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REVIEW ARTICLE

Losing Your Voice: Etiologies and Imaging Features of Vocal Fold Paralysis

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ABSTRACT

Neurogenic compromise of vocal fold function exists along a continuum encompassing vocal cord hypomobility (paresis) to vocal fold immobility (paralysis) with varying degrees and patterns of reinnervation. Vocal fold paralysis (VFP) may result from injury to the vagus or the recurrent laryngeal nerves anywhere along their course from the brainstem to the larynx. In this article, we review the anatomy of the vagus and recurrent laryngeal nerves and examine the various etiologies of VFP. Selected cases are presented with discussion of key imaging features of VFP including radiologic findings specific to central vagal neuropathy and peripheral recurrent nerve paralysis.

Key words: Computed tomography, imaging, paralysis, recurrent laryngeal nerve, vagus nerve, vocal cords, vocal-fold-paralysis

INTRODUCTION

Vocal fold paralysis (VFP) is the immobilization of the true vocal fold by neural injury (ipsilateral vagus or recurrent laryngeal nerve dysfunction) or by mechanical fixation of the vocal fold due to neoplastic invasion of the thyroarytenoid muscle, or cricoarytenoid joint fixation from joint ankylosis or posterior glottic scar formation.[1] Clinically, these patients will often present with hoarseness, dysphonia, breathy voice, or aspiration. However, as many as 30-50% of patients are clinically asymptomatic[2] and the presence of VFP may be only incidentally detected. In many such asymptomatic cases, a slow-growing malignancy with secondary involvement of the vagus or recurrent laryngeal nerves may result in computed tomography (CT) imaging findings that precede the clinical manifestation of VFP.[3] Alternatively, clinical identification of vocal fold immobility may prompt CT imaging to identify occult lesions along the vagus or recurrent laryngeal nerve. Hence, once VFP has been identified in symptomatic or asymptomatic patients, every effort should be made to determine the underlying etiology of the VFP by carefully evaluating the course of the vagus and recurrent laryngeal nerves using appropriate imaging. With recent improved imaging techniques, a causative reason for VFP is often identified, resulting in a decrease in the incidence of cases labeled "idiopathic" after clinical examination.[3-5]

This article reviews pertinent anatomy and highlights key imaging findings of VFP. In addition, we describe a

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systematic imaging approach that seeks to identify the main etiologies resulting in VFP, illustrating them with typical cases and outlining potential imaging pitfalls.

ANATOMY

The larynx is divided into supraglottic, glottis, and subglottic divisions by two pairs of horizontal mucosal folds – the false (vestibular) and true vocal folds. The false vocal folds are above and lateral to the true vocal folds and are formed by the mucosal surfaces of the laryngeal vestibule. The true vocal folds are involved in phonation and extend from the arytenoid cartilage posteriorly to the thyroid cartilage anteriorly. They consist of the thyroarytenoid and the vocalis muscle (the most medial portion of the thyroarytenoid muscle) covered with mucosa. In addition, the vocal ligament, the most superior extent of the conus elasticus, consists of bands of yellow elastic tissue within the true vocal folds, which are attached anteriorly to the angle of the thyroid cartilage, and posteriorly to the vocal process of the arytenoid.

The larynx is innervated by sensory and motor branches of the vagus nerve. The vagus nerve originates from the nucleus ambiguous, exits the medulla, crosses the perimedullary cistern, and leaves the skull base though the pars vasularis of the jugular foramen anterior to the jugular vein.[4,6,7] The inferior/nodose ganglion of the vagus nerve, located immediately inferior to the jugular foramen, gives rise to the superior laryngeal nerve that divides into the internal laryngeal nerve (which provides sensory innervation to the larynx above the vocal folds) and the external laryngeal nerve that innervates the cricothyroid muscle.[4,6,7] More recent evidence points to extensive terminal arborization of both the recurrent and superior laryngeal nerves, leading to variable contralateral innervation. Additionally, Galen's anastomosis between the ipsilateral recurrent and superior laryngeal nerve is present variably in up to 50% of patients.[8]

In the neck, the vagus nerve descends within the carotid sheath in the post-styloid parapharyngeal space (carotid space), posterolateral to the internal carotid artery and posteromedial to the internal jugular vein.[3,7] The right recurrent laryngeal nerve arises from the right vagus nerve at the cervicothoracic junction, passes posteriorly around the right subclavian artery and ascends out of the thorax along the apical lobe pleura. It passes posterior to the common carotid artery to recur within the thoracoesophageal groove at the level of cricothyroid joint. In 1% of cases, the right recurrent laryngeal nerve arises from the vagus nerve at the level of the thyroid and is at risk of injury during thyroid surgery.[4] The left recurrent laryngeal nerve arises from the left vagus nerve at the level of the aortic arch in the mediastinum, loops under the aortic arch at the aorto-pulmonary window posterior to the ligamentum arteriosum, and exits the thorax to recur in the left thoracoesophageal groove.

