

Imaging evaluation of vocal cord paralysis*

Avaliação por imagem da paralisia de pregas vocais

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Abstract Vocal cord paralysis is a common cause of hoarseness. It may be secondary to many types of lesions along the cranial nerve X pathway and its branches, particularly the laryngeal recurrent nerves. Despite the idiopathic nature of a great number of cases, imaging methods play a very significant role in the investigation of etiologic factors, such as thyroid and esophagus neoplasias with secondary invasion of the laryngeal recurrent nerves. Other conditions such as aortic and right subclavian artery aneurysms also may be found. The knowledge of local anatomy and related diseases is of great importance for the radiologist, so that he can tailor the examination properly to allow an appropriate diagnosis and therapy planning. Additionally, considering that up to 35% of patients with vocal cord paralysis are asymptomatic, the recognition of radiological findings indicative of this condition is essential for the radiologist who must warn the referring physician on the imaging findings. In the present study, the authors review the anatomy and main diseases related to vocal cord paralysis, demonstrating them through typical cases evaluated by computed tomography and magnetic resonance imaging, besides describing radiological findings of laryngeal abnormalities indicative of this condition.

Keywords: Vocal cord; Paralysis; Imaging.

Resumo Paralisia das pregas vocais é causa frequente de rouquidão, podendo ser secundária a várias lesões ao longo do trajeto do X par craniano e seus ramos, particularmente os nervos laríngeos recorrentes. Apesar de grande parte dos casos ser idiopática, os métodos de imagem são muito importantes na pesquisa de fatores etiológicos, tais como lesões neoplásicas da tireoide e esôfago com invasão secundária dos nervos laríngeos recorrentes. Além destas, outras anormalidades como aneurismas do arco aórtico e da artéria subclávia direita podem ser encontradas. É fundamental que o radiologista conheça a anatomia pertinente a esta região e as principais afecções que podem ocorrer, para que o estudo seja corretamente planejado, auxiliando o diagnóstico e o planejamento terapêutico. Além disso, como até 35% dos casos de paralisia da prega vocal são assintomáticos, o conhecimento dos sinais radiológicos que indicam esta condição é indispensável, cabendo ao radiologista alertar o médico assistente sobre os achados do exame. Neste trabalho realizamos uma revisão da anatomia e das principais doenças responsáveis pela paralisia de cordas vocais, demonstrando-as por meio de estudos de tomografia computadorizada e ressonância magnética de casos típicos. Mostramos, também, as alterações radiológicas próprias da laringe que indicam a presença de paralisia das pregas vocais.

Unitermos: Cordas vocais; Paralisia; Imagem.

Garcia MM, Magalhães FP, Dadalto GB, Moura MVT. Imaging evaluation of vocal cord paralysis. *Radiol Bras.* 2009;42(5):321-326.

INTRODUCTION

Vocal cord paralysis is a frequent cause of hoarseness, and may be secondary to several conditions along the vagus nerve

(paired cranial nerve X) pathway and its branches. The recognition of specific radiological findings, as well as the knowledge on the anatomy and diseases that may affect the paired cranial nerve X are extremely important for the choice and programming of the best imaging method to be utilized, and for an appropriate interpretation of images.

In the present study, the authors sought to demonstrate radiological findings indicative of vocal cord paralysis. Additionally, the strategies for evaluation of computed tomography (CT) and magnetic reso-

nance imaging (MRI) studies were focused in the research of the main causes of the condition, illustrating them with typical cases.

ANATOMY

The vagus nerve is the longest of the cranial nerves, extending from the brainstem to the abdomen⁽¹⁾. It originates from four nuclei in the bulb, three of them converging in the basal cistern to form a single nerve that emerges from the skull through the jugular foramen, passing through the neck and chest to the abdomen. In this long course, the vagus nerve gives rise to a number of branches to innervate the larynx and

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Received November 18, 2008. Accepted after revision July 28, 2009.

the pharynx. In the cervical region, it passes between the carotid artery (medially) and the internal jugular vein (laterally). There are three major branches of the vagus nerve in this region: the pharyngeal, superior laryngeal and the recurrent laryngeal nerves.

