ORTNER'S CARDIOVOCAL SYNDROME PRESENTING AFTER ENDOTRACHEAL INTUBATION FOR GENERAL ANAESTHESIA: A CASE REPORT

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SUMMARY

Aim:

To present a case of postoperative hoarseness which was initially attributed to endotracheal intubation but was later diagnosed as Ortner's cardiovocal syndrome.

Methodology:

This is a clinical case presentation of 70-year-old man who underwent prostatectomy under general anaethesia with endotracheal intubation.

Result:

The patient developed hoarseness on the 3rd postoperative day and an exacerbation of his congestive cardiac failure. Hoarseness slowly resolved with successful management of congestive cardiac failure.

Conclusion/ Recommendation:

Ortner's syndrome is a cause of postoperative hoarseness and anaesthesia providers should be aware of it as a potential aetiology since early recognition and correction of underlying pathology is critical to a successful recovery.

Key words: *Postoperative Hoarseness, Ortner's syndrome, anaesthesia.*

INTRODUCTION

Post-operative hoarseness is a recognized complication of endotracheal intubation. Important aetiologies are airway instrumentation with resultant oedema, granuloma or nodule formation, haematoma, arytenoids dislocation, and vocal cord paralysis[1.] However, the development of unilateral vocal cord paralysis postoperatively and consequent hoarseness may also be caused by abnormal cardiac and/or vascular anatomy induced by specific disease states. Several disease-induced changes in cardiovascular and pulmonary anatomy cause impaired conduction through the left recurrent laryngeal nerve [2-6].

We present a case that occurred postoperatively in a patient with left atrial enlargement secondary to mitral regurgitation and significant pulmonary hypertension whose hoarseness was initially attributed to endotracheal intubation for a surgical procedure carried out under general anaesthesia.

CASE REPORT:

A 70-year-old man with a history of compensated congestive heart failure, chronic atrial fibrillation, and mitral regurgitation presented for prostatectomy because of benign prostatic hypertrophy.

His preoperative haematology and serum biochemistry were normal. A chest radiograph demonstrated marked cardiomegaly and a tortuous aorta. Electrocardiogram showed atrial fibrillation with ventricular rate of 70 beats/min, and a prominent U wave.

Options to have the prostatectomy done under regional or general anaesthesia were discussed with the patient. The patient opted to have general anaesthesia and gave written consent for the surgery.

A wide-bore intravenous catheter was inserted in a peripheral vein and infusion of lactated Ringer's solution was started to run slowly. Monitoring of the patient when under general anaesthesia was by non-invasive blood pressure measurement, electrocardiography, and pulse oximetry

After an intravenous induction with thiopentone and suxamethonium, the trachea was intubated easily and uneventfully with a well-lubricated 8.0mm oral endotracheal tube. Excellent visualization of the vocal cords indicated normal appearance and symmetrical relaxation. Maintenance anaesthesia included halothane, morphine, and atracurium. The lungs were ventilated manually with oxygen /air mixture. The period from intubation of the trachea to extubation at the end of the operation was one hundred minutes.

Twenty-four hours after the surgery, the patient was transferred to the intensive care unit because of progressive shortness of breath, tachypnoea, and hypoxaemia (SPO_2 of 85%).

Oxygen therapy was instituted to correct the hypoxaemia. A chest radiograph demonstrated marked cardiac enlargement with prominent pulmonary vasculature and bilateral pleural effusion.

A diagnosis of congestive heart failure exacerbation was made, and the cardiologist (PIC) was invited for evaluation and treatment. On the 3rd postoperative day, the patient complained of hoarseness. He remained hoarse at the time of his discharge from the hospital on the 15th postoperative day.

On the 21st postoperative day, the patient was evaluated by an Otorhinolaryngologist because of persistent hoarseness. He saw *"paramedian paralysis of the left vocal cord that structurally appeared grossly normal"*. Patient continued to be treated as an outpatient for repeated exacerbation of his congestive heart failure. At 5 months postoperatively he continued to experience hoarseness, though improving. A follow-up consultation and indirect laryngoscopy by an Otorhinolaryngologist

revealed "*satisfactory adduction of both vocal cords and near resolution of hoarseness"*.
The patient continued to have minimal hoarseness up to the time of last review, 6 months post-operatively.

DISCUSSION

Ortner's cardiovocal syndrome is a clinical entity manifested by hoarseness caused by an impaired ability of the left recurrent laryngeal nerve to transmit impulses to laryngeal musculature because of stretching or impingement of the nerve from disease induced changes in cardiac or great vessel anatomy. It was first described in 1897 by Ortner in patients with left atrial enlargement secondary to mitral valve stenosis[7]. Subsequently, it has been reported to occur in a variety of cardiopulmonary disease states including thoracic aneurysm; patent ductus arteriosus, primary pulmonary hypertension, atrial and ventricular septal defects, Eisenmenger's syndrome and recurrent pulmonary embolism[2-6].