Both recurrent laryngeal nerves supply the intrinsic muscles of the larynx with variable, though suspected limited motor afferents supplied by the internal branch of the superior laryngeal nerve. The exception in the intrinsic laryngeal musculature is the cricothyroid, which is wholly supplied by the external branch of the superior laryngeal nerve. The recurrent laryngeal nerve provides sensory innervation to the larynx below the vocal folds.

EVALUATION OF VOCAL FOLD PARALYSIS

A complete clinical history, including alcohol and smoking history (to determine risk factors for neoplasms), prior trauma, surgeries, prolonged intubations, and upper or lower respiratory infections should be undertaken to determine the possible etiology of VFP. A thorough head and neck physical examination should include evaluation of the cranial nerves with particular focus on the vagus nerve.

Direct evaluation with flexible laryngoscopy will determine if there is grossly normal or asymmetric movement of vocal folds. Flexible or rigid videostroboscopy can rule out the presence of mucosal or submucosal lesions with deeper extension, and visualize more subtle asymmetry of mucosal wave, indicative of unilateral vocal fold paresis [Figure 1 a-c]. Clinically, the affected

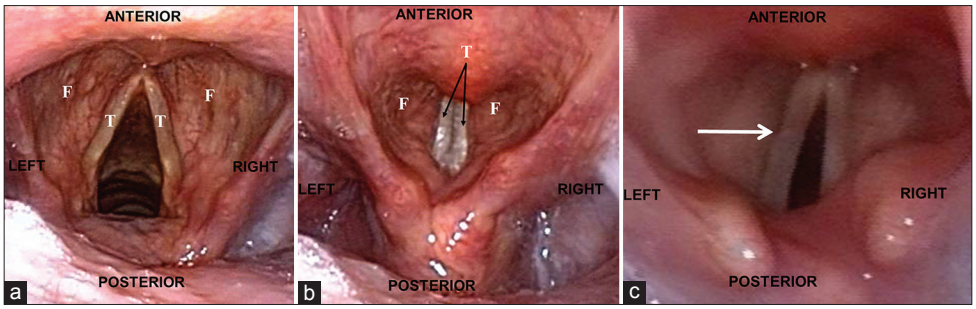

Figure 1: Flexible distal chip laryngoscopy shows normal vocal folds at (a) inspiration and (b) phonation, (c) A different patient with left vocal fold paralysis (VFP): Note the lateral position of the left vocal fold (arrow). T: True vocal folds, F: False vocal folds.

vocal fold will be either immobile or demonstrate decreased abduction or adduction. Based on contrast laryngography and cine studies, Landman[9] described at least 10 findings suggestive of recurrent laryngeal nerve paralysis including dilated ipsilateral laryngeal ventricle, incomplete abduction of both the true and false vocal cords in quiet breathing, flattened ipsilateral subglottic arch, interarytenoid notch displaced to the normal side during phonation, anterior and inferior position of the paralyzed arytenoid cartilage, concavity of the free edge of the paralyzed true vocal fold, dilated ipsilateral pyriform sinus, flattened ipsilateral lateral wall of the vestibule, and higher or lower position of the paralyzed vocal fold compared to the normal fold, depending on the degree of synkinetic reinnervation. Laryngeal electromyography (EMG) is helpful in confirming neuropathy, determining relative duration of neuropathy, and prognosticating return of function.[10,11] In the absence of iatrogenic causes (intubation, surgery placing the RLN/vagus at risk), central neurologic symptoms, or more rarely, systemic symptoms, additional evaluation with appropriate imaging is performed.

IMAGING OF VFP

Although both CT and Magnetic Resonance Imaging (MRI) may be used to assess etiology of VFP, the fast acquisition time and high-resolution capabilities of CT make it the imaging modality of choice to evaluate features consistent with VFP, if direct visualization has not been performed. Quiet respiration is recommended for CT evaluation of the larynx as the vocal cords are imaged in an intermediate position.[3] Contrast-enhanced CT should extend at least from the posterior fossa/skull base through at least the aorto-pulmonary window in the case of left VFP, and to the thoracic inlet in the case of right VFP, to ensure that the entire course of the vagus and recurrent laryngeal nerves is evaluated. Axial images provide the most accurate information, with coronal and sagittal reformats aiding the determination of VFP. Figure 2a demonstrates the normal CT appearance of the true vocal folds. The level of the true vocal folds is identified on CT when both arytenoid and cricoid cartilages are seen. Figure 2b demonstrates the normal CT appearance of the pyriform sinuses and aryepiglottic folds.

