

CASE REPORT

Bilateral Vocal Cord Paralysis with Acute Ischemic Stroke

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Abstract

Vocal cords receive nerve supply from recurrent laryngeal nerve which is a branch of the vagus nerve. Cerebral motor cortex projects corticobulbar tracts to the motor nucleus (nucleus ambiguus) of the vagus found in medulla. Vagus nerve leaves the skull through jugular foramen and travels through the carotid sheath. The left vagus gives rise to left recurrent laryngeal nerve at the aortic arch, whereas the right vagus gives rise to right recurrent laryngeal nerve at the subclavian artery. Any disruption of this pathway by any entity will cause unilateral vocal cord paralysis. Strokes that affect the motor cortex do not cause unilateral vocal cord paralysis since vagus nerve nucleus receives corticobulbar tract from both sides of the brain. We present an unusual case of bilateral vocal cord paralysis caused by unilateral right insular stroke which is not related to this pathway.

Key words: Vocal cord paralysis, Stroke, Vagus nerve.

Introduction

Bilateral vocal cord paralysis is a serious condition manifested by hoarseness, stridor and aspiration. One of the most common causes of bilateral vocal cord paralysis is recurrent laryngeal nerve damage from thyroid thoracic surgery or compression by a tumor. Brain stem strokes and cortical strokes are rare causes of bilateral and/or unilateral vocal paralysis as the nucleus solitarius of the vagus nerve receives corticobulbar tracts from both sides of the brain (1).

Case report

A ninety two years old African American male with past medical history of hypertension, syphilis, chronic kidney disease, dyslipidemia, and paroxysmal atrial fibrillation presented with sudden onset of difficulty in speaking and left sided weakness. A brain CT scan showed no bleeding and he received t-PA. MRI brain showed acute ischemic stroke involving right insular cortex and right

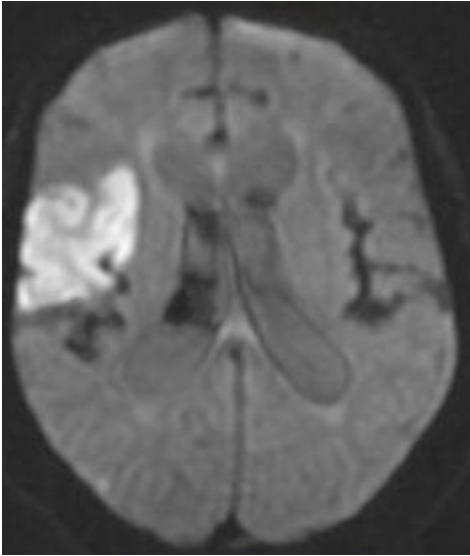


Figure 1. DWI sequence showing diffusion restriction involving right insular and right frontal operculum consistent with acute stroke.

frontal operculum (Figure 1). He initially did well and his symptoms improved. However, on day 5 of admission, the patient developed stridor and hoarseness of voice. The ear, nose and throat (ENT) was consulted and bilateral vocal paralysis was detected. The patient was intubated, T-tube and PEG tube were placed and he was discharged home thereafter.

Discussion

Vocal cords receive nerve supply from recurrent laryngeal nerve which is a branch of the vagus nerve (1). Cerebral motor cortex projects corticobulbar tracts to the motor nucleus (nucleus ambiguus) of the vagus found in medulla. Vagus nerve leaves the skull through jugular foramen and travels through carotid sheath. Left vagus gives rise to left recurrent laryngeal nerve at the aortic arch, while the right vagus gives rise to right recurrent laryngeal nerve at the subclavian artery (1). Any disruption of the presented pathway by any entity will cause unilateral vocal cord paralysis. Strokes that affect the motor cortex will not cause unilateral vocal cord paralysis since vagus nerve nucleus receives corticobulbar tract from both sides of the brain. We presented here an unusual case of bilateral vocal cord paralysis caused by unilateral right insular stroke which is not related to this pathway.

Our patient had unilateral right insular, right frontal operculum stroke and developed bilateral vocal paralysis which may indicate that there could be a dominant vocal

cord center in these areas affected by the stroke. A previously published study in 54 patients with vocal cord paralysis after stroke showed that all cases were unilateral and occurred after few days from the stroke (2). It is well established that the insula has connections with brain structures such as perisylvian structures, pericentral cortex, parietal and temporal cortex (3). A few published cases have shown that patients with insular epilepsies may suffer from recurrent choking spells and respond well to antiepileptic medications (4), choking may occur due to overstimulation of vagus nerve on laryngeal muscle (5). Other published studies showed that insular strokes are associated with increased level of metanephrine and sympathetic hyper activation (6). This could be due to decreased vagus nerve activity (parasympathetic) due to insular stroke which also supports our proposal of a connection between vagus nerve and insular cortex. The present case report increases the awareness of connections between insular cortex and vagus nerve.

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