Unilateral Vocal Cord Paralysis: A Review of CT Findings.

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Learning objectives

The current exhibit aims to:

- To assess the course of the vagus and recurrent laryngeal nerves (RLN).
- To identify the reliable CT signs of unilateral Vocal Cord Paralysis (VCP) and avoid mimics and pitfalls.
- To recognize the different etiology of unilateral VCP.

Background

Vocal cord paralysis (VCP) due to recurrent laryngeal nerve (RLN) dysfunction is a frequent cause of hoarseness, and may be secondary to several conditions along the vagus nerve pathway and its branches, anywhere along its course, from the brainstem to the inferior margin of the nerve. Up to 40% of individuals with VCP may be asymptomatic [1]. Conversely, VCP can be the initial signs of serious disease secondary to invasion or compression of the RLN.

Vocal cord paralysis (VCP) may be caused by a variety of mediastinal disease entities, including various neoplastic, inflammatory, and vascular conditions, and may be the presenting symptom of an otherwise clinically occult disease. Careful evaluation of findings at CT examination, which should include the upper mediastinum, can then be helpful in directing and guiding appropriate intervention, whether surgery or watchful waiting.

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. Fortunately, the improved sensitivity of several diagnostic imaging modalities has decreased the incidence of "idiopathic VCP," with more frequent recognition of a causative lesion. Even in the absence of a history of hoarseness, the
radiologist can reliably suggest the presence of VCP with routine computed tomography (CT) of the neck. By understanding and assessing the entire course of the vagus and recurrent laryngeal nerves, the radiologist can avoid missing causative lesions, many of which have a clinical significance far beyond that of the VCP itself.

ANATOMY

The vagus nerves and RLNs are not directly visualized at CT. Therefore, knowing their expected courses is essential when searching for disease (fig 1).

The vagus nerve is the longest of the cranial nerves, extending from the brainstem to the abdomen. 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 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 recurrent laryngeal nerves (RLNs) are responsible for the innervation of the other intrinsic muscles of the larynx. These nerves carry motor, sensory and parasympathetic fibers, dividing itself into internal branch and external branch that bears the motor function of intrinsic laryngeal muscles [1, 2, 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 [1, 4, 5].

On average, the right RLN is 5-6 cm in length from its origin at the level of the brachiocephalic artery to the cricothyroid joint, whereas the left RLN is nearly 12 cm in length from its origin at the aortic arch, with much of its course being mediastinal. For this reason, the left RLN is more vulnerable to stretch or compression neuropathy from mediastinal abnormalities [1, 6].

Both nerves enter the larynx in the region of the cricothyroid joint through the fibers of the inferior constrictor muscle of the pharynx. The posterior cricoarytenoid muscle is the main vocal cord abductor, receiving its innervation from the ipsilateral RLN. Paralysis of the posterior cricoarytenoid muscle causes the arytenoid cartilage to subluxate anteromedially, resulting in medialization of the posterior aspect of the true cord, and is responsible for many of the CT signs of VCP.
In the RLN ascending course from the chest to the larynx, a number of anatomical variations may be found. 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 [6, 7, 8].

Images for this section:

![Diagram A](image1.png)
![Diagram B](image2.png)

**Fig. 1:** Drawings illustrate the normal anatomy of the vagus nerves (VN) and RLNs. (A-B) As the left vagus nerve passes anterolateral to the aortic arch, the left RLN branches off and passes below the arch posterior to the ligamentum arteriosum. It then ascends within the left tracheoesophageal groove to enter the larynx posteriorly at the level of the cricoarytenoid joint. The right vagus nerve descends posterolateral to the internal and common carotid arteries from the right jugular foramen, giving rise to the right RLN as it passes anterior to the right subclavian artery. The right RLN
then passes posterior to the right brachiocephalic artery before ascending to the larynx within the right tracheoesophageal groove. (C) At the level of the origin of the right subclavian artery (SCA), the right vagus nerve passes anterior to the subclavian artery, with the RLN crossing the mediastinum immediately below this point to reach the right tracheoesophageal groove. BCA Bifurc. = brachiocephalic artery bifurcation, CCA = common carotid artery, Tr = trachea. (D) At the level of the aortic arch (AoA), the left vagus nerve gives rise to the RLN, which passes below the arch to reach the left tracheoesophageal groove. SVC = superior vena cava, Tr = trachea.
Findings and procedure details

Unilateral vocal cord paralysis is caused by a number of common and uncommon mediastinal disease entities and may be the presenting symptom of an otherwise clinically occult disease. VCP is reliably identified at CT by recognizing key findings at the level of the true vocal cords. The most sensitive signs on CT scans are ipsilateral piriform sinus dilatation, medial rotation and thickening of the aryepiglottic fold and ipsilateral laryngeal ventricle dilatation.