Many of the findings of VFP described by Landman[9] on contrast laryngography and cine studies can be commonly identified on CT images of the neck. The three most reliable findings of VFP observed in more than 75% of cases in a study by Chin et al.,[12] include dilation of the ipsilateral pyriform sinus [Figure 3a], thickening and medial position of the ipsilateral aryepiglottic fold [Figure 3a], and dilated ispilateral laryngeal ventricle –also known as the "Sail Sign" [Figure 3b]. Three additional findings, anterio-medial position of the ipsilateral arytenoid cartilage [Figure 3c], atrophy of the ipsilateral posterior cricoarytenoid muscle first described by Romo et al.,[13] [Figure 3d], and atrophy with fatty replacement

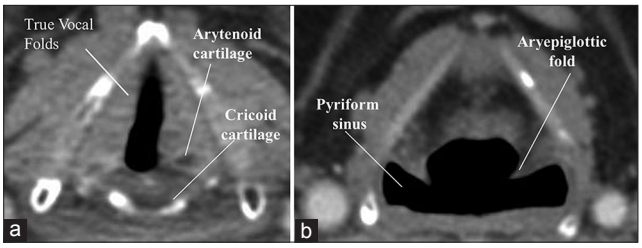

Figure 2: (a) Axial CT image shows the normal appearance of the true vocal folds: The level of the true vocal folds is confirmed with visualization of the crico-arytenoid joints, (b) Axial CT shows the normal symmetric appearance of the aryepiglottic folds and pyriform sinuses.

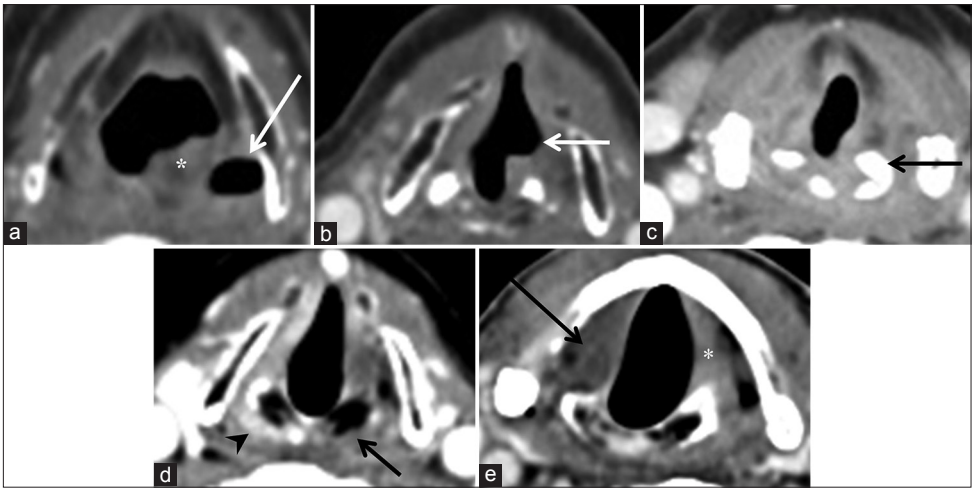

Figure 3: Axial CT images demonstrate imaging features of vocal fold paralysis (VFP): (a) Axial CT image shows dilation of the left pyriform sinus (arrow), medial position of the thickened left ipsilateral aryepiglottic fold (asterisk), (b) Axial CT image reveals dilated left laryngeal ventricle (arrow), (c) Axial CT image shows anteromedial deviation of the left arytenoid cartilage (arrow) and (d) axial CT image in a patient with left VFP demonstrates atrophy of the left posterior cricoarytenoid muscle (arrow) compared to the contralateral normal posterior cricoarytenoid muscle (arrowhead), (e) Axial CT at the level of the true vocal folds demonstrates fatty atrophy of the ipsilateral thyroarytenoid muscle (arrow) in a patient with right VFP. Contrast this with the normal, nonparalyzed left thyroarytenoid muscle (asterisk).

of the ipsilateral thyroarytenoid muscle [Figure 3e], form part of the constellation of imaging features of VFP.

An imaging distinction of central vagal neuropathy from peripheral neuropathy in patients with VFP is important because the differential diagnoses and management of these two conditions differ.[12] Central vagal neuropathy implies involvement of both the pharyngeal plexus and the recurrent laryngeal nerves. The pharyngeal plexus is made up of branches of cranial nerves 9, 10, and 11. Therefore, patients with central vagal neuropathy may present with atrophy of the ipsilateral pharyngeal constrictor muscle (innervated by cranial nerve 10) with consequent ballooning of the oropharyngeal or hypopharyngeal walls or other signs of pharyngeal plexus involvement such as atrophy of the trapezius (innervated by cranial nerve 11) in addition to the classic imaging features of recurrent laryngeal nerve palsy [Figure 4].

