Brain lesions in the course of generalised tetanus

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CASE REPORT

A 47 year old woman developed left hemiparesis primarily affecting the lower limbs during the course of severe generalised tetanus. MRI on the 82nd hospital day revealed cortical and subcortical lesions predominantly in the right frontal and parietal lobes in addition to marked brain atrophy. Three months later, the enhancing lesions were still present on follow up MRI. We postulate that structures above brainstem may be involved in severe generalised tetanus.

Tetanus is an exotoxin mediated disease caused by Clostridium tetani, an anaerobic, Gram positive, spore forming rod. Tetanus toxin produced by this organism exerts its effect on the spinal cord, brainstem, and at neuromuscular junctions.1, 2 The toxin reaches the CNS by retrograde axonal transport through motor and vegetative efferent axons.3 Once transported into the CNS, the toxin can continue rostal movement by retrograde transport.4 This implies the possibility of involvement of structures above the brainstem by tetanus toxin.5 We present a patient with generalised tetanus who developed left hemiparesis with unusual brain imaging findings. Our observations suggest the involvement of structures above brainstem in generalised tetanus.

DISCUSSION

The effects of tetanus toxin on the spinal cord and brainstem are due to the inhibition of γ-aminobutyric acid (GABA) and glycine transmission between inhibitory neurons and motor-neurons.1 Cortical GABAergic inhibitory networks probably exist in humans, however, to what extent structures above brainstem are involved in patients with tetanus remains unknown.2 To our knowledge, there have been few clinical reports describing the possibility of this involvement. Warren et al7 reported the involvement of the cortex in a patient with generalised tetanus by physiologic study. Transcranial magnetic brain stimulation in their patient revealed enlarged electromyographic (EMG) responses and absence or reduction of the late phase of EMG silence following the motor evoked potential. They proposed that the disruption of central nervous inhibitory mechanisms in generalised tetanus might be widespread. Barlow et al8 reported that children who survived neonatal tetanus had evidence of brain damage that manifested as microcephaly, mild neurologic abnormalities, and developmental impairment.

The manifestations of our patient such as muscular spasm and autonomic involvement are consistent with generalised tetanus.
tetanus. In contrast, hemiparesis in the course of tetanus is uncommon. The brain lesions in our patient were not likely to represent ischemic infarction for at least three reasons. Firstly, the patient had no risk factors for brain infarction before the admission. She had no history of hypertension, diabetes mellitus, drug misuse, or oral contraception. Symptoms, signs, and laboratory data supporting diagnosis of collagen disease or coagulation abnormality were absent. Brain infarction in such a middle aged woman is uncommon. Secondly, we started ventilatory support before the onset of any severe hypoxia. No complications except for sinus tachycardia occurred that could have caused brain ischaemia such as hypotension, hypoxia, or severe ventricular arrhythmia. Thirdly, enhancement of lesions on MRI was still present three months later. This finding is not consistent with brain infarction because the enhancement may be seen in a recent but not an old infarct. Thus, we speculate that the lesions were associated with causes other than brain infarction. The enhancing lesions were not periventricular but were associated with cortical lesions. This finding is uncommon in demyelinating diseases such as multiple sclerosis. We also speculate that brain atrophy in our patient occurred in the course of tetanus because she developed cognitive impairment after admission. Furthermore, it is interesting that the enhancing lesions on MRI in our patient were present in the right frontal lobes near the central sulcus, an area that includes upper motor neurons monosynaptically or multisynaptically connected to anterior horn cells innervating muscles below the left knee, where the patient sustained the infected wound. From this topography and brain atrophy, the brain lesions may be associated with the disruption of central nervous inhibitory mechanisms by
tetanus toxin as proposed by Warren et al.\textsuperscript{7} The amount of toxin in our case may have been large enough to gain access to inhibitory pathways above brainstem considering the severity of the clinical course. Our observations suggest that tetanus may have a broader clinical spectrum of CNS involvement than previously realised.

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