I. CLINICAL FINDINGS

Usually, patients with vocal cord paralysis present with complaint of hoarseness. Other more severe symptoms include frequent aspiration and pneumonia. However, up to 40% of patients may be asymptomatic. 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.

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. 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.

II. CT of the Vocal Cords and RLNs course:

Multidetector CT is ideal for imaging the larynx because of its fast acquisition time, multiplanar capability and high resolution. With multidetector CT, an isotropic volume set of the neck can be obtained in approximately 10 seconds, which is particularly important with respect to motion artifact due to swallowing and breathing.

CT should include pre- and post-contrast phases. Depending on the indication for the imaging study, the patient may be asked to hold his or her breath, breathe quietly, "e-phonate," or attempt a modified Valsalva maneuver. The breathing technique used during image acquisition is important, given the affect it has on the appearance and position of both normal and immobilized cords (discussed later). 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. When VCP is suspected or confirmed clinically, quiet respiration is currently preferred for CT assessment of the larynx, since it brings the vocal cords to an intermediate position. This is ideal because the neck is
routinely imaged with CT during quiet respiration; thus, the cords can be adequately evaluated for signs of paralysis even in patients in whom there is no suspicion for vocal cord abnormality.

The study includes axial acquisitions with later 3D coronal and sagittal multiplanar reconstructions. Axial images parallel to the true vocal cords should be used to avoid pitfalls related to oblique section planes. The level of the true vocal cords is determined by identifying the cricoarytenoid joints on an axial image. The angled plane of the vocal cords can then be estimated from a paramedian sagittal image, and appropriate reformatted images can be obtained. For this reason, a minimum section thickness of 3 mm is recommended.

### III. Imaging diagnosis

With the advent of multidetector CT, the radiologist can make an accurate diagnosis of VCP at neck CT, even when it is not suspected.

At least ten findings associated with this nerve paralysis are described in laryngoscopic studies. These findings were transferred for analysis by axial CT.

High-resolution reformatted coronal images have provided a higher capacity for analyzing the larynx. However, axial images allow a correct diagnosis in most of cases.

The most sensitive signs on axial CT scans are [1, 2, 5, 7, 8]:

- Ipsilateral piriform sinus dilatation (**Fig 2**).
- Thickening and medial displacement of the ipsilateral aryepiglottic fold.
- Dilatation of the ipsilateral laryngeal ventricle (**Fig 2**).
- Antero-medial displacement of the ipsilateral arytenoid cartilage with medial displacement of the posterior vocal cord margin (**Fig 2**).
- Dilatation of the ipsilateral vallecula.

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

Flattening of the subglottic arch demonstrated on coronal images: On coronal reformatted images from CT data obtained during quiet respiration, the angle formed by the lateral wall of the trachea and the undersurface of the vocal cord becomes less obtuse, forming a superolateral concave contour referred to as the subglottic arch. In the case of UVCP, the normal subglottic arch below the ipsilateral adducted cord is lost secondary to a medially positioned vocal cord.
The "sail" sign: a combination of medialization of the posterior vocal cord margin and air distending the ipsilatérale laryngeal ventricle results in the residual airway having a shape similar to a ship's sail [1, 3].

The normal cord has a bowed appearance, convex toward the abnormal cord, and may even cross the midline of the laryngeal vestibule: When the breath is held, the paralyzed vocal cord cannot adduct; thus, the contralateral normal cord will extend further medially than normal in an effort to close the glottis in a compensatory manner.

We suggest that the indirect findings of ipsilateral pyriform sinus dilatation, medial positioning and thickening of the ipsilateral aryepiglottic fold, and ipsilateral laryngeal ventricle dilatation are more reliable imaging criteria than the appearance of the true vocal cord itself for assessing unilateral vocal cord paralysis. In difficult cases, the use of coronal reformatted images of the larynx may further refine diagnosis by revealing ipsilateral flattening of the subglottic arch [2, 5].