On fluorodeoxyglucose (FDG) positron emission tomography (PET)-CT images, unilateral VFP may demonstrate an asymmetric increase in FDG uptake in the normal vocal fold, with no uptake in the paralyzed vocal fold [Figure 5] and should not be mistaken for abnormal uptake of glucose secondary to malignancy. To avoid misinterpretations when presented with increased FDG uptake in one vocal fold, FDG PET-CT imaging findings should be correlated with a history of VFP with side of increased metabolic activity on FDG PET-CT confirmed to be opposite to the side of VFP noted on CT. It is important to remember that patients who have undergone injection augmentation of the paralyzed vocal fold with Teflon may demonstrate increased FDG accumulation in the treated vocal fold secondary to a granulomatous reaction incited by Teflon, which may mimic a neoplasm or metastasis.[3]

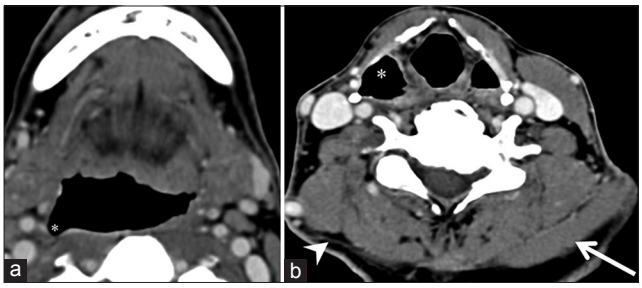

Figure 4: A 69-year-old woman with right VFP due to a known paraganglioma within the right jugular foramen: (a) Axial contrast enhanced CT demonstrates atrophy of the right pharyngeal constrictor muscle with subsequent ballooning of the oropharyngeal airway (asterisk) suggesting long standing right cranial nerve 10 involvement, (b) Axial contrast enhanced CT in the same patient demonstrates asymmetric dilation of the right pyriform sinus (asterisk) compared to the left consistent with right VFP. In addition, there is atrophy of the right trapezius muscle (arrowhead) suggesting long standing right cranial nerve 11 involvement. Compare this to the normal left trapezius muscle (arrow). In patients with central vagal neuropathy as in this patient, there is an involvement of the ipsilateral pharyngeal plexus, which consists of branches of cranial nerves 9, 10, and 11.

Finally, patients with malignant infiltration of the vocal fold may show increased FDG avidity in the affected vocal fold. In these cases, direct visualization to determine presence of malignancy and CT imaging findings showing fullness of the affected vocal fold will help confirm that the increased FDG avidity is secondary to malignancy.

ETIOLOGIES OF VFP

There are a number of etiologies of VFP including surgery/trauma, infectious/inflammatory conditions (e.g. Lyme, syphilis, and post viral), radiation, myasthenia gravis, neoplasms, toxicity (e.g., vinca alkaloid and alcohol), and vascular conditions. A large number of cases have no identifiable cause and have been deemed idiopathic, although as previously mentioned, with recent improved imaging techniques, the incidence of truly "idiopathic" cases has decreased.[3,4]

An abnormality, anywhere along the course of the vagus or recurrent laryngeal nerves, can result in VFP. Hence, a systematic imaging approach that evaluates the course of the nerves from the medulla to the aortopulmonary window (for left VFP) and from the medulla to the origin of the right subclavian artery (for right VFP) should be performed in all cases of VFP where the etiology of VFP remains unclear after clinical evaluation. For the purposes of organization and discussion, we consider etiologies affecting three segments of the nerves: At the level of the brainstem and skull base, in the suprahyoid neck, and in the infrahyoid neck and mediastinum (in cases of left VFP).

Brainstem and skull base etiologies

Vascular, neoplastic, and inflammatory lesions are the most common pathologies affecting the vagus nerve at

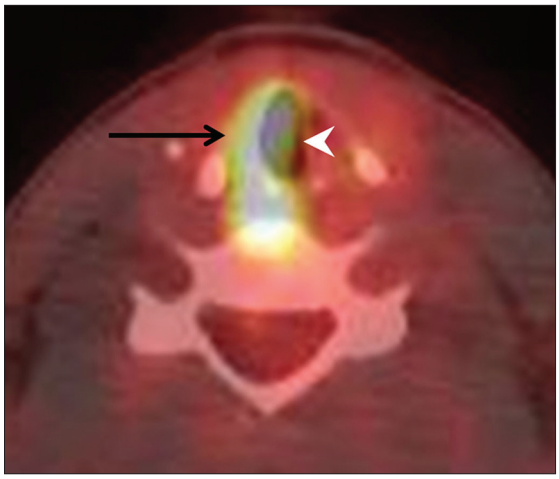

Figure 5: A 50-year-old woman with left recurrent laryngeal nerve palsy: Fused PET-CT image at the level of the vocal fold demonstrates subtle medialization of the left posterior vocal cord (arrowhead) with increased muscle activity within the normal right side (arrow), findings that are typical for left VFP.

the level of the medulla. Occlusion of the posterior inferior cerebellar arteries may result in a lateral medullary infarct (Wallenburg syndrome) with consequent involvement of the exiting ipsilateral vagus nerve resulting in ipsilateral VFP.[14,15] Hypertension and vascular malformations may occasionally cause hemorrhagic foci in the medulla, and hence, central vagal neuropathy.[7] The most common neoplasms involving the medulla, and hence, compromising the nucleus ambiguous, are metastases, gliomas, and lymphomas.[7] Although demyelinating diseases such as multiple sclerosis more commonly affect the pons and midbrain, involvement of the medulla may result in vagal neuropathy with resultant VFP.

