Case Report
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Korean J Otorhinolaryngol-Head Neck Surg 2013;56:169-71 / pISSN 2092-5859 / eISSN 2092-6529
http://dx.doi.org/10.3342/kjorl-hns.2013.56.3.169

Guillain-Barré Syndrome Presenting as Bilateral Vocal Cord Paralysis

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양측 성대마비를 주소로 내원한 길랑-바레 증후군

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Received October 18, 2012
Revised December 17, 2012
Accepted December 17, 2012
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Guillain-Barré syndrome (GBS) presenting as bilateral vocal cord paralysis is extremely rare. We report an unusual case of GBS in which the patient manifested hoarseness resulting from bilateral vocal cord paralysis. In conclusion, GBS needs to be considered as possible causes of new onset bilateral vocal cord paralysis. We emphasize that early recognition of atypical presentations of GBS warrants further evaluation and appropriate management.

Korean J Otorhinolaryngol-Head Neck Surg 2013;56:169-71

Key Words Guillain-Barré syndrome · Intravenous immunoglobulin · Vocal cord paralysis.

Introduction

Guillain-Barré syndrome (GBS) is an inflammatory demyelinating polyneuropathy associated with rapid progression and characterized by progressive ascending motor weakness with areflexia, primarily arising in the lower extremities. However, GBS presenting as bilateral vocal cord paralysis is extremely rare with only few cases reported in the literature.[1-4] Herein, we report an unusual case of GBS in which the patient manifested hoarseness resulting from bilateral vocal cord paralysis in a 61-year-old woman.

Case

A 61-year-old female patient arrived at the Chonnam National University Hospital with a 2-day history of progressive hoarseness, dysphagia, and aspiration. The medical history of the patient was unremarkable except for an upper respiratory tract infection 7 days prior to the onset of the symptoms she was experiencing. The patient denied taking any medications and was a non-smoker with no history of alcohol consumption.

Vital signs, including blood pressure, respiratory rate, and body temperature, were normal. Head and neck examination finding were within normal limits. Flexible laryngoscopic examination revealed that both vocal cords were fixed in the lateral position during inspiration and expiration (Fig. 1). The initial neurologic evaluation results, including all cranial nerves, deep tendon reflexes, muscular strength and sensation, were normal. The initial laboratory test results were unremarkable. The patient underwent a computed tomography (CT) scan of the head and neck. The findings in all areas were normal.

On the first evening after admission, the patient experienced

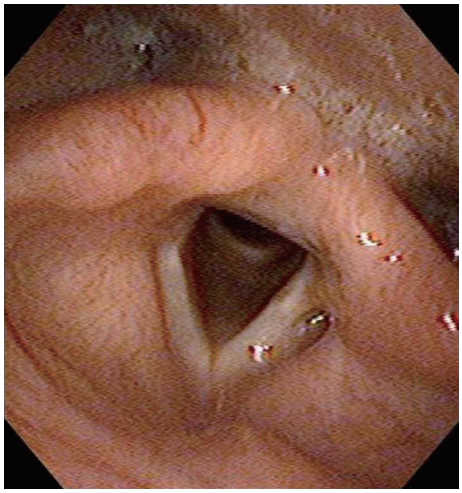

Fig. 1. Flexible laryngoscopic examination shows that both vocal cords were fixed in the lateral position during inspiration and expiration.

progressive weakness in both upper extremities with diminution of deep tendon reflexes. However, her respiratory status remained stable. A neurology consultation was requested. The neurologic evaluation did not uncover any signs of ophthalmoplegia, ptosis, or facial muscle weakness, but the gag reflex was absent. The muscle strength of both upper and lower extremities was 2/5 (muscle can move only if the resistance of gravity is removed).

A lumbar puncture was performed and revealed an elevated protein concentration. The remainder of the cerebrospinal fluid (CSF) was normal. Electrodiagnostic studies showed that the patient had reduced motor nerve conduction velocities and prolonged distal latencies. The results of serologic tests and real time polymerase chain reaction for *Mycoplasma pneumoniae* and Ebstein-Barr virus were positive. Based on clinical symptoms, CSF findings, and electrophysiologic examination, the patient was diagnosed with GBS.

The patient was treated with high-dose immunoglobulin delivered intravenously at 400 mg/kg per day for 5 consecutive days. One day after treatment initiation, she developed acute respiratory distress and required mechanical ventilation because of respiratory muscle weakness. Nine days after treatment, oral intubation was removed. Within 2 weeks of treatment initiation, the patient showed markedly improved vocal cord mobility and the weakness in her extremities began to disappear. 1 month later, the patient presented no hoarseness and aspiration with completely return of vocal cord mobility (Fig. 2).

This article was approved by the Institutional Review Board of Chonnam National University Hwasun Hospital.