IV. CAUSES

The RLN can become paralyzed secondary to an abnormality anywhere along its course from the brainstem to the caudal margins of each recurrent nerve. Therefore, maintaining a course-based approach to the differential diagnosis is useful.

Because of its longer course and its extension into the mediastinum, the left side is more often affected than the right side. For the investigation of right VCP, levels from the medulla to the brachiocephalic artery should be considered. With left VCP, the assessment should extend inferiorly to include the aorticopulmonary window.

Etiology of unilateral VCP can occur because of dysfunction at the brain and brainstem nuclei, at the vagus nerve and at the RLN.

There are several causes of vocal cord paralysis. The most common causes of VCP includes neurologic or central etiology, traumatic injury (iatrogenic and non iatrogenic), systemic diseases, medications (Vinca alkaloids), tumor infiltration (skull base, thyroid, lung) or mass compression, a variety of mediastinal diseases.

VCP can be idiopathic, approximately 50% of reported cases of VCP are toxic or idiopathic [2, 4, 8].

Surgery seems to be the most common cause unilateral VCP (47%) followed by idiopathic (18%) and malignancy(13%), in which lung was most common [9].

1. Idiopathic unilateral VCP

Idiopathic unilateral VCP is not well understood. A possible infectious cause has been proposed because many patients report an antecedent URI before the onset of vocal
symptoms. However, there is no data to suggest that steroids or antivirals affect the course of the disease.

In a retrospective analysis, Urquhart et al showed that 26% of patients with a diagnosis of idiopathic VCP had a preexisting neurologic condition and 20% developed a subsequent CNS condition. Thus careful neurological evaluation of patients with a diagnosis of idiopathic VCP is recommended.

2. Traumatic

Traumatic injury is the most common cause of unilateral VCP [8, 9]. Traumatic causes are iatrogenic and non iatrogenic.

Surgical causes are the most common in this category, and this includes thyroidectomy, anterior cervical spine procedures, esophagectomy, thymectomy, CEA, and CT surgeries [1, 3, 7].

Rosenthal et al. analyzed the surgical causes of unilateral vocal cord paralysis and showed that the most common cause were non-thyroid surgeries at 67%. These included cervical spine procedures, CEA and cardiac procedures. Thyroid and parathyroid surgeries comprised the remaining 33% [3, 6].

Non surgical causes include the Tapia syndrome. It is a recurrent laryngeal and hypoglossal nerve paralysis caused by cuff of the endotracheal tube on the thyroid cartilage.

Finally, non iatrogenic traumatic causes include blunt or penetrating trauma to the neck.

3. Neurologic or Central causes:

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 (Fig.3, 4). 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 (Fig.5).

If work-up CT for VCP does not show a causative lesion, making the distinction between a proximal vagal neuropathy (brainstem to immediately inferior to the jugular foramen) and a distal neuropathy involving the RLNs in isolation is important for directing further imaging and management. In addition to findings of VCP, proximal involvement can manifest at CT as dilatation of the ipsilateral oropharynx (outward bowing), thinning of the pharyngeal constrictor muscles due to atrophy, and uvular deviation away from the
side of the causative lesion. If central-proximal involvement is suspected, further imaging with magnetic resonance (MR) imaging of the posterior cranial fossa is indicated [1, 2, 5].

Overall, vocal cord paralysis has been reported, among other causes, to be the result of a peripheral neuritis associated with alcoholism, viruses, acute bacterial infections, and drug toxicities [5, 7]. Neuropathies associated with multiple sclerosis, polio, myasthenia gravis, Parkinson's disease, amyotrophic lateral sclerosis, cerebrovascular diseases, and complications of acromegaly have also been implicated [2, 4].

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1. Mediastinal Causes of VCP

The Vocal cord paralysis (VCP) may be caused by a variety of mediastinal disease entities. The most common mediastinal causes of VCP include lung cancer, aortic dissection, metastatic disease, tuberculosis, and esophageal cancer, although a variety of other conditions, including pulmonary embolism and amyloidosis, have also been implicated.

a) Vascular-Cardiac Causes

Vascular causes of VCP are of particular concern because hoarseness due to VCP may be the initial sign of serious disease as an expanding dissection or aneurysm at risk for catastrophic rupture (Fig 6). Other vascular mediastinal causes of VCP include pulmonary artery enlargement or embolism.

