Clinical Reports

Horner's syndrome in severe tetanus

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Summary: A 66 year old man was admitted with a left Horner's syndrome, and trismus due to tetanus. Three days later he had respiratory arrests, classical tetanic spasms and was ventilated. He had associated severe autonomic dysfunction, tachyarrhythmias, hypotension, sweating and constipation. There was complete resolution of the left Horner's syndrome with recovery from tetanus.

Introduction

Tetanus is mainly a problem of the third world, and is an unusual infection in Britain. There were about 14 cases reported in 1989 and 7 cases reported up to 26 October 1990.¹ Mortality in a severe case of tetanus is still appreciable at about 7–10%. Common autonomic manifestations are tachycardia, arrhythmias, labile hypertension, peripheral vasoconstriction, refractory hypotension, fever and profuse sweating which are presumed to be due to β and α adrenergic activity.² This is probably due to uptake of tetanospasmin by peripheral endings of motor neurones, and sensory and autonomic nerve fibres. The toxin is selective and blocks the function of inhibitory neurones giving rise to reflex excitability. Toxins may also affect medullary centres.³

We report a case of severe tetanus presenting with a left-sided Horner's syndrome which, to our knowledge, has not been reported previously.

Case report

A 66 year old retired lorry driver who had not previously been immunized against tetanus presented with a history of sweating attacks for 4 days. These sweating attacks occurred every 3–4 hours, lasting up to an hour and were associated with nausea. He also had difficulty in opening his mouth for 3 days and painful muscle spasms, mainly affecting the left side of the face, sternal area and back. He had difficulty eating and drinking because of a combination of dysphagia and trismus.

He had relative polycythaemia for 6 years needing regular venesections, the last of which was

carried out a week earlier. He also had severe osteo-arthritis affecting both hips and had had a single epileptic fit in July 1989 with a negative computed tomographic scan and electroencephalogram.

On examination he was restless, afebrile, had dental caries and halitosis. There was evidence of an obvious left Horner's syndrome. He had a trismus but no other abnormality of the central nervous system, chest, cardiovascular system or abdomen.

Investigations showed a leucocytosis of $13.5 \times 10^9/l$, haemoglobin of 16.5 g/dl and a haematocrit of 48.6%. The liver function tests were normal except for an elevated AST of 82 U/l (normal up to 32 U/l). Blood cultures were negative. Chest X-ray showed a calcified primary in the left middle zone but no other abnormalities. Cervical spine X-ray showed disc degeneration in C5/6, C6/7, osteoarthritis and spondylitic changes. X-rays of the teeth showed dental caries affecting the right upper 3rd molar and periodontal disease affecting most of the upper molars.

Tetanus was suspected and he was started on intravenous benzylpenicillin and had a prophylactic dose of anti-tetanus immunoglobulin, 250 units intramuscularly. His condition deteriorated. He had difficulty bringing up thick, tenacious throat secretions. He continued to have back pain and was unable to swallow anything other than sips of water. Three days later he had a brief respiratory arrest and was transferred to the intensive therapy unit. He had classical tetanic spasms while having a mini-tracheostomy and during suction of tracheal secretions. He also had classical risus sardonicus associated with these attacks. There was an associated generalized sweating of the whole body except for the left half of the face.

He was paralysed, sedated with morphine, intu-

bated and ventilated. He was also given a further course of 3,000 units of anti-tetanus immunoglobulin intramuscularly. Whilst on the ventilator he was noted to have a labile hypertension and episodes of hypotension needing treatment with plasma expanders. He developed a paralytic ileus in spite of normal electrolytes necessitating total parenteral nutrition. He also had a staphylococcal pneumonia, whilst on the ventilator, which was treated successfully with intravenous flucloxacillin. He improved and was successfully weaned off the ventilator after 25 days. It was noted that the left Horner's syndrome had resolved completely. He made a total recovery and was discharged from hospital 2 weeks later.

The site of the primary infection in this patient was not obvious but dental caries was considered a possibility.

Discussion

Horner's syndrome is due to lesions usually involving sympathetic rami of T1 and is manifest by ipsilateral meiosis, ptosis, enophthalmos and absence of sweating over the face and neck.

Autonomic dysfunction is well recognized in tetanus.^{3,4} This is thought to be caused by the effects of tetanospasmin, predominantly acting on the brainstem and autonomic inter-neurons. Damage to the dorsal nucleus of the vagus has been reported in fatal tetanus.⁵ Life-threatening complications may occur including dysrhythmias, bradycardia

needing a temporary pacemaker, progressive hypotension and cardiac arrest.⁶ Tachycardia is an early sign.⁴ Profound sympathetic nervous system inhibition and increased parasympathetic activity also occur. Chlorpromazine, morphine and labetalol have all been used to treat these complications.

Horner's syndrome is seen as a manifestation of involvement of the fibre of the 1st thoracic nerve root of the stellate ganglion. Lesions may also arise in the hypothalamus, midbrain, pons, or medulla affecting the central sympathetic pathways giving rise to Horner's syndrome. We postulate that the left Horner's syndrome, in this patient, was a manifestation of autonomic nervous system dysfunction associated with severe tetanus and presumably central in origin. He also had episodes of tachycardia, hypotension, hypertension, increased sweating, salivation and paralytic ileus, seen as part of the associated autonomic nervous system dysfunction.

The Horner's syndrome resolved completely with recovery from tetanus. The other unusual observation was the significantly elevated titre of anti-nuclear antibody seen in the early stage of acute tetanus, which normalized with the recovery from tetanus. We have no explanation for this unusual finding.

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