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Case Report

Hoarseness of Voice with respiratory failure: A Clue to Guillain Barre Syndrome

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Abstract

Guillain Barre Syndrome (GBS) is a very rare cause of bilateral vocal cord paralysis. We report an unusual case of GBS in an 80 years old man who came to us in respiratory failure. He was subsequently ventilated and weaned off, but required tracheostomy for bilateral vocal cord paralysis to prevent recurrence of respiratory failure. We highlight the need to consider GBS as possible cause of new onset of hoarseness of voice because of bilateral vocal cord paralysis under appropriate circumstances.

Keywords: *Guillain Barre Syndrome; Hoarseness; Respiratory Failure; Vocal Cord Paralysis*

Introduction

The causes of hoarseness are diverse. Neurogenic vocal cord paralysis is present in only 2.8 to 8% of patients.[1]Here we report a case of bilateral vocal cord paralysis due to Guillain Barre Syndrome (GBS). GBS is an inflammatory demyelinating polyneuropathy. It is autoimmune in nature. It is commonly associated with symmetrical, progressive weakness with decreased or absent deep tendon reflexes, primarily arising in the lower extremities.[2]GBS presenting with bilateral vocal cord paralysis is extremely rare.

Case Report

An 80year-old man, chronic smoker with smoking index of 40 pack years presented with respiratory distress in emergency. He had suffered from respiratory tract infection (RTI) about 1 month ago. He had been having generalized weakness, hoarseness of voice and dyspnea on exertion after recovering from RTI. An emergency visit, 1 week ago was recorded as exacerbation of chronic obstructive lung disease. On examination, pulse rate was 120/min, systolic blood pressure was 70 mmHg, and SpO_2 was 78% on 6-lit/min O_2 via nasal cannula. On auscultation of respiratory system there were decreased breath sounds bilaterally. Examination of the other systems was unremarkable. Arterial blood gas (ABG) analysis showed PaO_2- 28 mmHg, $PaCO_2$- 72 mmHg, HCO_3- 36mmol/L, pH- 7.30. He was intubated and ventilated in view of respiratory failure and impending respiratory arrest. Post intubation the chest radiograph was normal. He was shifted to respiratory intensive care unit for further management. On investigation, his hemoglobin was 12.6 gm/dl and white cell count was 29,700/mm^3. Serum potassium and phosphorus were 3.3 meq/l and 2.3 mg/dl respectively. His enzyme linked immunosorbant assay for human immunodeficiency virus (HIV) was non reactive. His routine biochemical investigations and blood sugar were normal.

The patient was extubated after 2 days. We performed indirect laryngoscopy and fiberoptic bronchoscopy after extubation. It showed that both vocal cords were fixed in the midline (Figure 1). Hence, tracheostomy was done. On inquiry after extubation, he also gave history

of difficulty in swallowing and lower limb weakness since last 3 weeks. Emergency department evaluation 1 week ago possibly mistook stridor with rhonchi and was erroneously diagnosed as COPD. Neurology consultation was taken for weakness. The neurology evaluation showed bilateral lower limb paresis with decreased deep tendon reflexes. He did not have any signs of ophthalmoplegia, ptosis or facial muscle weakness.

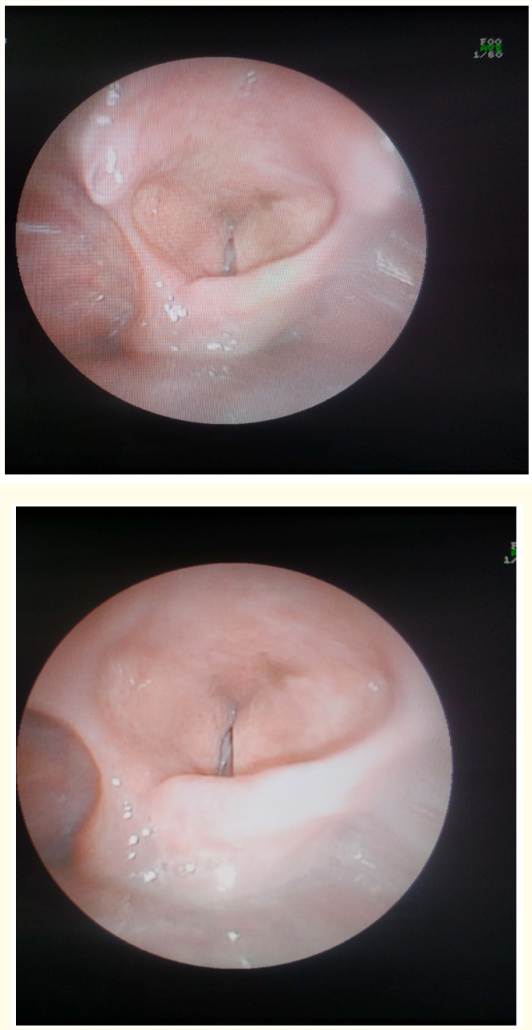

Figure 1a and 1b: *Fiberoptic bronchoscopy showing both vocal cords are fixed in the midline.*

