the high caloric requirement secondary to hypermetabolism in patients with tetanus. Subsequently, since the amount of tetanus toxin released is insufficient to induce an adequate immune response, the patient will also need immunisation with tetanus toxoid.

QUESTION 4

Tetanus is a clinical diagnosis. The combination of a history of injury with a contaminated wound, muscular spasms (with or without risus sardonicus, trismus or opisthotonus) and signs of sepsis should raise the possibility of the diagnosis. Tetanus immunisation history is a poor predictor of immune status in elderly

Learning points

- tetanus is a disease of the elderly population
- tetanus is preventable with appropriate wound care and prophylaxis
- admission into a nursing home without a proper diagnosis should be avoided
- autonomic dysfunction can cause labile blood pressure and pulse in tetanus

people with a positive predictive value of 50% and a negative value of 76%.⁵ It is important to exclude other differential diagnoses such as hypocalcaemic tetany, orofacial infection, status epilepticus and drug-induced dystonia (eg, phenothiazines, metoclopramide). Strychnine poisoning can mimic tetanus but it does not cause trismus or abdominal rigidity. Culture of *Clostridium tetani* from the affected wound is positive in less than 30% of cases. Moreover, a positive culture is not diagnostic since not all patients with wound colonisation develop tetanus. Serological tests may be used to detect the level of anti-tetanus antibody but their use in diagnosis is limited.

Discussion

In the UK, tetanus is a disease of the elderly population and elderly women are most at risk probably due to a combination of inadequate immunity and the increasing risk associated with a physically active old age. Tetanus immunisation began in 1938 for those who served in the Armed Forces. In 1961, primary tetanus immunisation of infants was introduced nationally. In 1970, tetanus immunisation was recommended as part of the routine management of all wounds. Between 1984 and 1995, there were 145 notified cases of tetanus in England and Wales; 53% of the cases were in individuals over 65 years and two-thirds of them were women.

Although tetanus is now rare, it is preventable by undertaking appropriate wound care and tetanus prophylaxis as recommended by the Department of Health.¹ Our case illustrates that admission of any elderly person into a private sector nursing home without a proper diagnosis may delay investigations and treatment of the underlying illness. The most important step in diagnosing tetanus is the maintenance of a high suspicion for the disease.

Final diagnosis

Autonomic dysfunction in an elderly patient with severe tetanus.

Keywords: tetanus; elderly; autonomic dysfunction

- 1 Salisbury D, Begg NT, eds. Tetanus. In: *Immunisation against infectious disease*. London: HMSO, 1996; pp 205–13.
- 2 Ernst ME, Klepser ME, Fouts M, Marangos MN. Tetanus: pathophysiology and management. *Ann Pharmacother* 1997; 31:1507–13.
- 3 Moughabghab AV, Lefilliatre P, Fenides A, Provot F. Management of autonomic dysfunction in severe tetanus: the use of fentanyl. *Can J Anaesthesia* 1995;42:955.
- 4 Gregorakos L, Kerezoudi E, Dimopoulos G, Thomaides T. Management of blood pressure instability in severe tetanus: the use of clonidine. *Intens Care Med* 1997;23:893–5.
- 5 Murphy SM, Hegarty DM, Feighery CS, Bernard Walsh J, Williams Y, Coakley DP. Tetanus immunity in elderly people. *Age Ageing* 1995;24:99–102.

Unusual presentations to a lipid clinic

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We describe the case-histories of two young men who both presented to a lipid clinic with mixed hyperlipidaemias.

Case 1

A 33-year-old man had been referred to the lipid clinic by the dermatology unit because of planar xanthoma that been found on a skin biopsy. Apart from previously seeing the Ear, Nose and Throat department for an 18-month history of snoring, there was no other relevant medical history nor other symptoms. He was a non-smoker, rarely took alcohol and at the time was not taking any medication. On examination he was overweight with a body mass index (BMI) of 29 kg/m². His blood pressure at presentation was 140/90 mmHg and pulse 72 beats/min. He was clinically euthyroid and had planar xanthoma on his hands and tuberous xanthoma on his elbows. He had been commenced on a lipid-lowering diet and a number of investigations were performed. His fasting serum cholesterol was 12.6 mmol/l, triglyceride 5.3 mmol/l and high-density lipoprotein (HDL) cholesterol 1.31 mmol/l. Plasma urea, creatinine and fasting glucose were normal, as were his liver function tests. However, thyroid function tests showed a thyroid-stimulating hormone of 257.7 mU/l (normal range 0.3–5.0) and free thyroxine of 1.1 pmol/l (10.3–19.4). Anti-thyroglobulin and antithyroid peroxidase antibodies were 396 and 240 IU/ml, respectively (0–180 and 0–50). Apolipoprotein (apo) E phenotype performed by Western blotting showed him to be a E2/E2 homozygote. On a lipid-lowering diet instigated by a dietician and thyroxine 150 µg per day his fasting serum cholesterol improved to 5.11 mmol/l and triglyceride to 2.15 mmol/l and his thyroid function tests were rendered normal.

Case 2

A 33-year-old man was referred to the lipid clinic by his general practitioner because of hyperlipidaemia and a family history of premature coronary heart disease. His mother had hypothyroidism and pernicious anaemia. On examination he was euthyroid and had no lipid stigmata. His BMI was 31 kg/m², blood pressure was 150/92 mmHg, and pulse 72 beats/min. Initial fasting serum cholesterol was 16.49 mmol/l, triglyceride 20.83 mmol/l and HDL-cholesterol 0.59 mmol/l. His renal and liver function were normal, as was a fasting plasma glucose. However, his TSH was 37.6 mU/l and free T4 8.5 pmol/l. Anti-thyroglobulin and anti-thyroid peroxidase antibodies were 2153 and 711 IU/ml, respectively. He was commenced on thyroxine which resulted in normalisation of his thyroid function, although his fasting serum cholesterol remained elevated at 11.8 mmol/l with triglyceride of 16.7 mmol/l. His apoE phenotype also by Western blotting was E2/E2. He was subsequently started on bezafibrate 400 mg (MONO) nocte with considerable improvement in his lipids.

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Questions

- 1 What lipid disorder do these two patients display?
- 2 How can the diagnosis be confirmed?
- 3 What other conditions is this lipid disorder associated with?