Guillain-Barré syndrome presenting as bilateral vocal cord paralysis

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Guillain-Barré syndrome (GBS), also known as acute idiopathic polyneuritis, is the most common acquired demyelinating neuropathy, characterized by muscular weakness and mild distal sensory loss. GBS presenting as bilateral vocal cord paralysis (BVCP) is extremely rare, with only 1 other case reported in the literature.[1] The following is a case of BVCP as the presenting symptom of GBS in an elderly man.

CASE REPORT

A 70-year-old man came to the emergency department with a 3-week history of progressive dysphonia and increasing shortness of breath for the previous 3 days. His medical history was significant only for an upper respiratory tract infection 1 week before the onset of symptoms. He denied taking medications and had a 50 pack-year smoking history. His family history was negative for significant disease, including diabetes mellitus, thyroid disease, and myasthenia gravis.

Physical examination revealed a blood pressure of 130/90 mm Hg, respiratory rate of 24 breaths/minute, and temperature of 37.2°C. Oxygen saturation was 91% on room air. The head and neck examination findings were within normal limits. Chest auscultation revealed biphasic stridor. Flexible laryngoscopy showed BVCP, with the cords positioned in the midline. The initial neurologic evaluation, including all cranial nerves, deep tendon reflexes, muscular strength and sensation was normal. Results of a Tensilon test were negative, thereby excluding myasthenia gravis. We elected to perform an urgent tracheotomy to protect the airway. Panendoscopy at the time of the tracheotomy was unremarkable.

On postoperative day 1, the patient underwent a CT scan of the head, neck, and chest. Findings in all areas were within normal limits. A lumbar puncture was performed, showing a marked elevation of the protein concentration. The remainder of the cerebrospinal fluid (CSF) was normal. On postoperative day 2, he experienced progressive weakness of both lower extremities, with diminution of deep tendon reflexes. On the basis of the CSF findings and neurologic examination, the diagnosis of GBS was made.

The patient began a course of plasmapheresis. Within 2 weeks of treatment initiation, he had marked improvement of his vocal cord mobility and lower extremity weakness. At 4-month follow-up, his neurologic examination had returned to normal with respect to his extremities; however, he remains cannulated, with only partial return of vocal cord mobility.

DISCUSSION

GBS is an acute polyneuritis caused by segmental demyelination.[2] Typically, peripheral nerves are affected, although cranial nerves may be involved as well. It affects males and females equally and occurs in all age groups. It commonly occurs after upper respiratory or gastrointestinal viral infection, often with a 1- to 3-week delay. Most often, GBS presents with lower extremity weakness and paresthesias, progressing as an ascending muscular weakness with loss of deep tendon reflexes. The disease can progress to the development of cardiac arrhythmia, extreme blood pressure lability, and acute respiratory distress warranting mechanical ventilation.

GBS presenting as BVCP is extremely rare, with only 1 case having been reported in the literature.[1] Five additional cases of BVCP developing during the course of treatment have also been reported.[3-6] The cause of BVCP is extensive and includes postthyroidectomy recurrent laryngeal nerve damage, poliomyelitis, Parkinson's disease, cerebrovascular accident, multiple sclerosis, myasthenia gravis, central nervous system/neck/chest neoplasm, infection, and congenital paralysis.[4]

Diagnosis of GBS is based on the CSF and neurologic examinations.[2] The CSF shows a markedly elevated protein concentration, with normal pressure, cell count, and glucose concentrations. CSF cultures reveal no growth. There are no typical radiologic findings. Pathologic examination reveals inflammatory segmental demyelination of nerve sheaths with perivascular lymphocytic infiltration.

The cause of GBS is unclear. Physiologic stressors such as surgery, immunization, and infection have been indicated. Possible infectious sources include Epstein-Barr virus, infec-

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tious hepatitis, cytomegalovirus, and influenza A virus. The differential diagnosis of GBS includes diphtheria polyneuropathy, poliomyelitis, HIV-associated chronic inflammatory neuropathy, Lyme disease, botulism, myasthenia gravis, acute intermittent porphyria, acute toxic neuropathy, and hysterical paralysis. The findings of a progressive ascending weakness coupled to an elevated CSF protein content should alert the physician to the possibility of GBS in cases of BVCP.

The treatment of GBS involves plasmapheresis within 2 weeks of the onset of symptoms. Seventy-five percent of patients make a full recovery, with the recovery phase typically plateauing at several weeks and potentially lasting up to 2 years. In general, children recover more quickly than adults. Pathologic examination reveals normal remyelination.

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