Hoarseness revealing a rare pathology

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Clinical history

An 86-year-old man consulted for dysphonia of progressive onset, with no associated false passage or dyspnea. He had a history of smoking with 20 years abstention, assessed at 40 pack-years, and of high blood pressure, myocardial infarction, grade 2 lower-limb arteriopathy and obstructive chronic bronchitis.

Flexible endoscopy found left immobile arytenoid and vocal fold (LVF), which was in a paramedian position. There was nascent atrophy of the LVF, which was displaced downward with respect to the facing fold, with persistent moderate glottic chink on phonation (Fig. 1). All three laryngeal compartments could be examined, with no visible mucosal lesion, notably in the left laryngeal ventricle and subglottic compartment.

The patient came with a recent plain antero posterior (AP) chest X-ray, prescribed by his family physician (Fig. 2).

Figure 1 Direct laryngoscopy using a flexible nasal endoscope on inspiration, revealing left vocal fold atrophy and downward displacement (→).
Questions

Question No. 1: How do you interpret the plain chest X-ray (Fig. 2)?

Question No. 2: What complementary radiologic examinations do you prescribe?

Question No. 3: What are your findings on the complementary radiology images?

Question No. 4: What other complementary examinations would you prescribe on discovering left laryngeal immobility?

Question No. 5: What is your diagnosis?
Replies

Reply no. 1

The aortic knob shows increased volume and partial calcification. There is no pulmonary mass. The cardiothoracic index is elevated (0.55).

Reply no. 2

Contrast-enhanced thoracic CT with frontal and sagittal reconstruction (Fig. 3).

Reply no. 3

CT shows enhancement with contrast-medium uptake, with a thick and partly calcified wall, developing in the aorto-pulmonary window to the detriment of aortic arch concavity, corresponding to a 40-mm atheromatous aneurysm with partial thrombosis of the wall. There is no tumor along the intrathoracic route of the left recurrent laryngeal nerve (RLN). The frontal and sagittal slices show filling of the aorto-pulmonary window.

Reply no. 4

In case of left laryngeal immobility, the entire anatomic route of the vagus nerve (CN X) and its RLN branch should be explored. Skull-base contrast-enhanced CT passing through the posterior tear and cervico-thoracic contrast-enhanced CT should be performed. In the present case, skull-base and cervical CT were normal.

Laryngeal examination under general anesthesia should be performed:

- to minutely examine (under microscope and endoscope) the three laryngeal compartments, and especially the laryngeal ventricles and subglottic region, to explore for possible tumor;
- to check arytenoid mobility, by palpator, to rule out cricoarytenoid ankylosis.

In the present case, this examination was prevented by a contra-indication for general anesthesia, but high-quality flexible endoscopy under local anesthesia ruled out tumor without, obviously, testing arytenoid mobility.

Laryngeal electromyography should be performed to check possible innervation abnormality of the intralaryngeal muscles: immobility in a vocal fold does not necessarily mean paralysis. The patient, however, refused this examination.

Reply no. 5

By a process of elimination, even though not all complementary examinations could be performed in this patient, Ortner’s syndrome was diagnosed.

Comments

Ortner’s syndrome, or cardio-vocal syndrome, is one of the several possible etiologies of left RLN palsy. The generally agreed physiopathologic mechanism is nerve stretching and/or compression by a cardiac or vascular structure. The etiology underlying the syndrome may be [1]:

- left auricle dilation due to tight mitral stenosis;
- left ventricle dilation in myocardiopathy of whatever cause;
- persistent arterial canal;
- or aneurysm of the aortic arch or origin of the left common carotid.

Aneurysm is usually of atheromatous origin [2], but has also been described in association with mycotic arterial infection or Horton’s disease. Less than 5% of thoracic artery aneurysms are associated with left RLN palsy [3]. The implantation, shape and size of the aneurysm sac are determining factors, as only large aneurysms in the aorto-pulmonary window can impair left RLN function. Contrast-enhanced thoracic CT is the examination of choice for diagnosing thoracic artery aneurysm. It enables not only diagnosis but exploration for signs of complications such as fissure and/or rupture. Helical CT angiography with volume acquisition provides reconstruction images comparable to classic angiography, which still keeps its role in pre-treatment assessment. The spontaneous evolution of aortic arch aneurysm is generally fatal, but surgical management by thoracotomy or implant is sometimes possible. Thoracotomy not only treats the aneurysm, but also enables the RLN to be dissected and spared, in some rare cases curing the paralysis [4].

Less than 10% of intrathoracic causes of RLN palsy involve compression, far less than tumor or surgical trauma [5]. The incidence of Ortner’s syndrome among these compressive pathologies is hard to determine, but is certainly small:

![Figure 3](image-url)  
**Figure 3** Contrast-enhanced thoracic CT: axial (A), frontal (B) and sagittal slice (C).
fewer than 20 cases of thoracic aorta aneurysm associated with Ortner’s syndrome have been reported [6].

In the present case, the aneurysm-related risk persists, as surgery was not suggested due to age and comorbidity. Regular ENT examination is recommended to screen for salivary or liquid and/or solid feeding false passage in case of complete vocal fold atrophy due to thyroarytenoid muscle amyotrophy. In that case, corrective surgery—either endoscopic with injection of autologous fat or heterologous material under short general anesthesia, or thyroplasty under local anesthesia—should be envisaged. Published reports fail to specify the role of such surgery, probably due to very short follow-up.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

References