Case Report

Guillain-Barré syndrome (Paraplegic variety) following snake bite- A rare case presentation

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ABSTRACT

A 60-year-old man presented with weakness in bilateral lower limbs for 10 days and radicular pain in both lower limbs for 5 days. He had been bitten by a snake while working in a field 20 days earlier and developed cellulitis of left lower limb following the bite and had received tetanus toxoid and anti–snake venom for two days. He had clinical, biochemical, and electrophysiological features of Guillain-Barré syndrome, with motor and sensory neuropathy—suggestive of demyelinating pattern. Recognition of this unusual complication following snake bite or use of anti–snake venom / tetanus toxoid has considerable epidemiological, therapeutic, and prognostic significance.

Keywords: Guillain-Barrésyndrome, paraplegic variety, snake bite.

Introduction

Guillain-Barre Syndrome (GBS) can occur after viral or bacterial infections, in autoimmune diseases, after administration of certain drugs and vaccines, following surgery or organ transplantation, or following snake bite. Till date no case has been reported of GB syndrome (paraplegic variety) following snake bite.In this article, we present one such case of GBS (paraplegic variety)following snake bite.

Case Report

A 60-year-old man, a farmer by profession, presented with a healing wound on the left ankle that had been present for the last 20 days, weakness in all four limbs of 10 days and radicular pain in both feet for the last 5 days. He had been bitten by a snake while working in a field 20 days earlier. He had received one dose of tetanus toxoid and two days of anti–snake venom at a local hospital. After about 10 days, he experienced weakness in lower extremities, followed by the development of radicular pain in both lower limbs over the next 5 days. There was no history of dysphagia, dysphonia, diplopia, or bowel and bladder involvement. The past history and family history were non-contributory. He had a healing wound over the left. Cranial nerves examination was normal. The patient had mild sensory impairment in both lower limbs below the knee, generalized hypotonia and areflexia, reduced hand grip strength, and grade 2/5, Medical Research Council (MRC) motor power in both lower limbs.plantars-bilateral flexor response. The autonomic functions were normal. Routine hemogram and biochemistry were unremarkable. CSF revealed albumino-cytological dissociation (proteins: 158 mg/dl, glucose-70mg/dl, cell count: 5 lymphocytes/mm^3,). MRI lumbar spine is normal. Sensory nerve conduction study showed prolonged F,multifocal conduction blocks suggesting demyelinating pattern. Repetitive nerve stimulation test did not show any decremental response. The features were suggestive of motor

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and sensory neuropathy—primarily demyelinating variety with secondary axonal degeneration .There are no antibodies specific for paraplegic variant, so not performed.

The patient was diagnosed to have GBS (paraplegic variety) and was treated with Immunoglobulins. He underwent short course of in patient rehabilitation and was completely recovered.

Discussion

The features of GBS in our patient could be attributed to the snake bite or the administration of tetanus toxoid or anti–snake venom. To the best of our knowledge nocase of GBS with paraplegic variant following snake bite has been reported in the literature.

GBS with post vaccination is an autoimmune process, the end result is an attack on the peripheral nerves and myelin, the fatty insulating layer of the nerve and a nerve conduction block leading to muscle paralysis that may be accompanied by sensory or autonomic disturbances.In mild cases nerve axon function remains intact and recovery rapid if remyelination occurs. In severe cases axonal damage occurs, about 80% of patients have myelin loss and 20% have pathological axonal loss.

The neuromuscular complications of snake bite include neuromuscular junction transmission abnormality, which can be caused by elapid and some sea snake venom, and myotoxicity, which can be caused by elapid as well as viper venom. Chuang et al. described a patient who developed axonal GBS following the bite of a Formosan krait. The patient presented with symmetric paresis and sensory signs in the lower limbs, autonomic dysfunction and mild elevation of cerebrospinal fluid (CSF) protein at about 4 weeks after the bite, but had good functional recovery. Electrodiagnostic studies revealed profound sensory and motor polyneuropathy. Repeat electrophysiologic examination confirmed nerve regeneration. One case of axonal sensory motor neuropathy following snake bite has been reported from India against the background of a sepsis syndrome, with sparing of the cranial nerves but with autonomic dysfunction. CSF study, even at 2 weeks after the illness, was normal and favored critical illness neuropathy rather than GBS.

Newton and Janati reported a case of GBS that developed after the injection of pure tetanus toxoid. They demonstrated a hypersensitive lymphoblastic transformation occurring in response to purified tetanus antigen; also, typing for disease-associated antigens was homozygous for HLA-B8.

Conclusion

GBS can occur following a snake bite or after the administration of tetanus toxoid and ASV. When patients present with sudden onset weakness with radicular pains following snake bite GB syndrome should be considered as a possible etiology.

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