The pharyngeal branch originates from the inferior ganglion of the vagus nerve and is part of the pharyngeal plexus, responsible for the motor innervation of the pharyngeal and levator veli palatine muscles.

The superior laryngeal nerve also originates from the inferior ganglion of the vagus nerve, caudally to the pharyngeal branch, and is divided into external laryngeal nerve (motor) and internal laryngeal nerve (sensory). It is responsible for the cricothyroid muscle innervation.

The recurrent laryngeal nerves (RLNs) (Figure 1) are responsible for the innervation of the other intrinsic muscles of the larynx⁽²⁾. These nerves carry motor, sensory and parasympathetic fibers, dividing itself into internal branch, responsible for the sensory function of the vocal cords and subglottic region, and external branch that bears the motor function of four intrinsic laryngeal muscles: thyroarytenoid, anterior and posterior cricoarytenoid, oblique and transverse arytenoid muscles⁽³⁾. At right, the RLN emerges anteriorly from the vagus

nerve at the intersection with the right subclavian artery to loop under and around the artery to reach its posterior surface. Then, it runs superiorly in the tracheoesophageal cleft towards the larynx. The left RLN emerges from the vagus nerve in the mediastinum, after crossing it anterolaterally to the aortic arch to loop under the arch and running between the aorta and the left pulmonary artery, extending posteriorly to the tracheoesophageal cleft to reach the larynx. Because of its longer and partially intrathoracic course, the left RLN may be affected by mediastinal diseases⁽¹⁾. Both nerves enter the larynx in the region of the cricothyroid joint through the fibers of the inferior constrictor muscle of the pharynx.

In the RLN ascending course from the chest to the larynx, a number of anatomical variations may be found. Rustad⁽⁴⁾, in a post-mortem series has observed that 43% of the RLNs split into one or more branches on both sides. The findings of nerve branching before reaching the larynx have also been surgically confirmed by Nemiroff & Katz⁽⁵⁾ in 40% of cases. The path of the RLNs may change because of congenital vascular abnormalities or anatomical distortions resulting from the presence of goiter, neoplasias or inflammatory processes. In cases of aberrant subclavian

artery, the right RLN runs directly from the vagus nerve to the larynx, without looping around the respective artery. This variation is closely related to surgical injuries and is known as “non-recurrent” inferior laryngeal nerve.

The RLN is localized in the tracheoesophageal cleft that is a relevant anatomical landmark, in 65% of cases at right, and in 77%, at left. The nerve is found at the right side of the trachea in 33% of cases, and at left, in 22%. It rarely ascends anterolaterally to the trachea and, in this case, it is more susceptible to surgical injury.

CLINICAL FINDINGS

Usually, patients with vocal cord paralysis present with complaint of hoarseness. Other more severe symptoms include frequent aspiration and pneumonia⁽⁶⁾. However, up to 35% of patients may be asymptomatic⁽⁵⁾. In these cases, vocal cord paralysis will be incidentally identified, and this finding should alert the clinician to the necessity of additional evaluation. The previous and direct evaluation by laryngoscopy must be initially performed to rule out the presence of mucosal and submucosal lesions like those caused by squamous cell carcinoma, to justify a subsequent imaging study.

