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LETTER TO EDITOR

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Acute axonal polyneuropathy following resection of a glioblastoma multiforme

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Sir,

We describe a patient with a multicentric, bifrontal glioblastoma multiforme (GBM), who after surgery and radiotherapy, presented with acute axonal sensorimotor polyneuropathy. We aim to highlight the rare association of acute axonal polyneuropathy (Guillain Barre syndrome [GBS] variant) in patients with GBM. Our patient presented with descending motor paralysis that started with cranial nerve involvement, which has never been reported before. Multiple imaging studies of the spine and a lumbar puncture were performed to rule out the causation of his neurological presentation due to the leptomeningeal spread of the GBM. The nerve conduction study was consistent with acute axonal sensorimotor neuropathy. The patient was treated with intravenous immunoglobulin and showed clinical improvement.

A 28-year old male patient underwent surgical resection of a bifrontal (left > right) brain mass. The histopathological diagnosis was a GBM. He was readmitted to the hospital after 4 weeks with a high blood sugar and mild urinary tract infection. Neurological exam was normal at admission. Three days after the admission, he developed right eye ptosis followed by progressive third nerve involvement. Over the next few days, he had difficulty in abducting the right eye (VI nerve palsy), bi-facial paralysis (bilateral VII nerve palsy), difficulty in swallowing and weakness of pharyngeal muscles (IX and X nerve palsy), and weakness of muscles of the tongue (XII nerve palsy). He also started developing difficulty in breathing and started getting tired easily. He was intubated due to his deteriorating respiratory function. The weakness progressively involved bilateral upper and then lower limbs leading to quadriplegia. Neurological exam revealed hypotonia and absent muscle stretch reflexes. Magnetic resonance imaging (MRI) of the brain with contrast administration revealed stable and unaltered postoperative changes that were present as a result of the previously performed surgery. There was no involvement of the brainstem or enhancement of the meninges. Magnetic resonance imaging (MRI) of the spine was also unremarkable. Lumbar puncture was performed 2 days after the onset of cranial nerve paresis, which showed a normal white cell count and protein level. Cytology showed no malignant cells; and, the immunological titres for Lyme's disease and the vasculitic panel were normal. GQ1b and GM1 antibody tests could not be done due to their unavailability. Nerve conduction studies showed the presence of a diffuse sensorimotor axonal polyneuropathy. The amplitude of the compound motor action potential and sensory nerve action potential were severely reduced in the upper and lower limbs; *F* waves were absent. The patient was started on intravenous immunoglobulin for a 5-day course. Two weeks after starting the infusion, the patient had improvement in muscle strength in the upper extremities and recovered the entire spectrum of cranial nerve function.

GBS is a well-known entity that presents with rapidly evolving ascending weakness, mild sensory loss, and loss of muscle reflexes. The most common variant of GBS is acute inflammatory demyelinating polyneuropathy (AIDP).

Other variants of GBS present with variable involvement of motor and sensory nerves. Based on the clinical picture and nerve conductions studies, we established the diagnosis of acute axonal polyneuropathy. Laboratory investigations helped in ruling out autoimmune diseases, infection, and other causes of GBS.[1] Patients with GBS typically have an antecedent event that precipitates weakness, such as infection, surgery, or physiological stress. In this case, the precipitating event may have been the brain surgery that the patient underwent. GBS commonly presents as an ascending motor paralysis. Our patient presented with descending motor paralysis and involvement of both sensory and motor nerves.[2] There are rare variants of GBS that can present with descending paralysis with involvement of both sensory and motor nerves. Acute motor sensory axonal neuropathy (AMSAN) variant of GBS is known to affect the motor as well as the sensory nerves.[3] AMSAN variant is usually associated with antecedent *Campylobacter jejuni* infection and has a more aggressive course, which would confirm to our patient's clinical presentation.[4],[5] GD1a and GM1 antibody titres could not be obtained due to the unavailability of the test in our setting; however, our patient had no history of a prior diarrhoeal illness suggestive of *C.jejuni* infection. This case highlights the possibility of occurrence of acute axonal polyneuropathy-like illness in a patient with GBM following definitive surgery for tumor removal. A review of the literature revealed two prior case reports of GBS with GBM. Both cases presented with ascending motor paralysis, and in one of these cases, the onset of GBS occurred following surgery that the patient underwent. We did not come across any case where the patient presented with the GBS variant of descending paralysis.[6],[7] Various pathogenic mechanisms ranging from immunosuppression (due to the administration of steroids) to molecular mimicry, leading to an exaggerated autoimmune response, have been discussed;[6],[7] however, no conclusive evidence exists due to the paucity of the published cases. It is important to be aware of this presentation as, most of the times, due to the poor prognosis of the underlying GBM, the presence of this entity may go undiagnosed and untreated. Our patient received a course of immunoglobulin and showed clinical improvement; therapeutic membrane plasmapheresis has also been suggested for treating this entity.[8]

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Conflicts of interest

There are no conflicts of interest.

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