A 28 year old female, case of thalassemia minor, diagnosed 2 years ago with adenocarcinoma of the colon, presented to the emergency room with progressive neck pain, sudden onset of dysphonia and mild dyspnea. Patient had history of hemicolectomy and had been started on chemotherapy (FOLFOX-5-FU and oxaliplatin) more than a year ago. She had reported upper extremity venous catheterization for the delivery of her chemotherapy. Patient was hemodynamically stable and had mild right neck swelling and tenderness anterior to the right sternocleidomastoid muscle, on perceptual evaluation she had a breathy voice. Flexible naso-pharyngo-laryngoscopy revealed a fixed right vocal fold in the paramedian position with incomplete closure during phonation. Computerized tomography of the neck and chest with intravenous contrast was ordered (Fig. 1.)

**Fig. 1**

What is your diagnosis?

**Diagnosis**

Right Internal Jugular Vein Thrombosis

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Discussion

Vocal fold immobility is described as restricted movement of the vocal folds\(^1\). Its true incidence may be hard to draw in view of the disparities in the work-up of inflicted subjects. The left side is usually more affected than the right side and elderly seem to be more at risk. The clinical presentation varies with the position of the cord and whether one or both sides are affected. A large percentage of patients with vocal fold fixation may be asymptomatic, whereas a few mandate emergency care. In cases of unilateral vocal fold paralysis, patients may present with change in voice quality often described as breathiness. Other phonatory symptoms include loss of power and range, inability to project the voice and fatigability. Patients may also complain of intractable cough, aspiration and throat clearing attributed to glottic insufficiency. Respiratory discomfort and decrease in exercise tolerance are also common complaints. In cases of bilateral paralysis with the vocal cords in the midline, patients may have a normal voice but suffer from stridor and respiratory distress\(^2\).

The evaluation of vocal fold impaired mobility begins with a detailed medical and vocal history, a thorough head and neck assessment, a fiberoptic and or telescopic laryngeal examination, and radiologic evaluation. Contrast-enhanced computed tomography of the head and neck region is usually ordered in order to span the region from the cerebral cortex, throughout the tenth nerve from the base of the skull to the chest\(^1\). When fixation of the cricoarytenoid joint is suspected, laryngeal electromyography is recommended to differentiate mechanical fixation from neural immobility.

The etiology of vocal fold impaired mobility includes, neoplastic lesions, surgery, laryngeal manipulation, viral infections, and idiopathic causes. In a report by D.Myssiorek, surgery is still the leading cause of recurrent laryngeal nerve injury. The most common surgeries are thyroid and parathyroid surgery, carotid endarterectomies, skull base operation, chest surgery, and cervical spine surgeries using the anterior approach. Netterville et al found thyroid surgery to be the most common cause of iatrogenic recurrent laryngeal nerve injury with the incidence being higher when the nerve is not identified intra-operatively\(^4,5\). In a large series of carotid endarterectomies 2.5% were found to have post-operative vocal fold immobility\(^6\). Similar rates have been reported following anterior approach to the cervical spine. Transient cranial neuropathy is also commonly described after skull base surgery, with the vagal nerve commonly affected. As for the neoplastic lesions, non laryngeal tumors account up to 37% of cases of recurrent laryngeal nerve paralysis. These include neoplastic lesions of the thyroid gland, lungs, esophagus and mediastinum\(^7-9\). Other causes include endotracheal intubation, viral etiologies and drug induced paralysis. Still up to 27% of cases of vocal fold impaired mobility remain listed as idiopathic\(^10\).

No previous report has described internal jugular vein thrombosis as the cause of vocal fold impaired mobility. Based on an extensive literature review, this is the first case of internal jugular vein thrombosis presenting with sudden onset of dysphonia and mild dyspnea. The exact mechanism responsible for the impaired mobility of the vocal fold remains unknown. Possible etiologies include vagal nerve compression, thrombosis of the vasa nervosum, perineural inflammation and or viral neuropathy. These remain hypothetical etiologies because of lack of further investigation. Another possible etiology in our case is the repeated intravenous injections and catherizations that could have lead to thrombosis of the internal jugular vein.

Patient was admitted and started on anti-coagulation. Two days later patient was reassessed and found to have significant improvement in her voice quality and breathing. Repeated fiberoptic naso-pharyngo-laryngoscopy revealed normal vocal fold mobility. The rapid improvement in the patient’s condition substantiates the fact that internal jugular vein thrombosis is most likely to be the etiology.
References