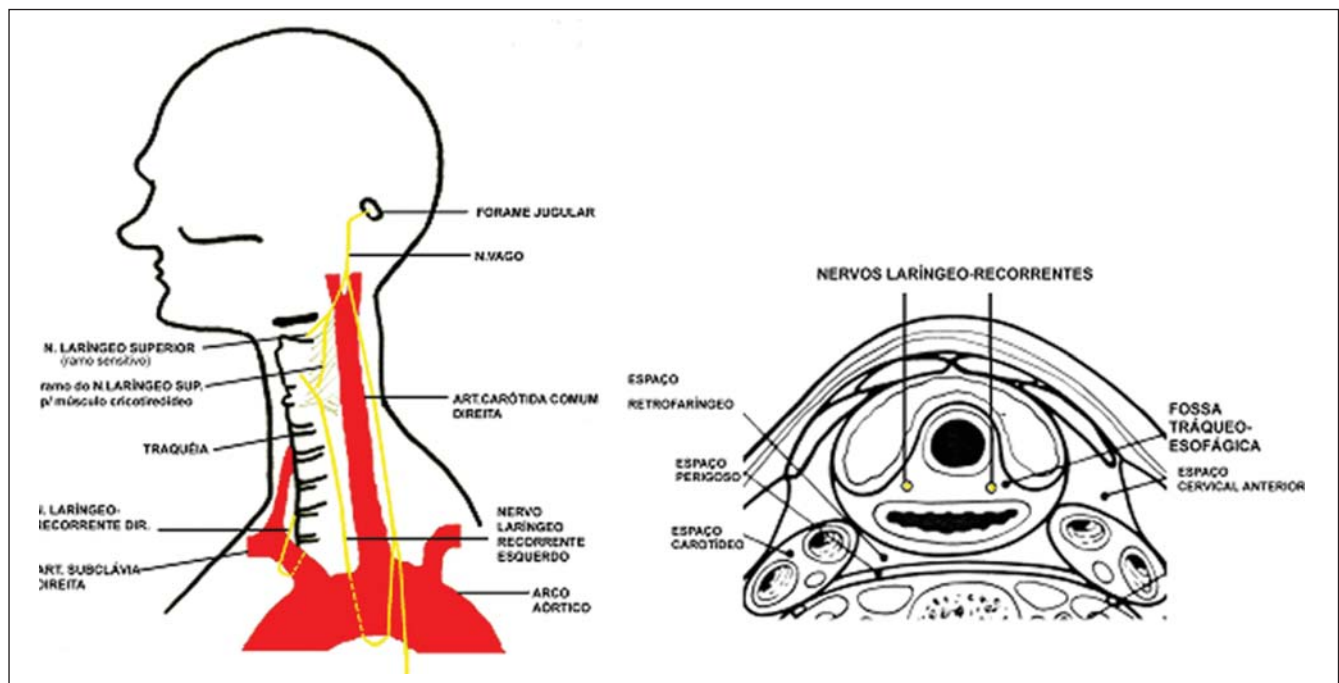


Figure 1. Scheme of anatomy of recurrent laryngeal nerves.

Vocal cord paralysis may be acute or chronic, and may occur in one or both vocal folds. The left vocal cord is most frequently affected, because of the longer course of the left recurrent laryngeal nerve, with about 12 cm in length from the aorta to the cricothyroid joining. At clinical examination, the identification of the affected side is relatively simple: the truly involved vocal fold will be with a complete or partially reduced mobility.

IMAGING DIAGNOSIS

The classic findings of vocal cord paralysis can be observed on plain radiographs and, particularly, on sectional imaging methods such as CT and MRI. These studies should include a scan from the skull base thru the aortic-pulmonary window, covering the whole course of the RLN. CT should include pre- and post-contrast phases, sometimes with a phonation phase with vocalization of the vowel *i*. This acquisition is useful in the evaluation of the true vocal folds mobility, but should be cautiously used to minimize the exposure of the patient to ionizing radiation. The study includes axial acquisitions with later 3D coronal and sagittal multiplanar reconstructions. The CT and MRI parameters utilized in the authors' institution are shown on Table 1.

The bulging of the oropharyngeal and hypopharyngeal contour in association with the thinning of the constrictor muscle

Table 1. Technical parameters utilized in computed tomography and magnetic resonance imaging studies.

