A Rare Cause of Vocal Cord Palsy due to Small Thyroid Nodule
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Abstract
Thyroid lesion is strongly associated with recurrent laryngeal nerve palsy. It is because of the close anatomical relationship between the gland on the trachea and the nerve which lies in the tracheoesophageal groove. The recurrent laryngeal nerve can be affected either from the thyroid disease itself or as the complication of thyroidectomy. However, benign thyroid lesion rarely causes recurrent laryngeal nerve palsy. We report a case of a small thyroid nodule causing unilateral vocal cord palsy. After lobectomy, the voice improved but due to the compensation mechanism of the opposite side. As the nerve was found to be atrophic intraoperatively, recovery should be allowed if it is going to take place, in a long period of time.

CASE SUMMARY
A 39 year-old Malay secretary complained of hoarseness of voice 3 months. It was gradual in onset but slowly progressive. It was persistent and more obvious through out the day. It was aggravated by increased in conversation and relieved by rest. The hoarseness was associated with shortness of breath on moderate to severe exertion. Occasionally there were choking and coughing sensations especially during drinking. The condition affected her daily office work. There was no history of upper respiratory tract infection, recurrent fever, history of trauma or surgery to the neck.

General examination revealed normal findings. The voice was hoarse but not breathy. She was able to count 1-10 in 1 breath and cough was also elicited during drinking. Except small vague nodule on the left side of the neck, there was no other abnormality detected. Laryngoscopy revealed the left vocal cord was in paramedian position and there was a glottic gap during phonation.

Fine needle aspiration showed colloid goiter. Computed tomography scan showed presence of left lobe nodule measuring 1.6 x 1.7 cm. Ultrasonography thyroid was performed. It was interpreted as possible malignant thyroid nodule. Thyroid radioiodine scan was ordered as an adjunct investigation.

Figure 1
Fig. 1: Left vocal cord palsy with glottic gap on phonation
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Figure 2
Fig. 2: CT scan showed left thyroid nodule.

Figure 3
Fig. 3: Presence of nodule was confirmed in thyroid scan.

After evaluation of all the results, left lobectomy was planned with frozen section microscopy. Left recurrent laryngeal nerve was hardly identified except a thin strand of whitish fiber which was not responding to the nerve stimulator. However, for the benefit of the doubt, subcapsular excision of the nodule was performed in view of possible preserving the atrophic recurrent laryngeal nerve.

Figure 4
Fig. 4: Nerve stimulator pointed at external branch of superior laryngeal nerve.

During follow-up, patient claimed that the voice was improved. However, laryngoscopy still showed that the left vocal cord position was still in paramedian. The improvement of voice quality was due to the compensating right vocal cord functions.

Magnetic resonance imaging (MRI) of the neck and chest were performed three weeks after the operation to look for the primary cause that possibly lead to nerve compression. However, the study was normal.

DISCUSSION

Recurrent laryngeal nerve (RLN) is always at risk whenever thyroid disease is managed. It can be either due to the complication of the disease itself, which is most commonly due to malignancy or the paralysis can be due to the surgical intervention namely thyroidectomy. Thyroid surgery is the most common cause of vocal cord paralysis.1,2

Vocal cord paralysis found in a benign thyroid disease patient is very rare. A review of literature showed that a pre-operative incidence of 0.7% of vocal cord paralysis among surgical patients who proved to have benign thyroid disease.3

The pathophysiology of vocal cord paralysis in the few benign cases may be attributed to compression, distension or stretching of the nerve especially in cases of goiter which has gone retrosternally, retroclavicularly or intrathoracic. In our case, the solitary nodule is too small and yet the nerve was atrophic most probably due to long standing.
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compression. Other theories include local inflammation, oedema, thrombosis of the blood supply, perineural fibrosis, calcification, toxic neuritis and idiopathic paralysis.1,2,4,5

The provisional diagnosis in this case was malignant thyroid nodule. This was based on the findings of nerve palsy, ultrasonographic and computed tomography features and cold nodule on thyroid scan. Frozen section was done intraoperatively keeping in view to proceed the surgery with total thyroidectomy. However, it turned out to be negative for malignancy. Although frozen section microscopy has some limitation. The evaluation of thyroid neoplasm is highly accurate and specific.6

The left external branch of superior laryngeal nerve (EBSLN) was identified with the help of intraoperative nerve monitoring. Although it carries less significant complication as compared to the RLN, the damage to this branch also could affect the voice quality. The EBSLN damage will lead to bowing of the affected cord and cause glottic gap to be presence during phonation. These catastrophic consequences may be more prominent in those who are professional voice users.7 As the rate of EBSLN injury increased when not exploring the nerve during surgery, it must be localized expressly to reduce the risk.8

The left RLN was found to be atrophic and not responding to the stimulator in this case. However, in view of the benign nature of the mass from frozen section evaluation, we performed subcapsular lobectomy on the left side without taking the risk of damaging the nerve.

The voice improvement on follow-up may be due to the compensation from the opposite cord. The recovery of the nerve was still in progress as the laryngoscopic examination of the vocal cord still showed the left side fixed at paramedian position.

References
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