Tumors, infections, and trauma may involve the vagus nerve as it traverses the skull base. Paragangliomas, nerve sheath tumors such as schwannomas [Figure 6] and meningiomas located in the vicinity of the jugular foramen can compress the vagus nerve and result in VFP.[16] Primary tumors or metastases involving adjacent structures such as the petrous apex and the clivus can extend into the jugular foramen with resultant lower cranial neuropathies [Figure 7].

Suprahyoid neck

The vagus nerve traverses the carotid space within the carotid sheath in the suprahyoid neck, and a wide range of abnormalities including benign and malignant tumors, inflammatory process, and vascular lesions can result in vagal nerve involvement with subsequent VFP. As in the region of the jugular foramen, paragangliomas/glomus vagale [Figure 8], and vagal schwannomas are slow growing tumors that originate from the vagus nerve in its course through the post-styloid parapharyngeal space and are associated with vagal nerve palsies in the late clinical course of the disease.[7,16] Other potential etiologies of VFP are dissection of the internal carotid artery and internal carotid artery aneurysms with mechanical compression of and/or impairment of blood supply to the vagus nerve as it traverses within the carotid sheath.[7,17]

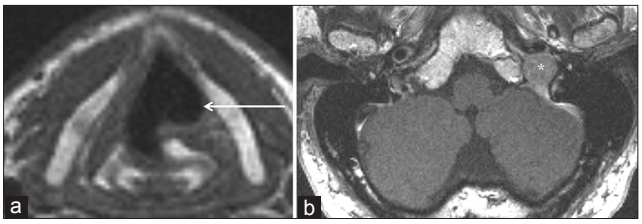

Figure 6: A 68-year-old woman with left VFP: (a) Axial T1W MRI demonstrates dilated left laryngeal ventricle (arrow), (b) Axial T1W MRI in the same patient demonstrates a jugular foramen schwannoma (asterisk) as the cause of the left VFP.

Infrahyoid neck and mediastinum

The close proximity of the recurrent laryngeal nerve to the esophagus, trachea, and thyroid in the trachea-esophageal groove makes it vulnerable to injury secondary to pathologies involving these structures. Squamous cell carcinomas and other malignancies of the esophagus are extrathyroid causes of VFP.[18,19] Thyroid etiologies that result in recurrent laryngeal nerve palsies include extracapsular extension of thyroid malignancies [Figure 9] and thyroid surgery. Until recently, thyroid surgery was a common iatrogenic cause of recurrent laryngeal nerve paralysis, although its incidence has declined with improved operative techniques.[1] Extension of tumors from the trachea and other tracheal abnormalities such as diverticuli [Figure 10] can impinge on the recurrent laryngeal nerve and cause VFP. Recurrent laryngeal nerve palsy may be a complication of iatrogenic causes such as prolonged endotracheal intubation and anterior cervical discectomy as well as fractures secondary to non-iatrogenic cervical trauma.

The left recurrent laryngeal nerve is longer (approximately 12 cm from the aortic arch to cricothyroid joint) than the right recurrent laryngeal nerve (approximately 5-6 cm from the subclavian to cricothyroid joint)[20] and has a longer intrathoracic course, which makes it more vulnerable to

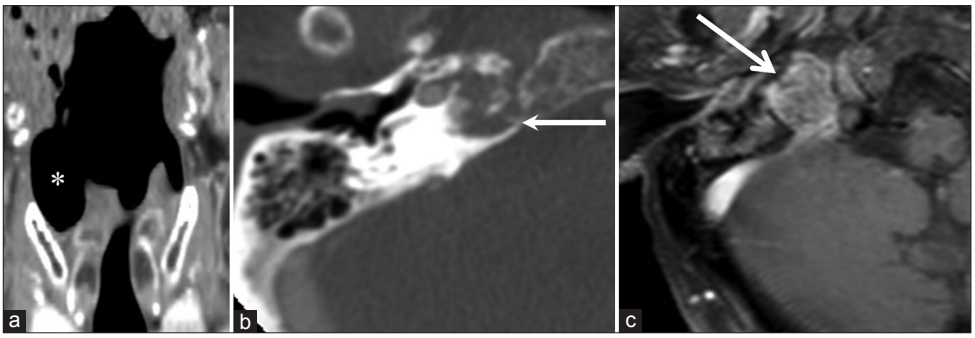

Figure 7: A 40-year-old male with right VFP: (a) Coronal CT image demonstrates a dilated right pyriform sinus (asterisk) in a patient with known right VFP, (b) Axial CT image reformatted with bone windows demonstrates a lytic lesion of the right petrous apex extending into the right jugular foramen (arrow), (c) Axial T1W MRI demonstrates a right petrous apex lesion with enhancement post gadolinium contrast (arrow). The lesion was determined to be a chondrosarcoma of the right petrous apex extending into the right jugular foramen with resultant vagal neuropathy and right VFP.