Computed tomography (multislice, 64-channel equipment)						
	Collimation	mAs	kV	Acquisition time		
Pre-contrast	1.5 mm	150	140	8 s		
Post-contrast	0.75 mm	150	140	15 s		
Magnetic resonance imaging (1.5 T, 8-channel coil equipment)						
	TR	TE	TI	Thickness	Interval	Acquisition time
Coronal STIR	5275 ms	45 ms	150 ms	5.0 mm	0.5 mm	3.45 s
Coronal T1	342 ms	Minimum	—	5.0 mm	0.5 mm	2.59 s
Axial T1	550 ms	Minimum	—	5.0 mm	0.5 mm	2.26 s
Axial T2 FAT	4334 ms	Minimum	—	5.0 mm	0.5 mm	2.58 s
Axial T2	3650 ms	85 mA	—	5.0 mm	0.5 mm	2.34 s
Axial/coronal/sagittal post-contrast	534 ms	Minimum	—	5.0 mm	0.5 mm	3.30 s

constitutes an evidence of a lesion in the ipsilateral pharyngeal plexus⁽⁷⁾. Such findings indicate that the abnormality is localized immediately below the skull base or more cranially in the posterior fossa.

Most of imaging findings related to RLN paralysis are secondary to the thyroarytenoid muscle atrophy (Figure 2)⁽⁷⁾. At least ten findings associated with this nerve paralysis are described by Landman⁽⁸⁾ in laryngoscopic studies. Notwithstanding the limitations of the method, these findings were transferred for analysis by axial CT. Several findings correlated the CT images with vocal cord paralysis⁽⁷⁾; among them, the following can be mentioned:

- thickening and medial displacement of the ipsilateral aryepiglottic fold (the most frequent finding) (Figure 3);
- dilatation of the pyriform sinus (Figure 4);
- dilatation of the ipsilateral laryngeal ventricle (Figure 5);
- antero-medial displacement of the ipsilateral arytenoid cartilage (Figure 6);
- dilatation of the ipsilateral vallecula;
- flattening of the subglottic arch demonstrated on coronal images.

The first two findings above described are observed in more than 75% of cases, constituting the most reliable diagnostic criteria.

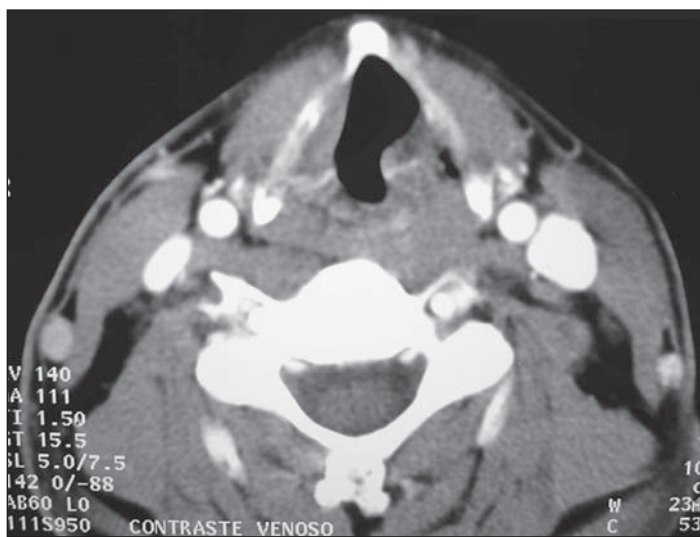


Figure 2. Retraction of left true vocal cord corresponding to thyroarytenoid muscle hypertrophy.

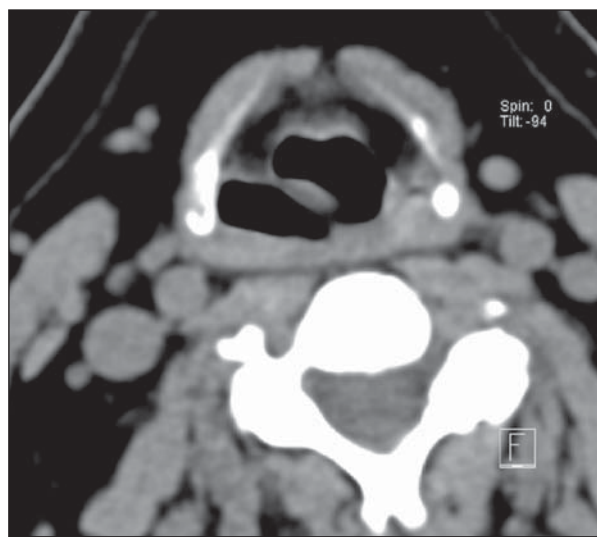


Figure 3. Thickening of right aryepiglottic fold.

