CASE REPORT

Bilateral vocal cord palsy post thyroidectomy: lessons learnt

Khairunnisak Misron,¹ Anusha Balasubramanian,¹ Irfan Mohamad,¹ Nik Fariza Husna Nik Hassan^{1,2}

¹Department of Otorhinolaryngology-Head and Neck Surgery, Universiti Sains Malaysia, Kota Bharu, Kelantan, Malaysia

²Department of Speech Pathology Programme, Universiti Sains Malaysia, Kota Bharu, Kelantan, Malaysia

Correspondence to

Dr Anusha Balasubramanian, aansangel@yahoo.com

Accepted 23 February 2014

SUMMARY

Bilateral vocal cord paralysis is a known possible complication following thyroid surgery. It owes to the close relationship between the recurrent laryngeal nerve and the thyroid gland. The most feared complication of bilateral vocal cord paralysis is airway compromise. We report the case of a 39-year-old woman who underwent total thyroidectomy for multinodular goitre. The surgery was uneventful. However she developed stridor in the recovery bay needing intubation. We postulate that the cause was attributed to bilateral vocal cord paresis due to the use of the intraoperative nerve monitoring (IONM) whose high setting throughout the surgery was overlooked. She made a complete recovery without the need of a tracheostomy. We share our lessons learnt from this case.

BACKGROUND

Thyroidectomies are performed for many reasons. They include primary treatment for differentiated thyroid malignancies, compressive symptoms of benign lesion, cosmesis, surgical access to the airway and multinodular goitre as in this case. Patients are thoroughly informed regarding the potential complications of the surgery, namely bleeding, infection and recurrent laryngeal nerve injury causing vocal cord immobility. The incidence of post-thyroidectomy vocal cord paralysis has been reported in the range of 3.5–6.6%, and 93–100% of these patients had made complete recovery.^{1,2} Vocal cord paralysis may be unilateral or bilateral, and could be transient or permanent. Bilateral vocal cord paralysis following thyroidectomy has been reported as low as 0.58%.² The symptoms vary from hoarseness to severe life-threatening respiratory distress, especially if bilateral cords are involved. We discuss the possible causes for post-thyroidectomy vocal cord palsy as well as the role of the IONM.

CASE PRESENTATION

A 39-year-old Asian woman presented with an anterior neck swelling for 1 year. She, a homemaker, has had hoarseness for 2 years. Otherwise, she had no obstructive symptoms or noisy breathing. She was clinically and biochemically euthyroid. On examination, there was a 10 cm×5 cm anterior neck swelling, which was firm in consistency and moved with swallowing.

INVESTIGATIONS

Ultrasound of the neck showed multiseptated cystic component with intervening solid component and

specks of calcification within. Fine-needle aspiration cytology revealed the finding of colloid goitre. Preoperative rigid 70° laryngoscopy showed normal movement of bilateral vocal cords with an incidental finding of a small cyst-like lesion at the middle segment of the right vocal cord without a phonation gap, which explained the cause of hoarseness in this patient.

TREATMENT

She was planned for total thyroidectomy and excision of the vocal cord lesion. She was intubated using electromyography (EMG) endotracheal tube size 7.0 mm via direct laryngoscopy, which was successful in a single attempt. The surgery was uneventful. Intraoperatively, both superior laryngeal nerves and recurrent laryngeal nerves were identified and preserved. Intraoperative nerve monitoring (IONM) was used and it was stimulated at 1 mA throughout the operation.

Postoperative extubation was uneventful, but half an hour later, she developed acute stridor, vomiting and desaturated at the recovery bay. Due to worsening symptoms, she was reintubated immediately. During intubation, severe laryngeal oedema involving the entire length of both vocal cords was noted. She was initially planned for reintubation with endotracheal tube size 7.0 and 6.5 mm, but both attempts failed due to resistance at the level of the vocal cords. Reintubation was successful with a size 6.0 mm endotracheal tube, which was one size smaller than the one initially used. Otherwise, there was no neck swelling. The drain was functioning with minimal hemoserous fluid.

OUTCOME AND FOLLOW-UP

She was successfully extubated 24 h postoperatively. Postextubation, she had mild hoarseness and cough each time she consumed fluid. Clinically, there was no shortness of breath or stridor. Rigid 70° laryngoscopy that was performed on the sixth postoperative day revealed both vocal cords in the paramedian position with minimal symmetrical mobility of the vocal cords and the presence of a phonation gap. Otherwise, the laryngeal oedema had completely resolved and the vocal cord cyst regressed in size. The corrected serum calcium level was normal. She was diagnosed with laryngeal oedema with bilateral recurrent laryngeal nerve paresis secondary to overstimulation of the IONM. She was started on intravenous dexamethasone 8 mg stat three times a day for 3 days. There was no episode of aspiration during swallowing. She

CrossMark

To cite: Misron K, Balasubramanian A, Mohamad I, et al. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2013-201033

was discharged home on the sixth day after surgery with oral methylcobalamine 500 µg three times a day, and was advised to return to the emergency department immediately in the event of noisy breathing or breathing difficulty.