injury secondary to mediastinal abnormalities.[3,19] A study by Song et al.,[19] demonstrated that in patients with VFP secondary to thoracic diseases, left VFP was 1.75 times more frequent than right VFP. Isolated left recurrent laryngeal nerve involvement may occur due to invasion or compression of the nerve at the aortopulmonary window by primary lung carcinoma [Figure 11]. Other etiologies resulting in left recurrent laryngeal nerve paralysis in the mediastinum include aortic aneurysm [Figure 12], pulmonary artery enlargement secondary to hypertension, and cardiomegaly.[21] Mediastinal lymphadenopathy due to a variety of causes including metastases, tuberculosis, sarcoidosis or pneumoconiosis can cause left recurrent nerve compression, if it occurs at the aortopulmonary window, and right recurrent laryngeal nerve compression, if it involves mediastinal lymph nodes adjacent to the right subclavian artery.

IMAGING PITFALLS

An immobile vocal fold noted on clinical examination, or a medialized vocal fold on CT imaging, may not necessarily be secondary to vagal or recurrent laryngeal nerve injury. Laryngeal and pyriform sinus carcinomas may result in medialization of the vocal fold due to direct invasion of the thyroarytenoid, immobility due to invasion of the cricoarytenoid joint or immobility from secondary cartilagenous sclerosis and resultant joint fixation [Figure 13a]. In these instances, the tumor-infiltrated vocal fold appears thickened and may demonstrate mass-like enhancement. Associated findings of lymphadenopathy and invasion of adjacent cartilage by tumor may help make the distinction between vocal fold immobility and medialization due to tumor versus true VFP due to neural injury.[3]

Vocal fold injection has re-gained popularity as first line treatment for vocal fold augmentation in patients with VFP.[22] These injection materials include durable/permanent materials such as autologous fat, calcium hydroxylapatite, polydimethylsiloxane, and historically Teflon, as well as temporary injection materials such as hyaluronic acid, carboxymethylcellulose, and collagen based products.[22-24] These materials may occasionally be misinterpretated as laryngeal masses or unexpected foreign bodies.[3,25] Familiarity with the CT appearance of the larynx post vocal fold injection or medialization allows accurate radiologic evaluation and prevents the misinterpretation of post-treatment changes as pathology [Figure 13b].

CONCLUSION

Familiarity with the various imaging features of VFP and employing a systematic imaging approach aimed at identifying the underlying cause has resulted in a decrease in the incidence of cases labeled "idiopathic" post

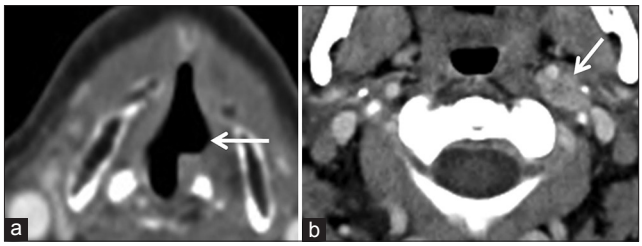

Figure 8: A 52-year-old woman with new onset hoarseness: (a) Contrast-enhanced axial CT image demonstrates radiologic findings of left VFP as evidenced by dilated left laryngeal ventricle (arrow), (b) Imaging along the course of left vagus nerve demonstrates a uniformly enhancing mass within the left post-styloid parapharyngeal space consistent with a vagal paraganglioma/glomus vagale (arrow).

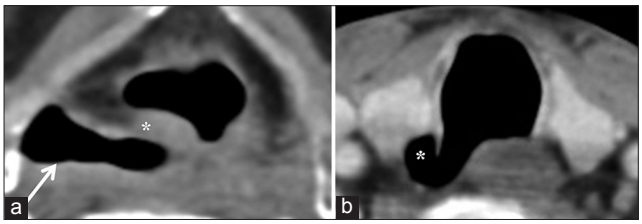

Figure 10: A 46-year-old woman with hoarseness: (a) Axial CT at the level of the hypopharynx demonstrates dilated right pyriform sinus (arrow) and thickening and medial position of the right aryepiglottic fold consistent with right VFP, (b) Contrast enhanced axial CT demonstrates a right sided tracheal diverticulum (asterisk) which likely causes compressive effects on the right recurrent laryngeal nerve and right VFP.