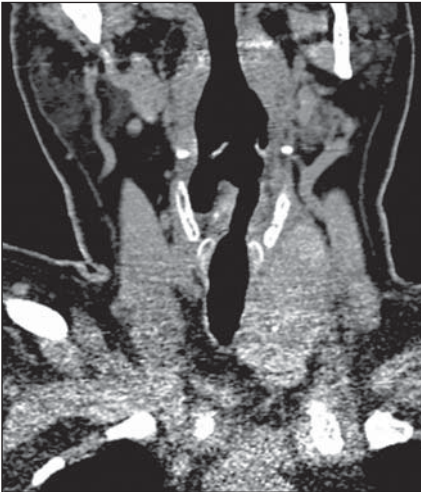


Figure 4. Right pyriform sinus dilatation.

With the advent of multidetector CT, high-resolution reformatted coronal images have provided a higher capacity for analyzing the larynx. However, the authors emphasize that axial images allow a correct diagnosis in most of cases.

CAUSES

There are several causes of vocal cord paralysis, and approximately 50% of reported cases are toxic or idiopathic⁽⁷⁾. Thus, in at least half of patients, sectional images may fail in identifying lesions along the vagus and recurrent laryngeal nerves path.



Figure 5. Left ventricle dilatation demonstrated by 3D reconstruction of multislice CT image.

In the past, the most common cause of vocal cord paralysis was thyroid surgery with dissection of the Berry's ligament, the site where the RLN penetrates the larynx. In these cases, the lesions result from an excessive traction of the thyroid gland, or occur at the moment of the hemostasis. The RLN may be connected with the inferior thyroid vessels, therefore its identification during the surgical procedure is essential. Recently, lung (Figure 7) and skull base

neoplasms, as well as surgical injuries have been the most frequent causes of vocal cord paralysis.

Several diseases may affect the vagus nerve tract (Figure 8), leading to vocal cord paralysis. Thus, it is necessary to evaluate this nerve from the brainstem to the carina. The causes of vocal cord paralysis may be divided into central origin (vagal neuropathy) and peripheral (RLN neuropathy), the latest being most frequently observed, achieving up to 90% of cases in some series. However, in most (up to 85%), lesions have not been identified along the nerves pathway.

Among the central causes, lesions affecting the brainstem, the skull base and the carotid bifurcation can be mentioned, all of them also associated with vagal neuropathy. In this group, vagal paragangliomas, hypervascularized masses involving or compressing the whole cranial nerve X pathway, as well as their nuclei in the jugular foramen can be clearly demonstrated on contrast-enhanced phases at CT and MRI⁽⁹⁾. Vascular events involving, for example, the posterior-inferior cerebellar artery leading to occlusion, may compromise the ambiguous nucleus in the dorsolateral aspect of the bulb. Thus, infarcts in this area may justify ipsilateral vocal cord paralysis⁽¹⁰⁾. Central vagal neuropathy may be suspected in the setting of paralysis or pare-

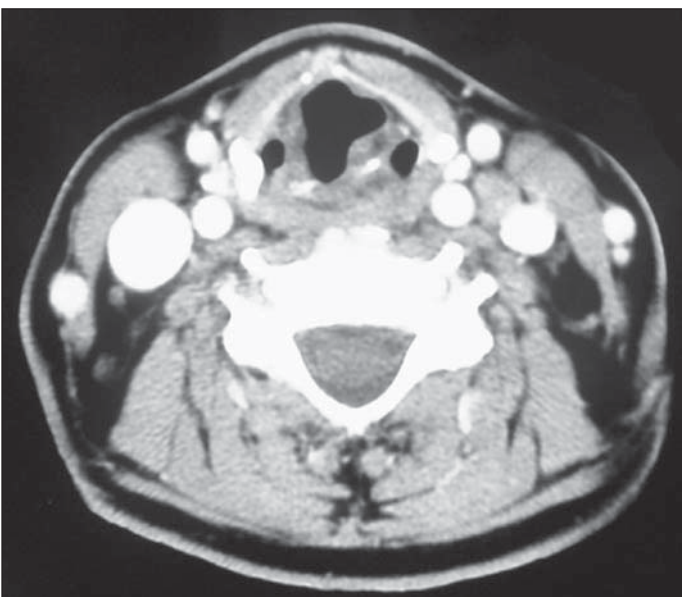


Figure 6. Anteromedial displacement of left arytenoid cartilage.