The incidence of left vocal cord paralysis in patients with disease induced changes in cardiac and vascular anatomy is infrequent. However, patients with mitral stenosis, tend to be at increased risk, demonstrating frequencies of 0.6% to 5%[8.] . Although our patient did not have mitral stenosis, he did have significant mitral regurgitation with left artial enlargement in addition to pulmonary hypertension. Ortner deduced that, in the presence of mitral stenosis, the enlarged left atrium pushed the laryngeal nerve upward compressing it against the aortic arch resulting in ischaemic injury and degeneration of nerve fibres.

However, subsequent anatomical and radiographic studies have made Ortner's original theory less plausible than other possibilities according to some investigators. Fetterolf and Norris used cadaveric studies to demonstrate that the distance between the aorta and the pulmonary artery within the aortic window is only 4mm[9]. Therefore, they concluded, that the primary mechanism of injury must include compression of the left recurrent laryngeal nerve between the left pulmonary artery and the aorta at this location.

This theory was further supported by Ari et al when they found the left recurrent laryngeal nerve being compressed between an enlarged left pulmonary artery and aorta near the ligamentum arteriosum in two patients with mitral stenosis and hoarseness who were undergoing mitral commissurotomy[10].

In summary, postoperative hoarseness has many potential causes. These include those with anaesthesia implications such as laryngeal trauma during laryngoscopy, extreme head rotation, flexion or extension, excessive or asymmetrical endotracheal cuff inflation, prolonged intubation, or nasogastric tube use, or transoesophageal echocardiography probe placement. Additional causes include direct surgical trauma, pre-existing neuropathies, or medical conditions such as Ortner's cardiovocal syndrome. Differentiating factors among these potential aetiologies may be difficult to elicit. However, the majority of "anaesthetic-related" causes of postoperative hoarseness was generally evident immediately after extubation, and was resolved within 4 to 6 weeks[11, 12].

In the present case, our patient complained of hoarseness from the third postoperative day till 6 months. This chronological time frame coincides with the exacerbation of patient's congestive heart failure.

In the absence of direct surgical trauma and apparent anaesthetic complications, we believe factors related to the patient's cardiopulmonary status contributed significantly to the onset and subsequent resolution of his left vocal cord palsy.

Although, this may be a rare postoperative event, early recognition and rapid treatment or correction of the underlying cardiac or vascular anatomy is critical to a successful recovery. Therefore, we advocate that anaesthesia providers should be aware of Ortner's cardiovocal syndrome as a potential cause of postoperative hoarseness. Patients should also be informed preoperatively that they may experience hoarseness if these risk factors are known to be present.

REFERENCES

1. Friedman M, Toriumi DM. Esophageal stethoscope: another possible cause of vocal cord paralysis. Arch Otolaryngol Head Neck Surg 1989; 115:95 98.
2. Chan P, Lee CP, KO JT. Cardiovocal (Ortner's) Syndrome: Left recurrent Laryngeal nerve palsy associated with cardiovascular disease. Eur J. Med 1992; 1: 492 – 495.
3. Kagal AE, Shenoy PN, Nair KG. Ortner's syndrome associated with primary pulmonary hypertension. J postgrad Med 1975; 21:91–95.
4. Sengupta A, Dubey SP, Chaudhuri D. Ortner's syndrome revisited. J Layngol Otol 1998; 112:377-378.
5. Thirlwall AS. Ortner's syndrome: a centenary review of unilateral recurrent laryngeal nerve palsy secondary to cardiothoracic disease. J. Laryngol Otol 1997; 111:869-871.
6. Stocker HH, Enterline HT. "Cardiovocal syndrome" laryngeal paralysis in intrinsic heart disease. AM Heart J 1958; 56:51-59.
7. Ortner's NI. Recurrenslahmung bei Mitralstenose. Wien Klin Wochenschr 1897; 10: 753-755.
8. Solanki SV, Yajnik VH. Ortner's Syndrome. Indian Heart J 1972; 4:43-46.
9. Fetterolf G, Norris G. The anatomical explanation of the left recurrent laryngeal nerve paralysis found in certain cases of mitral stenosis. AM J Med Sci 1911; 141:625-638.
10. Ari R, Harvey WP, Hufnagel CA. Etiology of hoarseness associated with mitral stenosis: improvement following mitral surgery. AM Heart J 1955;50:153-160.
11. Holley HS, Gildea JE. Vocal cord paralysis after tracheal intubation. JAMA 1971; 215:281-284.
12. Hahn FW, Martin JH, Lillie JC. Vocal cord paralysis with endotracheal intubation. Arch Otolaryngol 1970;92:226-229.