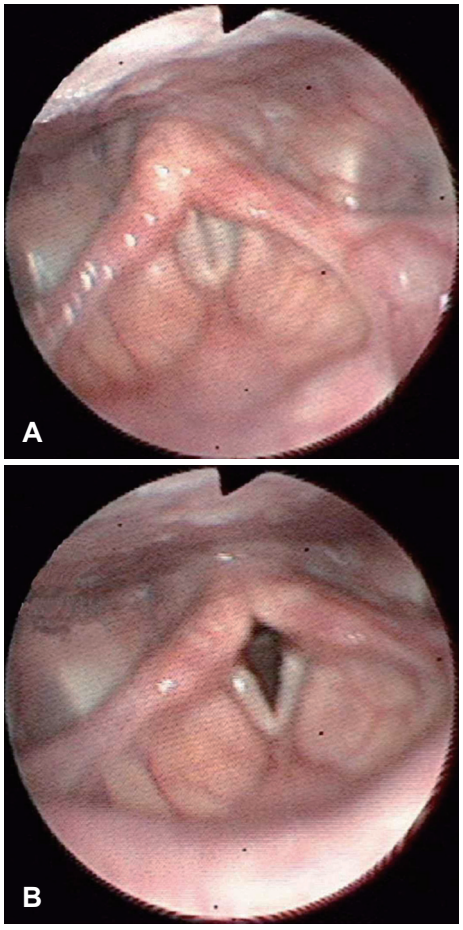

Fig. 2. Flexible laryngoscopic examination demonstrates completely recovery of vocal cord mobility 1 month later. "e" phonation (A), during inspiration (B).

Discussion

GBS, also known as acute idiopathic polyneuritis, is an acute inflammatory demyelinating neuropathy. It is characterized by the rapid onset of ascending, symmetrical motor weakness with loss of deep tendon reflexes.[1-4] GBS is thought to be an autoimmune disease and triggered by a preceding infection in two thirds of all cases. This occurs most frequently 1 to 4 weeks after an upper respiratory tract or gastrointestinal viral infection.[1-3] GBS can progress to cardiac arrhythmia, autonomic dysfunction, and acute respiratory distress which necessitates mechanical ventilation.[2,3] Respiratory muscle weakness is the most serious feature of this disease with mechanical ventilation required by approximately one third of all GSB patients.[1] In our patient also required mechanical ventilation because of respiratory muscle weakness.

The cause of GBS is unclear. Physiologic stressors such as surgery, immunization, and infection have been indicated.[2]

Possible infectious sources include Epstein-Barr virus, infectious hepatitis, cytomegalovirus, and influenza A virus.[2,4] In our patient, Epstein-Barr virus and *Mycoplasma pneumoniae* were identified by serologic tests.

The cause of vocal cord paralysis generally arises from trauma, surgery, tumor compression or neuromuscular disorders.[1,2] In our case, the patient did not have a history of trauma or surgery. In addition, CT images from the skull base to the diaphragm appeared normal. After excluding other causes, we diagnosed bilateral vocal cord paralysis due to GBS.

Typically, GBS affected peripheral nerves, but cranial nerves may be involved as well.[2,3] GBS begins in the cranial nerves in less than 5% of cases. Involvement of cranial nerves occurs in 50% of cases and is frequently bilateral.[5] Facial nerve weakness is the most common cranial neuropathy. The order cranial neuropathies seen, in order of decreasing frequency, include the extraocular muscles (10%) and cranial nerves V, IX, X, XI and XII.[6]

The diagnostic clues for GBS are progressive ascending weakness, lack of deep tendon reflexes, and CSF examination results.[1-4] However, our patient initially presented progressive hoarseness, dysphagia, and aspiration. An initial diagnosis of idiopathic bilateral vocal cord paralysis was made before we explored the cause of bilateral vocal cord paralysis. During the examination, the patient experienced progressive weakness of both upper extremities with diminution of deep tendon reflexes. Based on the clinical symptoms, CSF findings, and electrophysiologic examination, a final diagnosis of bilateral vocal cord paralysis with GBS was made. These finding should alert physicians to the possibility of bilateral vocal cord paralysis as a presenting symptom of GBS.

Treatment of bilateral vocal cord paralysis depends on the patient's symptoms. The need for airway intervention, such as intubation or tracheotomy, is determined by the severity of symptoms. In our case, the patient did not initially exhibit respiratory distress and required no airway intervention because the bilateral vocal cords were fixed in the lateral position with a relatively unobstructed airway.

Treatment of GBS involves plasmapheresis within 2 weeks of the onset of symptoms.[1-3] However, intravenous administration of immunoglobulin has replaced plasmapheresis as the preferred treatment because of its greater convenience and availability,[3,4] as seen in our case. Most patients (80% to 90%) make a full recovery with the recovery phase typically plateauing at several weeks and potentially lasting up to 2 years.[1-3] GBS relapse has been described but is rare.[1]

Our patient had some unique characteristics. First, the bilateral vocal cord was fixed in the lateral position. Due to the position of the bilateral vocal cord, the patient presented with aspiration and a breathy voice instead of stridor and dyspnea; these symptoms differed from ones described in previous reports.[1-4] Second, vocal cord paralysis occurred early, acutely, and was the primary presenting symptom of GBS. Initial predominate respiratory symptoms can mask neurologic signs and may lead to a delay in diagnosing GBS. In conclusion, GBS needs to be considered as possible causes of new onset bilateral vocal cord paralysis. We emphasize that early recognition of atypical presentations of GBS warrants further evaluation and appropriate management.

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