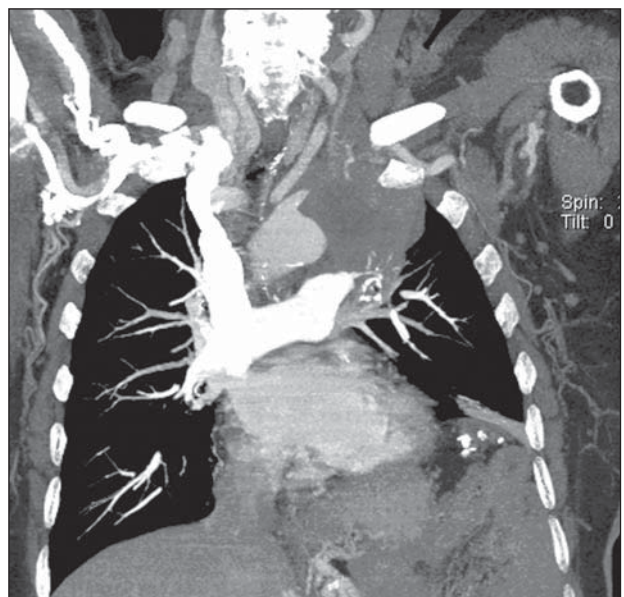


Figure 7. Pancoast tumor obliterating aortic-pulmonary region.

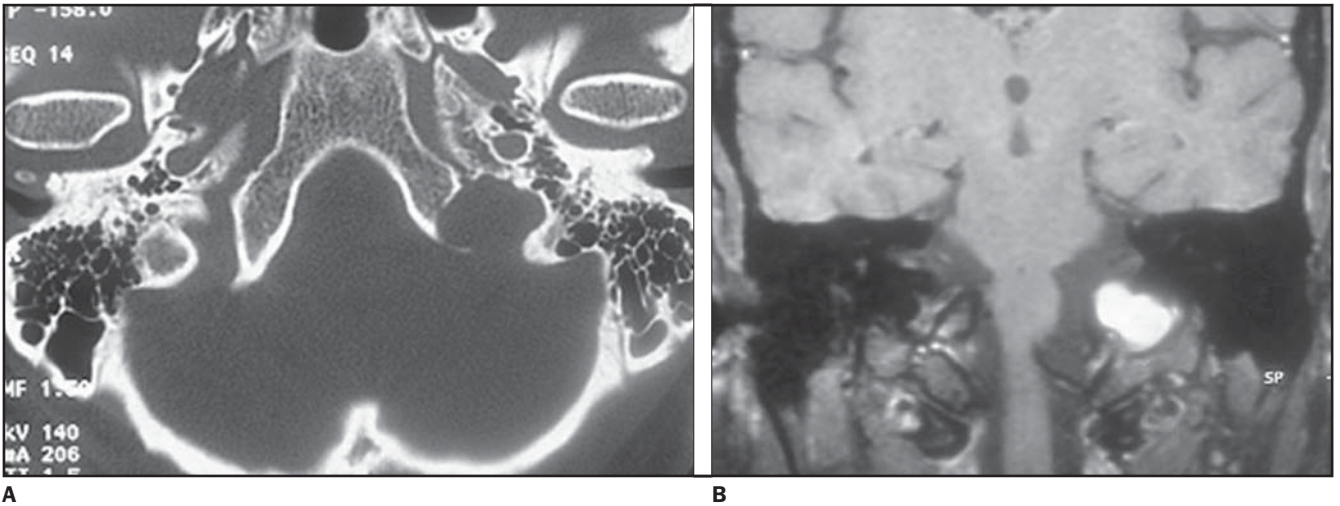


Figure 8. Axial CT image demonstrating left jugular foramen enlargement. Contrast-enhanced coronal MRI spin-echo, T1-weighted sequence with fat-saturation demonstrating a solid mass in the left jugular foramen, with intense and homogeneous contrast uptake: vagal schwannoma.