The anterior bronchoesophageal artery frequently supplies the left vagus nerve and RLN. For this reason, vascular damage occurring during mediastinal lymph node excision, especially in the subaortic region, may contribute to postoperative VCP.

Ortner's syndrome, also known as cardiovocal syndrome, refers to hoarseness due to recurrent laryngeal nerve palsy secondary to cardiovascular disease which comprises all kinds of disease such as mitral stenosis, pulmonary artery hypertension, aortic aneurysm, aortic dissection, pulmonary embolism, left atrial enlargement [3, 6, 7, 8].

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a) Primary Neoplasms

Thoracic neoplasms can result in malignant invasion of the RLNs and other important neural and vascular structures. Important neoplasms to consider include bronchogenic carcinoma (Fig.7) and thymic malignancies (Fig 8), esophageal cancer, and neurogenic tumors such as paragangliomas and schwannomas [1, 2, 4, 7]. Malignant tumor progression can also be heralded by the development of VCP. Of particular importance is the fact that new VCP in a patient with a known intrathoracic malignancy can suggest
a more advanced disease stage by indicating previously unrecognized mediastinal invasion or compressive adenopathy [1, 3].

Lymphoma is a common malignant cause of VCP and may produce VCP by compression, rather than invasion, of the RLN (Fig 9).

UVCP may be caused not only by direct invasion of malignant tumors, but also by complications of cancer therapy. Extensive excisions in thyroid, lung and esophageal cancer may all cause vocal fold paralysis from mechanical damage or excision of the vagus nerve or its recurrent laryngeal branch. Likewise, radiotherapy for nasopharyngeal and breast cancer can produce similar effects [2, 3, 7, 9].

b) Metastatic Disease

Metastatic disease in particular, high right paratracheal and aorticopulmonary window mediastinal adenopathy is often associated with VCP. Primary malignancies that commonly metastasize to the mediastinal lymph nodes include bronchogenic, breast, and esophageal carcinoma.

d) Thyroid Conditions

Benign thyroid disease rarely manifests with VCP. If VCP is associated with a benign-appearing thyroid mass (even if extremely large), possible malignancy or an alternative cause for paralysis should be sought (Fig 10). A large benign substernal goiter can be an indication for thyroidectomy, which poses an increased risk for permanent RLN paralysis due to surgical ligation or compression [1, 3, 5, 7, 8].

The Riedel thyroiditis, as a fibrotic process, can invade adjacent structures of the neck, including the RLNs, and cause VCP [1].

e) Infectious and Inflammatory Conditions

VCP is a rarely associated complication of thoracic tuberculosis [1, 4, 7, 9]. VCP due to pulmonary or mediastinal tuberculosis is often right sided and may be due to acute lymphadenopathy, caseating granulomatous inflammation, or late complications of chronic fibrosis (Fig 11).

Silicosis and sarcoidosis may result in VCP by either compression of the RLN by adenopathy or granulomatous and fibrotic lymph nodes encasing the nerve. Fibrosing mediastinitis can result in stenosis of the central pulmonary vasculature and RLN encasement, leading to pulmonary hypertension and VCP [4, 6, 8].

1. Mimics of VCP

VCP can be reliably identified at CT. Although there are a number of VCP mimics and imaging pitfalls, they can generally be avoided by carefully assessing the scan plane and level and evaluating for additional findings.
The radiologist should also be aware of several mimics of VCP to avoid misinterpreting the appearance of normal vocal cords or overlooking other potential diagnoses that can significantly alter management and prognosis.

Neck neoplasms, arytenoid cartilage subluxation or fracture, and oblique imaging findings can all mimic CT findings of VCP.

**Laryngeal malignancy**

Laryngeal and piriform sinus squamous cell carcinoma can mimic VCP by invading the aryepiglottic cartilages, resulting in vocal cord immobilization.

Soft-tissue thickening from tumor can give the appearance of medialization of the cord, which can be incorrectly interpreted as a sign of VCP [1, 2, 5].

Recognition of abnormally enhancing and thickened soft tissue, obliteration of normal fat planes, sclerosis of the adjacent cartilage, or adenopathy in the neck should raise concern for a neoplastic process.

**Arytenoid cartilage dislocation or subluxation**

It can easily be misdiagnosed as VCP (Fig 12). Intubation is the most common origin, followed by external laryngeal trauma. Also, incidental findings on CT of arytenoid malposition have been reported with rheumatoid arthritis, laryngeal cancer, or chondroradionecrosis from radiation therapy.