During her first follow-up after 2 weeks, examination in the clinic showed minimal improvement in the mobility of the vocal cords bilaterally with mild improvement in the phonation gap as compared to the initial findings before discharge. However, her vocal cord mobility had subsequently improved markedly and the phonation gap improved significantly with the aid of speech therapy. She completely recovered from her neuropraxia 3 months after surgery. A repeat assessment at 6 months, 8 months and 1 year, respectively, revealed a complete resolution of her vocal cord paresis with no phonatory gap. The vocal cord cyst disappeared completely most likely due to the treatment with dexamethasone postoperatively. The neck wound had healed well and she had a well-formed surgical scar.

DISCUSSION

Vocal cord paralysis is one of the most recognised complications following thyroidectomy. This condition is attributed to the close anatomical proximity of the recurrent laryngeal nerves as well as the external branch of the superior laryngeal nerves to the thyroid gland. It may be unilateral or bilateral, and could be transient or permanent. Permanent vocal cord paralysis should be considered if there is no improvement after 6 months. Vocal cord paralysis following thyroid surgery results in various symptoms that affect the voice, respiration, swallowing and coughing. The acute clinical presentation varies depending on whether the paralysis is unilateral or bilateral. Patients with unilateral vocal cord paralysis may present with hoarseness with or without a breathy voice, while patients with bilateral cord paralysis may develop more severe symptoms such as breathlessness, stridor and aspiration. Breathlessness and stridor indicate upper airway obstruction. Aspiration can be due to alteration in the sensory innervation supplying the laryngeal mucosa or ineffective motor innervation to the laryngeal muscles during laryngeal closure.

There are a few causes of vocal cord paralysis following thyroid surgery. Invasion of tumour into the nerves in the case of a thyroid carcinoma is a known direct cause for persistent vocal cord paralysis. It has been reported that the incidence of vocal cord paralysis in thyroid surgery is about 50%.¹ The presence of adhesions, derangement of normal anatomy in recurrent goitres and a huge multinodular goitre which is a highly vascular mass that can bleed massively during surgery, are factors that can obscure the surgical field, and make identifying the nerve intraoperatively potentially difficult. However, it has been reported that a small nodule or cyst located strategically at the tracheoesophageal groove can cause compression to the nerve as well.³ Transient vocal cord paralysis post thyroid surgery was also seen in a case of extralaryngeal ramification of the recurrent laryngeal nerve in the past.⁴ Another possible cause of transient vocal cord paralysis in thyroid surgery is due to neuropraxia while searching for the nerves with the aid of an IONM.⁵

The external branches of the superior laryngeal nerves are in close relationship with the superior pole of the thyroid glands and have a variable course in relation to the superior thyroid artery. In view of its highly variable course, identification of the nerve is essential to avoid injury during surgery.⁶ On the other hand, the recurrent laryngeal nerve is located in proximity to the inferior pole of the thyroid glands. Therefore, the inferior thyroid arteries are ligated away from the glands to avoid injury to the nerves. The use of IONM to identify the recurrent laryngeal nerves and superior laryngeal nerves during thyroid surgery

has become popular in the past few decades. Although studies cannot prove that the IONM is superior as compared to the direct visualisation of the nerves, nevertheless the IONM helps a surgeon to identify, confirm and monitor the nerve function intraoperatively, therefore minimising the risk of nerve injury.⁷ The use of a nerve stimulator is advisable in the case of difficult access to the nerves, or in training centres where many surgeons-in-training are doing the surgery. In other words, the IONM works as a safeguard for the nerve during thyroid surgery.

The nerves are first stimulated at 1–2 mA and it produces muscle twitching according to the nerve's distribution. When the nerve and its branches are visible directly, the stimulation is decreased to 0.4–0.5 mA in order to avoid overstimulation of the nerve. Donellan *et al*⁸ reported that the average stimulation for normal functioning nerves was around 0.44 mA, whereas abnormally functioning nerves responded to a higher stimulation of 0.62 mA. Occasionally, stimulation may no longer be needed once the nerve is identified and can be followed through. Overstimulation may cause conduction of stimulus along the involved nerves and this may impair the conduction velocity.⁷ In our case, the transient vocal cord paralysis could probably have been attributed to the less experienced surgeon as the surgery was performed by a young surgeon at our training centre. Literature shows that the experience of surgeons is associated with lower complications of the operations.⁹ Over and above this, a larger impact could have been due to the persistent stimulation of the nerves at high setting while searching for the nerves using the conventional technique of anatomical landmark, that is, the inferior thyroid artery, and the triangle, as well as the frequent stimulation with high current despite gross identification of the nerves intraoperatively. However, the blame should not be fully shifted to the surgeon as the monitoring of the system by other operating staffs also takes part to the outcome of the surgery.