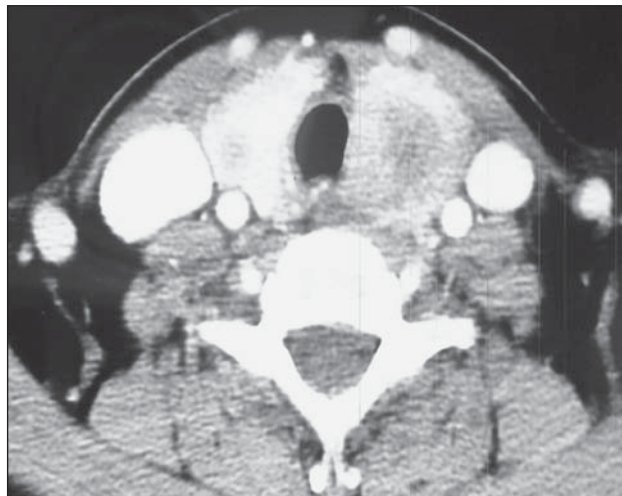


Figure 9. Left thyroid lobe neoplasm, with loss of cleavage planes with the left lateral tracheal wall invading the ipsilateral recurrent laryngeal nerve tract (same patient as Figure 6).

sis of the pharyngeal constrictor muscles in cases where the pharyngeal plexus is involved. The pharyngeal plexus is formed by branches of cranial nerves IX, X and XI, besides branches of the sympathetic trunk. The vagal branches emerge from the nodose ganglion localized below the skull base, and penetrate the pharyngeal muscles, branching into the superior and inferior constrictor muscles. A lesion affecting the pharyngeal plexus will cause paresis or paralysis of the ipsilateral constrictor muscles, eventually leading to atrophy.

Peripheral causes include RLN invasion by thyroid and cervical esophageal tumors (respectively Figures 9 and 10). Four per-

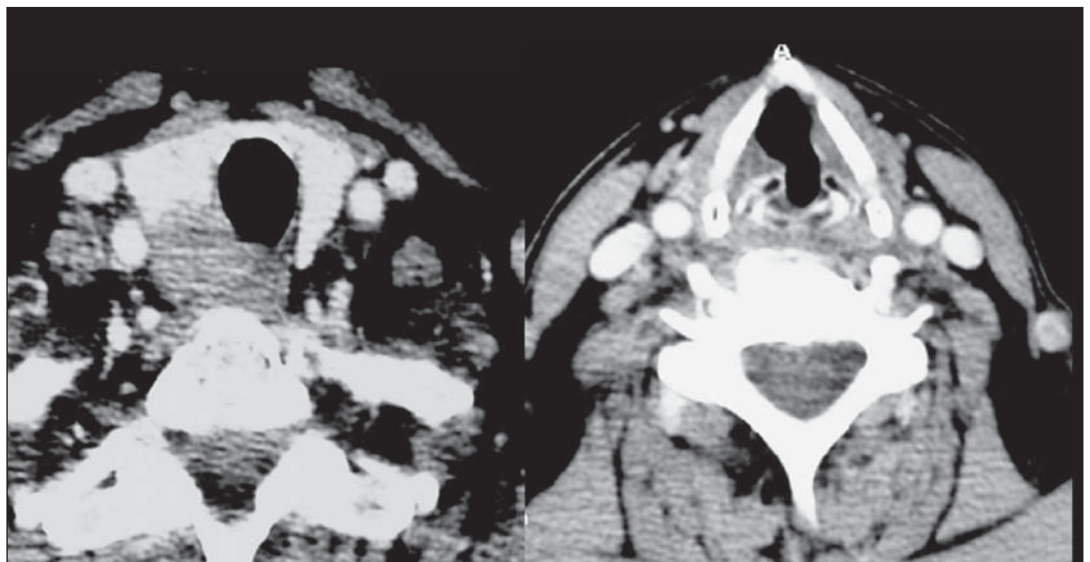


Figure 10. Esophageal neoplasia with invasion of right tracheoesophageal cleft and ipsilateral vocal cord paralysis.