A lumbar puncture was performed. The cerebrospinal fluid analysis showed sugar- 60 mg/dl, protein- 41 mg/dl, total leucocyte count- 100 cells /µl, lactate dehydrogenase- 18 U/l, adenosine deaminase- 0.2 IU/L. Gram stain was negative and culture showed no growth. Non-contrast magnetic resonance imaging (MRI) of brain revealed age related diffuse cerebral atrophy. Electrodiagnostic studies showed that the patient had reduced motor nerve conduction velocities and prolonged distal latencies in bilateral lower limb suggestive of de-

myelinating polyneuropathy. Based on clinical symptoms and electrophysiologic examination, the patient was diagnosed with GBS. The patient was treated with high-dose methylprednisolone (MPS) delivered intravenously at 1 gm/per day for 5 consecutive days. There was insignificant response to MPS. The patient could not be given further treatment as the patient and the relatives were unwilling.

Discussion

GBS is an autoimmune inflammatory demyelinating neuropathy, which includes progressive, symmetrical motor weakness with decreased or absent deep tendon reflexes. Majority of GBS occur 1 - 4 weeks after an acute respiratory or gastrointestinal infection [3]. Diagnosis of GBS is based on Brighton criteria given in table [4]. He satisfied all the criteria except cytoalbuminologic dissociation which is known to occur in half of Asian patients [5].

S. No.	Criteria
1	Bilateral and flaccid weakness of the limbs.
2	Decreased deep tendon reflexes in weak limbs.
3	Monophasic illness pattern and interval between onset and nadir of weakness between 12h and 28 days and subsequent clinical plateau.
4	Electrophysiologic findings consistent with GBS.
5	Absence of an identified alternative diagnosis for weakness.
6	Cytoalbuminologic dissociation (i.e. elevation of CSF protein level above laboratory normal value and CSF total white cell count < 50 cells/μL).

Table: *Brighton criteria.*

The usual pattern of GBS is an ascending paralysis, which typically evolves over hours to a few days. The lower limb weakness was attributed to old age in our patient and remained unnoticed. The legs are usually more affected than the arms. GBS mainly affects peripheral nerves, but the cranial nerves may be involved. Amongst those with cranial nerve involvement facial palsy is present in 50% of affected individuals [6,7]. The lower cranial nerves causing bulbar weakness is seen in only 5% of cases [8].

Cause of bilateral vocal cord paralysis are surgical trauma (44%), malignancies (17%), endotracheal intubation (15%), neurologic disease (12%) and idiopathic causes (12%) [9]. Neurologic causes are myasthenia gravis, amylotropic lateral sclerosis, diabetes mellitus, post polio syndrome, shydrager syndrome, hydrocephalus. We diagnosed bilateral vocal cord paralysis due to GBS, which is very rare, and there are only few reported cases [6,8,10,11]. Treatment of bilateral vocal cord paralysis depends on the patient's symptoms. The need for airway intervention, such as intubation or tracheostomy, is determined by the severity of symptoms. The need for mechanical ventilation is associated with more severe weakness on admission and the presence of facial and/or bulbar weakness [12-14]. In our case mechanical ventilation was required for bilateral vocal cord paralysis. The patients exhibited respiratory distress and required airway intervention because bilateral vocal cords were fixed in the median position with obstructed airway.

The definitive management of GBS requires plasma exchange and Intravenous Immunoglobulin. But it is effective only if given during the first few weeks of disease [15]. The supportive management consists of mechanical ventilation, tracheostomy, nutritional supplementation, monitoring for infectious complications, deep vein thromboprophylaxis, rehabilitation and speech therapy [16]. Corticosteroid therapy is usually ineffective for treating GBS [17]. Our patient could not be given definitive treatment, he was however given the supportive management.

Conclusion

To conclude, bilateral vocal palsy and respiratory failure can be rarely a presenting manifestation of GBS. Detailed history and evaluation thus is necessary in patients with respiratory failure to prevent re-intubation and recurrence of respiratory failure.

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