On coronal CT scans, a disparity in the height of the vocal cords will be present, a finding that is not seen in VCP, and the cricoarytenoid joint will be ill defined. Thinsection CT with three-dimensional reconstruction is very helpful in establishing the diagnosis [1, 3, 6, 8].

The use of oblique imaging planes may lead to asymmetric visualization of the true vocal cords. To avoid this potential pitfall, it is important to ensure that the cricoarytenoid joints are included on the same axial or oblique axial reformatted image.

**Images for this section:**
Fig. 2: (A) Axial CT scan in a patient with a left vocal cord paralysis shows enlargement of the left piriform sinus (white arrow) and anteromedial deviation of the arytenoid cartilage (black arrow). (B) Axial CT scan in the same patient shows atrophy of the left thyroarytenoid muscle as evidenced by enlargement of the left laryngeal ventricle (white arrow) relative to the right.
Fig. 3: Glomus jugulare in a 56-year-old woman with peripheral facial paralysis. (A) Axial CT image in bone window and (B) soft-tissue window demonstrates left jugular foramen mass with irregular margins and lytic changes of surrounding temporal bone. MRI (C) Axial flair T2-weighted image (D) Axial and (E) coronal gadolinium-enhanced T1-weighted image enhancing mass within the right jugular foramen which expands and erodes the adjacent petrous bone and extends to the cerebello-pontine angle cistern and parapharyngeal space.

Fig. 4: Osteomyelitis of the petrous apex and skull base in a patient with necrotizing otitis externa. (A, B) Axial CT image in bone window, MRI (C) Axial T1-weighted image (D) Axial T2-weighted image (E) axial and (F) coronal Gadolinium-enhanced fat-suppressed T1-weighted image show an infiltrative process involving the central skull base. Corresponding gadolinium-enhanced fat-suppressed T1-weighted MR image shows intense enhancement of the left petrous apex and clivus as well as of the left X nerve.
**Fig. 5:** VCP in a 50-year-old man who presented with a change in voice quality. Subsequent axial diffusion-weighted MR image of the posterior fossa shows a focal area of diffusion restriction (arrow), reflecting an infarct in the location of the vagus nerve nucleus.
Fig. 6: CT scan thorax showing aortic arch aneurysm and dissection into its wall with formation of an intramural haematoma containing thrombosed blood clot anteriorly. The aneurysm may directly compress and cause injury of the left recurrent laryngeal nerve.
**Fig. 7:** Non-small cell lung carcinoma with mediastinal invasion in a 45-year-old male smoker. CT scan shows a neoplasm with invasion of the aorticopulmonary window.

**Fig. 8:** Contrast-enhanced Chest CT demonstrated an extensive mediastinal tumor with invasion of the aorticopulmonary window. A mediastinal biopsy concluded to a thymic carcinoma. VCP is caused by direct invasion of the malignant tumor.
Fig. 9: Hodgkin lymphoma in a 40-year-old man with shortness of breath and hoarseness. Contrast-enhanced CT demonstrated bilateral mediastinal adenopathy extends in the high right paratracheal and retrotracheal space.
Fig. 10: Contrast-enhanced neck CT scan shows a mass lesion of the left thyroid gland (arrow) along the expected course of the left RLN. Results of thyroid biopsy confirmed carcinoma.

Fig. 11: Right VCP in a patient with a prior history of pulmonary tuberculosis. Thoracic CT scans demonstrate fibrotic changes related to chronic tuberculosis in the right upper lobe.

Fig. 12: Axial CT scan shows arytenoid cartilage dislocation mimicking right VCP. The right arytenoid cartilage (arrow) lies anterior to the cricoid cartilage, slightly below the expected level of the true vocal cords.
Conclusion

VCP may be caused by a number of common and uncommon disease entities and may be the presenting symptom of an otherwise clinically occult disease. 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. VCP can be reliably identified at CT by recognizing key findings at the level of the true vocal cords and aryepiglottic folds, and by avoiding the common pitfalls and mimics. By understanding and assessing the entire course of the vagus nerves and RLNs, including their mediastinal segments, radiologists can avoid missing causative lesions, many of which are of greater significance to the patient than is the VCP itself.

Personal information

References