cent of patients with unilateral vocal cord paralysis present thyroid disease, but only 0.7% are benign. Locally invasive thyroid cancers must be correctly evaluated by imaging methods, considering that the laryngotracheal invasion justifies the RLN involvement with subsequent vocal cord paralysis⁽¹¹⁾. Advanced malignant esophageal tumors may extend towards the tracheoesophageal groove, the RLN pathway⁽¹²⁾. On the left side, aortic aneurysms (Figure 11), cardiomegaly, and upper lobe lung tumors are implicated as potential causes of vocal cord paralysis. On the right side, supraclavicular tumors and subclavian artery aneurysms are considered as causal factors. Pancoast tumors, also known as superior sulcus tumors, are typically bronchogenic non-small-cell carcinoma, usually of squamous-cell type, affecting the superior sulcus that is the pathway of the right RLN⁽¹³⁾.

Vocal cord paralysis may also be associated with peripheral neuritis triggered by alcoholism, viruses, acute bacterial infections and drugs toxicity. Neurological diseases associated with multiple sclerosis, poliomyelitis, myasthenia gravis, amyotrophic lateral sclerosis, cerebrovascular disease and acromegaly complications are also implicated.

CONCLUSION

Considering that up to 35% of patients with vocal cord paralysis are asymptomatic, the recognition of radiological findings indicative of this condition is essential for the radiologist who must warn the referring physician on the imaging findings. Additionally, imaging methods, particularly the sectional ones, allow the study of the whole paired cranial nerve X and RLNs identifying the several diseases affecting this region. Thus, the knowledge of local anatomy and related diseases is of

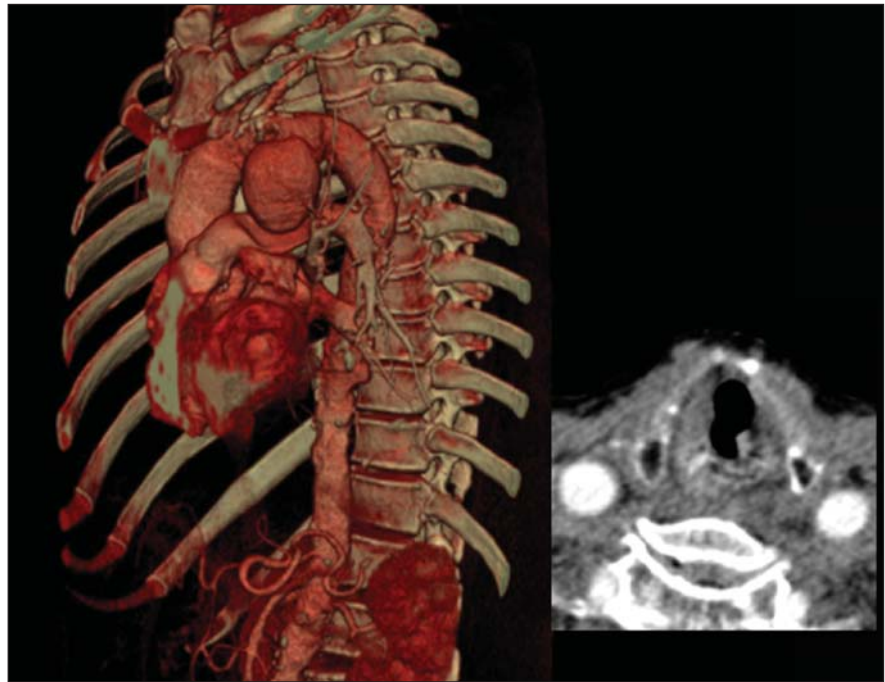


Figure 11. Saccular aneurysm of aortic arc projecting onto aortic-pulmonary window with consequential vocal cord paralysis at left.

great importance for the radiologist, so that he can tailor the examination properly to allow an appropriate diagnosis and therapy planning.

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