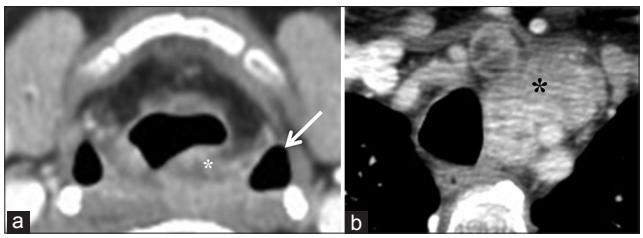

Figure 9: A 70-year-old woman with thyroid cancer: (a) Axial CT image at the level of the hypopharynx demonstrates dilated left pyriform sinus (arrow) and thickened, medialized left aryepiglottic fold (asterisk) consistent with left VFP, (b) Axial CT image demonstrates a large left thyroid lobe mass (asterisk) extending posteriorly and causing compressive effect on the left recurrent laryngeal nerve with resultant left VFP.

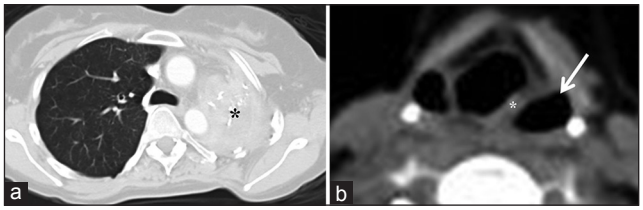

Figure 11: A 53-year-old woman with recurrence of lung carcinoma at the site of prior left pneumonectomy, which has resulted in compression of the left recurrent laryngeal nerve in its course at the aortopulmonary window. (a) Axial contrast enhanced CT viewed with lung windows demonstrates a large mass (asterisk) within the left upper lobe pneumonectomy bed. Hyperdensities within the mass represent prior surgical material and dystrophic calcification, (b) Axial CT at the level of the hypopharynx demonstrates dilated left pyriform sinus (asterisk) and medial position of the left aryepiglottic fold (arrow), findings suggestive of left VFP.

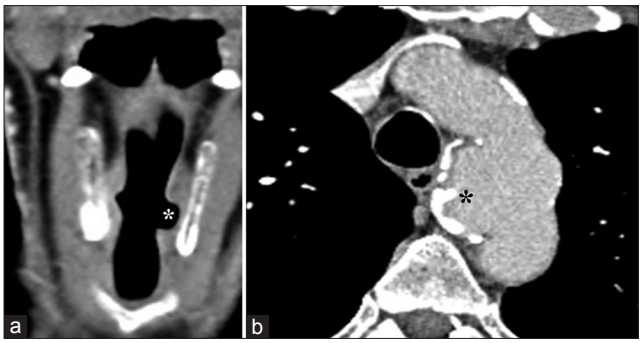

Figure 12: A 71-year-old man with left VFP: (a) Coronal CT image demonstrates dilated left laryngeal ventricle (asterisk) in a patient with left VFP, (b) Axial CT image demonstrates an aortic arch aneurysm (asterisk) causing compression of the left recurrent laryngeal nerve resulting in the left VFP.

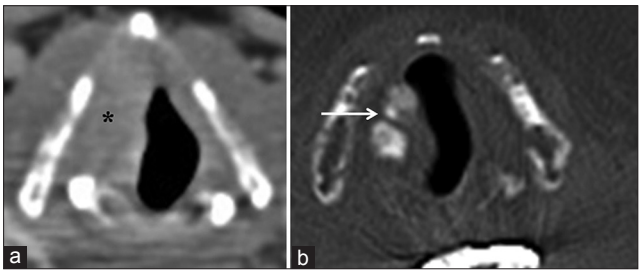

Figure 13: Tumor and post treatment changes that can mimic VFP: (a) Axial contrast-enhanced CT at the level of the true vocal folds demonstrates fullness and medial position of right vocal fold secondary to squamous cell carcinoma infiltration of the right vocal fold (asterisk), (b) Post injection of Teflon for treatment of right VFP, hyperattenuating material (arrow) seen in the right vocal fold.

clinical evaluation and can assist in guiding specific and appropriate treatment.

