

Acute transverse myelopathy complicating tetanus

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Summary

A 12-year-old boy presented with typical features of short incubation tetanus. Spastic paraparesis with a mid-thoracic sensory level was present from the time of the initial examination. X-rays of the spine and lumbar myelogram were normal. The paraparesis improved rapidly and within a month the patient was free of neurological deficit. The case is possibly a rare example of tetanus involving the central nervous system other than the anterior horn cells.

Introduction

The usual explanation for the muscular spasms and muscular rigidity encountered so often in tetanus is that the neurotoxin produced by *Clostridium tetani* spreads along the axis cylinders or perineural lymphatic channels to the neuraxis where it spreads up and down. It is fixed to the gangliosides of the grey matter where its mode of action is like that of strychnine, namely, diminution of synaptic inhibition. Parsons, Hormann and Feigen (1966) suggested that the toxin acts by lowering presynaptic resting membrane potential and that calcium and magnesium ions are involved in this action. Eccles (1957) suggested that the toxin possibly acts by blocking the Renshaw cell inhibiting system. Apart from its effect on the anterior horn cells, little is known about the effect of toxin produced by *C. tetani* on the rest of the central nervous system

Two surveys of the Nigerian experience stress hyperpyrexia, laryngeal spasms and retention of urine as the commonest neurological complications of tetanus (Johnstone, 1958; Adeuja and Osuntokun, 1971). Other authors have been particularly impressed by the high incidence of overactivity of the sympathetic nervous system, especially swings of arterial blood pressure and frequent cardiac dysrhythmias (Kerr *et al.*, 1968).

The clinical features of the case reported below would be consistent with a thoracic lesion, the nature of which remains uncertain.

Case report

A 12-year-old boy was admitted to Kenyatta

National Hospital with a 2-day history of chest pain radiating anteriorly and posteriorly, inability to open mouth fully, generalized backache and difficulties in walking because of generalized muscular spasms. He had cut his right hand with a dirty razor 3 days previously.

On examination, chest movements were impaired bilaterally and his abdomen was rigid. He had classical risus sardonicus. There was a moderate spastic paraparesis with hyper-reflexia and bilateral extensor plantar responses. A clinical diagnosis of short incubation period tetanus was made. A regime of i.m. tetanus toxoid 0.5 ml, antitetanus serum (25 000 units) after a test dose and i.v. diazepam 20 mg 6-hourly was instituted together with a single dose of i.m. procaine penicillin (2.4 Mu. units).

On this treatment the patient improved without the need for intensive care management. When reviewed 22 days after admission he was still unable to open the mouth fully but the cranial nerves were otherwise normal. He had a spastic paraparesis, more on the left than on the right. He had hyper-reflexia of the lower limbs and bilateral extensor plantar responses. Superficial abdominal reflexes were absent. There was no shift of the umbilicus. His gait was typical of bilateral spastic paraparesis. He had hyperaesthesiae to pin-prick and temperature extending to the costal margins. The posterior column sensory modalities were normal. Palpation of the spine caused tenderness at T₆/T₇ level. The cerebrospinal fluid was sterile but there were numerous polymorphs and a few lymphocytes. The CSF protein and sugar were normal. The CSF serology for syphilis was negative. The Mantoux test was negative. The plain films of the spine and the lumbar myelogram were normal. *C. tetani* was later isolated from the wound swab.

Progress

The clinical features of tetanus mentioned above subsided with treatment. The neurological signs too gradually improved and at the time of discharge (40 days after first being seen) the patient was walking without any difficulty. The only remaining

signs were increased deep tendon jerks in the lower limbs. When last reviewed (2 years after his admission) he was very well without any neurological signs.

Discussion

It is surprising that there are no neuropathological changes in muscle or neural tissue in the fatal cases of tetanus even when the spasms have been so prolonged as to cause or result in tonic contractures of skeletal muscles (Adams, Denny-Brown and Pearson, 1962). However, the work of Illis and Taylor (1971) suggests that other parts of the central nervous system may be involved more often than is realized. These authors found that a proportion of 25 survivors from tetanus had central nervous system symptoms such as irritability, fits, myoclonus and sleep disturbance. Furthermore, there were more electro-encephalographic abnormalities in these patients compared with the general population. However, there were no specific symptoms or signs of spinal cord lesions in these patients followed-up for 3 months to 11 years. Two of the patients, both females, had bladder symptoms such as urgency and difficulties in initiation of micturition. This accords with the reports of Johnstone (1958) Adeuja and Osuntokun (1971) who found a number of tetanus patients with retention of urine. Whether the bladder dysfunction is caused by cord lesion or not is uncertain but the possibility of autonomic nervous system disturbance cannot be discounted (Kerr *et al.*, 1968).

Fractures of the spine complicating tetanus as reported by Johnstone (1958) and Adeuja and Osuntokun (1971) might, if high enough, compress the cord. The patient reported here had normal plain radiographs of the spine, and significant spinal cord compression was reliably excluded by myelography. Moreover, the clinical progress from moderate paraparesis to full recovery in less than one month without specific treatment is a strong point against mechanical spinal cord compression. It is worth noting that although 31% of the patients (154 out of 503) reported by Adeuja and Osuntokun (1971) had limb hyper-reflexia, only 2% (10 patients) had fractures of the spine.

Antitetanus serum injection has been known to cause serious neurological complications. Valerio (1952) described 2 cases of oculomotor nerve palsy following antitetanus serum injections, and Guillain-Barré syndrome may occur as a complication of tetanus antitoxin injection (Arbseman *et al.*, 1958;

Miglets *et al.*, 1960). Acute transverse myelitis has been reported following diphtheria, tetanus and polio immunization (Whittle and Robertson, 1977). Whatever mechanisms may have been involved in these cases would not explain the present patient's neurological picture, typical of transverse myelopathy, which was evident well before the antitoxin or any other drug was given.

A possible explanation is that there was a transient focal area of demyelination in the thoracic spinal cord as a result of the neurotoxin produced by *C. tetani*. However, in absence of histopathological studies this suggestion remains unproved. This mechanism which has not been described in tetanus, if proved, might partly explain the frequent finding of hyper-reflexia and bladder disturbance in this disease.

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