The IONM is a safe and reliable method in identifying the recurrent laryngeal nerves and superior laryngeal nerves in thyroidectomy, but it should be used judiciously, as overstimulation of the nerves may cause debilitating outcomes such as in this case. It may be wise for the surgeon to check the proper placement of the electrodes, the appropriate setting of the device as well as to check the stimulus threshold of the IONM periodically during the course of surgery.

In the case of life-threatening respiratory distress secondary to bilateral vocal cord paralysis, an emergency tracheostomy may be required. The reason for reintubation in this patient was because she developed worsening stridor at the recovery bay most probably secondary to the laryngeal oedema. However, after prompt treatment with intravenous steroid, the laryngeal oedema had resolved. Therefore, tracheostomy was not performed for this patient although she had bilateral vocal cord paresis. Spontaneous recovery of vocal cord paralysis has been reported in the range of 79–87.5%.^{1 2} Literature shows that patients with transient vocal cord paralysis may show recovery as early as 3 days to 3 months.¹⁰ Although the role of oral methylcobalamine in vocal cord paralysis is not well understood, it has been shown that methylcobalamine promotes regeneration of axon in optic nerve crush injury in adult rats.¹¹ It has been postulated that this medication possesses neuroprotective properties, which may help in recovery following nerve injury. Speech therapy plays a critical role in the management of patients with bilateral vocal cord paralysis, as most of these patients may develop aspiration symptoms. The lady in this case was taught a proper vocal technique as well as some manoeuvres

to avoid choking during swallowing. She made a complete recovery 3 months postthyroidectomy without the need for a tracheostomy. On discharge, the patient must be given reassurance, and in any event of acute respiratory distress, the patient needs to come to the emergency department immediately. In bilateral vocal cord paralysis, surgical treatment is postponed between 6 months to 1 year from the onset of paralysis. The aim of surgery is to improve breathing, but patients must be aware that the procedure may worsen their voice quality. Several surgical procedures have been practiced, including endoscopic vocal cord lateralisation, endoscopic laser cordotomy and laser arytenoidectomy. Reinnervation has been proposed but is still under clinical evaluation.

Learning points

- ▶ The intraoperative nerve monitoring (IONM) is a safe and reliable method in identifying the recurrent laryngeal nerves and superior laryngeal nerves in thyroidectomy, but it should be used judiciously, as overstimulation of the nerves may cause debilitating outcomes.
- ▶ In any surgery requiring the aid of a nerve stimulator, it is wise for the surgeon to exercise caution and be vigilant in the setting of the stimulator in order to ensure a successful outcome with minimal avoidable complications.
- ▶ Any gadget, tool or equipment is no substitute for sound anatomical knowledge and meticulous surgical technique.

Contributors All authors have contributed equally in the: conception and design, acquisition of data or analysis and interpretation of data and drafting the article or revising it critically for important intellectual content. Final approval of the version published.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

- 1 Lo CY, Kwok KF, Yuen PW. A prospective evaluation of recurrent laryngeal nerve paralysis during thyroidectomy. *Arch Surg* 2000;135:204–7.
- 2 Hazem MZ, Naif AA, Ahmed AS. Recurrent laryngeal nerve injury in thyroid surgery. *Oman Med J* 2011;26:34–8.
- 3 Irfan M, Jihan WS, Shahid H. Unusual presentation of a solitary thyroid cyst. *Ann Acad Med Singapore* 2010;39:68–9.
- 4 Sancho JJ, Pascual-Damieta M, Pereira JA, et al. Risk factors for transient vocal cord palsy after thyroidectomy. *Br J Surg* 2008;95:961–7.
- 5 Brown WF, Bolton CF, Aminoff MJ. *Neuromuscular function and disease: basic, clinical and electrodiagnostic aspects*. W.B Saunders Company, 2002:1857–65. Chapter 104.
- 6 Aina EN, Hisham AN. External laryngeal nerve in thyroid surgery: recognition and surgical implications. *ANZ J Surg* 2001;71:212–14.
- 7 Higgins TS, Gupta R, Ketcham AS, et al. Recurrent laryngeal nerve monitoring versus identification alone on post-thyroidectomy true vocal fold palsy. *Laryngoscope* 2011;121:1009–17.
- 8 Donellan KA, Pitman KT, Cannon CR, et al. Intraoperative laryngeal nerve monitoring during thyroidectomy. *Arch Laryngol Head Neck Surg* 2009;135:1196–8.
- 9 Harness JK, Fang L, Thomson NW. Total thyroidectomy: complications and technique. *WJS*. 1986;10:781–5.
- 10 Khan JS, Malik K, Khawaja ZH, et al. Recurrent laryngeal nerve in thyroid surgery: is routine identification necessary? *J RMC* 2008;12:16–18.
- 11 Kong X, Sun X, Zhang J. The protective role of mecobalamin following optic nerve crush in adult rats. *Yan Ke Xue Bao* 2004;20:171–7.

Copyright 2014 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Become a Fellow of BMJ Case Reports today and you can:

- ▶ Submit as many cases as you like
- ▶ Enjoy fast sympathetic peer review and rapid publication of accepted articles
- ▶ Access all the published articles
- ▶ Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow