Cephalic tetanus following tooth extraction in a Nigerian woman

Sir,

Tetanus is an acute, often fatal, disease caused by an exotoxin produced by the bacterium *Clostridium tetani*. Out of the four major clinical types of tetanus (generalized, localized, neonatal and cephalic), cephalic type is the rarest.[1] We encountered a case of cephalic tetanus in a young Nigerian woman six months after she had tooth extraction.

The patient is a 22 year old Nigerian lady, who four days before admission, developed difficulty in opening her mouth as a result of pain, which was associated with inability to swallow, excessive salivation and neck pain. At the time of presentation, patient was noticed to be having intermittent spasm of the muscles of the face and neck. Angle of her mouth was also deviated to the right; however, patient was conscious with ability to appropriately respond to communication in between spasm. There was no discharge from the ear or the nostrils. No relevant history of cough or head trauma was given. Six months before the onset of her symptoms, patient had a mouth infection for which her tooth was extracted from a tertiary health centre. There was no tetanus toxoid given to the patient at the time, though the patient claimed she completed her infancy tetanus toxoid schedule according to the National Programme on Immunization. The patient had no history of genital instrumentation.

At the time of physical examination, the patient had a body temperature of 37.2°C, pulse of 102 beats per minute which was regular, a respiratory rate of 24 cycles per minute and a blood pressure of 100/80 mmHg. Significant physical findings were seen in the neurological examination. Though patient was conscious, she was tensed up and had trismus. She was able to communicate with difficulty amidst intermittent spasm of the muscles of the face and neck. She had neck stiffness but negative Kernig’s and Brudzinski’s sign. There was left incomplete ptosis and lower motor neuron type left facial nerve palsy. Power in the four limbs was normal and deep tendon reflexes were also normal. Sensory functions were apparently within normal limits though examination was limited because of the patient’s condition.

Chest radiograph and lumbar puncture results were within normal limits. Electrocardiogram revealed a sinus tachycardia with a ventricular rate of 103 and a non specific ST-T wave changes. There was leucocytosis with a total white blood cell count of 16,000/mm³. The patient was managed in the general ward with antitetanus sera, tetanus toxoid, intravenous antibiotic, diazepam and ophanedrine. Other supportive treatments were given as well.

Patient did well and was discharged home after 8 days on admission. She had since been seen twice in the medical outpatient with sustained improvement.

Cephalic tetanus presents with cranial nerve palsy, particularly of the facial nerve, and is always scored as severe or very severe, since approximately two thirds of cases progress to generalized tetanus.[2] However, our patient did not progress to develop generalized tetanus. We could not send the patient for neuroimaging such as Computerized tomography scan or magnetic resonance imaging scan because they were not available in our centre and the nearest facility where there was a functional one is more than 200 kilometer far from our centre.

In conclusion, it is important to highlight a few aspects of this case.

First, this patient’s incubation period was long and she never progressed to develop generalized tetanus which probably might have influenced the good clinical outcome.[3] Second, the management of this patient has once again exposed the poor facilities prevailing in our health sector. Finally, tetanus is still a major health burden in Nigeria. There is need to promote health education to reduce the prevalence of this disease in this part of the world.

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References
Sir,

We report a 43-year-old man who presented with history of left hemicranial headache and right upper limb and facial focal onset motor seizures with secondary generalization of 3-month duration. There was no past history of tuberculosis, immunosuppressive states, diabetes mellitus or trauma. He was not from a low socioeconomic status and there was no family history of tuberculosis. On examination he had slurring of speech, decreased word output, bilateral papilloedema, right upper motor neuron facial paresis and right upper limb grade 4 muscle power. Computed tomogram (CT) of brain revealed a left posterior frontal ring-enhancing lesion with perilesional edema and an isodense frontoparietal subdural collection [Figure 1]. Magnetic resonance imaging (MRI) of brain revealed a left posterior frontal 2.8-cm lobulated lesion, hypo to iso on T1W images, isointense to gray matter on T2W images with postgadolinium ring enhancement and perilesional oedema [Figure 2]. There was an associated frontoparietal subdural collection which was hyperintense on both T1W and T2W images suggestive of subacute blood. He underwent left fronto-temporo-parietal craniotomy, evacuation of subdural hematoma and near total excision of the lesion. At surgery it was found that there was altered blood in the subdural space with thickened inner and outer membranes. The motor cortex was identified using electrophysiological monitoring and the lesion was just anterior to the motor cortex. It was well-defined with central necrotic material. The posterior wall of the lesion close to the motor cortex was left behind. The biopsy was reported as tuberculous granuloma. He was discharged on an 18-month course of antituberculous drugs. At 1-year follow-up he was asymptomatic and CT brain showed resolution of the lesion and hematoma [Figure 3].

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