REFERENCES

1. Benninger M, Gillen J, Altman J. Changing etiology of vocal fold immobility. Laryngoscope 1998;108:1346-50.
2. Collazo-Clavell ML, Gharib H, Maragos NE. Relationship between vocal cord paralysis and benign thyroid disease. Head Neck 1995;17:24-30.
3. Paquette CM, Manos DC, Psooy BJ. Unilateral vocal cord paralysis: A review of CT findings, mediastinal causes, and the course of the recurrent laryngeal nerves. Radiographics 2012;32:721-40.
4. Myssiorek D. Recurrent laryngeal nerve paralysis: Anatomy and etiology. Otolaryngol Clin North Am 2004;37:25-44.
5. Robinson S, Pitkaranta A. Radiology findings in adult patients with vocal fold paralysis. Clin Radiol 2006;61:863-7.
6. Curtin HD. The larynx. In: Som PM, Curtin HD, eds. Head and neck imaging 4th ed. St Louis: Mosby;2003:1595–1699.
7. Policeni BA, Smoker WR. Pathologic conditions of the lower cranial nerves IX, X, XI, and XII. Neuroimaging Clin N Am 2008;18:347-68.
8. Naidu L, Ramsaroop L, Partab P, Satyapal K. Galen's "anastamosis" revisited. Clin Anat 2012;25:722-8.
9. Landman GHM. Laryngography and Cinelaryngography. Experta Medica Monographs. Amsterdam: Experta Medica Foundation; 1970.
10. Rubin AD, Sataloff RT. Vocal fold paresis and paralysis. Otolaryngol Clin North Am 2007;40:1109-31.
11. Statham MM, Rosen CA, Nandedkar SD, Munin MC. Quantitative laryngeal electromyography: Turns and amplitude analysis. Laryngoscope 2010;120:2036-41.
12. Chin SC, Edelstein S, Chen CY, Som PM. Using CT to localize side and level of vocal cord paralysis. AJR Am J Roentgenol 2003;180:1165-70.
13. Romo LV, Curtin HD. Atrophy of the posterior cricoarytenoid muscle as an indicator of recurrent laryngeal nerve palsy. AJNR Am J Neuroradiol 1999;20:467-71.
14. Tatu L, Moulin T, Bogousslavsky J, Duvernoy H. Arterial territories of human brain: Brainstem and cerebellum. Neurology 1996;47:1125-35.
15. Aydogdu I, Ertekin C, Tarlaci S, Turman B, Kiylioglu N, Secil Y. Dysphagia in lateral medullary infarction (Wallenberg's syndrome): An acute disconnection syndrome in premotor neurons related to swallowing activity? Stroke 2001;32:2081-7.
16. Rao AB, Koeller KK, Adair CF. From the archives of the AFIP. Paragangliomas of the head and neck: Radiologic-pathologic correlation. Armed Forces Institute of Pathology. Radiographics 1999;19:1605-32.
17. Mokri B, Silbert PL, Schievink WI, Piepgras DG. Cranial nerve palsy in spontaneous dissection of the extracranial internal carotid artery. Neurology 1996;46:356-9.
18. Furukawa M, Furukawa MK, Ooishi K. Statistical analysis of malignant tumors detected as the cause of vocal cord paralysis. ORL J Otorhinolaryngol Relat Spec 1994;56:161-5.
19. Song SW, Jun BC, Cho KJ, Lee S, Kim YJ, Park SH. CT evaluation of vocal cord paralysis due to thoracic diseases: A 10-year retrospective study. Yonsei Med J 2011;52:831-7.
20. Weisberg NK, Spengler DM, Netterville JL. Stretch-induced nerve injury as a cause of paralysis secondary to the anterior cervical approach. Otolaryngol Head Neck Surg 1997;116:317-26.
21. Jacobs CJ, Harnsberger HR, Lufkin RB, Osborn AG, Smoker WR, Parkin JL. Vagal neuropathy: Evaluation with CT and MR imaging. Radiology 1987;164:97-102.
22. Mallur PS, Rosen CA. Vocal fold injection: Review of indications, techniques, and materials for augmentation. Clin Exp Otorhinolaryngol 2010;3:177-82.
23. Mallur PS, Morrison MP, Postma GN, Amin MR, Rosen CA. Safety and efficacy of carboxymethylcellulose in the treatment of glottic insufficiency. Laryngoscope 2012;122:322-6.
24. Sulica L, Rosen CA, Postma GN, Simpson B, Amin M, Courey M, et al. Current practice in injection augmentation of the vocal folds: Indications, treatment principles, techniques, and complications. Laryngoscope 2010;120:319-25.
25. Mukherji SK, Weadock WJ. Imaging of the post-treatment larynx. Eur J Radiol 2002;44:108-19.

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Perdiendo tu voz: etiologías y características por Imagen de la parálisis de cuerdas vocales

ABSTRACT

El compromiso neurogénico de la función de las cuerdas vocales engloba desde la hipomotilidad de las cuerdas vocales (paresia) hasta la inmovilidad de las mismas (parálisis) con varios grados y patrones de reinervación. La parálisis de cuerdas vocales (PCV) puede ser el resultado de un daño al nervio vago o al laríngeo recurrente en cualquier sitio de su tracto desde el puente hasta la laringe. En este artículo revisamos la anatomía del vago y del nervio laríngeo recurrente y examinamos las diversas etiologías de la PCV. Presentamos casos selectos con una discusión de las principales características por imagen incluyendo hallazgos radiológicos específicos a la neuropatía vagal y parálisis del nervio recurrente periférico.