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Airway management in a retrosternal goitre with tracheal narrowing – a case report

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Introduction

Retrosternal goitre: A goitre is considered mediastinal if atleast 50% of the thyroid tissue is located intrathoracically[1]. Goitres are often idiopathic, however it can be caused due to,

1. Iodine deficiency
2. Goitrogens
3. Hereditery
4. Autoimmune disease
5. Malignancy[2]

Retrosternal goitre may lead to the compression of the surrounding structures such as trachea, oesophagus, nerves and great vessels. These furthur can lead symptoms such as dyspnoea, dysphagia, hoarseness of voice and facial plethora.[2]

Compression over the trachea will lead to airway obstruction, difficulty in breathing, hypoventilation, impaired gas exchange resulting hypercarbia and hypoxemia. Compression can be partial or complete. Partial compression will have insidious onset whereas the complete obstruction will be rapid which will cause hypoxia and may even cause cardiac arrest.

Compression over superior venacava(SVC) will lead to SVC syndrome gradually progressive leading to oedema and retrograde flow furthur leading to cough, dyspnoea, dysphagia, swelling or discoloration of face, neck and upper extremity. Engorgement of the veins over the neck and upper chest will be seen.

Case Summary

A 65 years old male weighing 67 kg hailing from shivamogga who is a known case of retrosternal goiter with airway compromise and SVC syndrome presented with progressive shortness of breath since 3 years, worsened past 3 months, hoarseness of voice, difficulty in swallowing food, palpitations, anxiety and unable to lie flat during sleep since 3 months.

He is on Tab. Methimazole (10mg thrice daily) which was started two years ago and Tab. Propranolol (40mg thrice daily) since 3 months

On examination, the neck is edematous and there is presence of engorged veins over the left side of neck and upper chest (fig. 1).

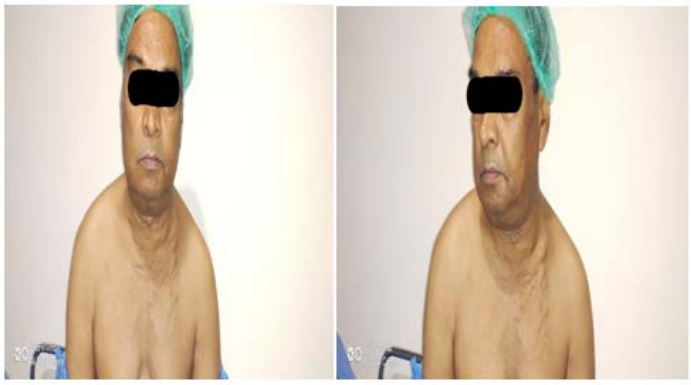

Figure 1 : Anterior and lateral aspect of neck showing engorged veins.

On further history evaluation, vital signs normal. He had adequate mouth opening and the Mallampati score was 2. On visual direct laryngoscopy epiglottis was tubular and bilateral vocal cords not visualised. His systemic examination showed no other abnormalities.

Blood count, renal profile and arterial blood gas were normal.

Currently patient was in Euthyroid state (Thyroid function test was normal)

Chest radiograph noted a large thyroid mass extending retrosternally to the level of the carina on the right side. The trachea was narrowed and diaphragmatic palsy on right side(fig.2)

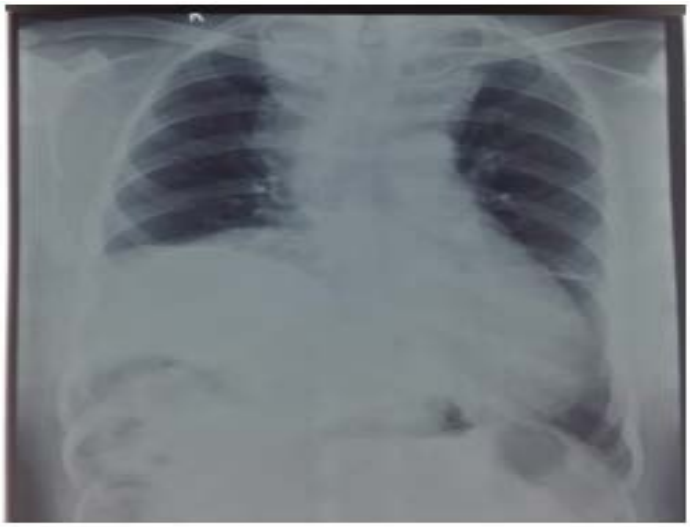

Figure 2 : Chest radiograph showing extension of mass into the mediastinum

Computed tomographs (CT) of neck and thorax confirmed that there is a well defined solid mass in superior mediastinum, it has a dense calcified area in inferior most part at the level of arch of aorta, the lesion appears to be in continuation with thyroid superiorly. Both lobes of thyroid appear to be enlarged and few calcified areas are noted in both lobes. There is extension of lesion into retro- tracheal area almost encasing it upto the level of tracheal bifurcation. The inferior most part of the lesion is almost at the level of the ascending aorta. It is in between the trachea, ascending aorta and superior venacava. Superior venacava compression is seen. There is severe narrowing of the airway at the level of hyoid bone with edema of soft tissue planes of larynx. The lesion extends towards paravertebral area from inlet of neck level upto carina. Right hemidiaphragm is significantly elevated (fig. 3)

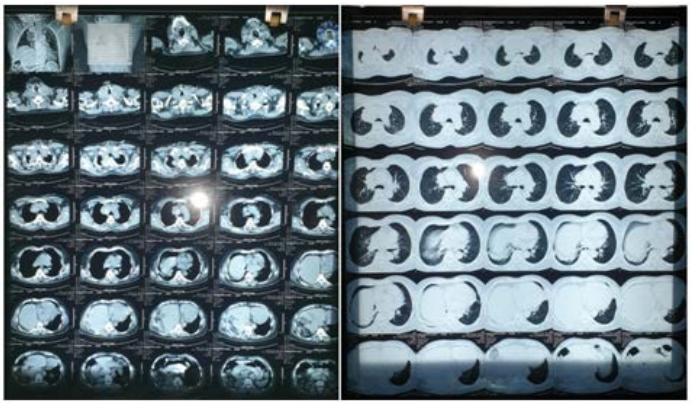

Figure 3 : Computed tomographs (CT) of neck and thorax showing mass in superior mediastinum and narrowing of airway at the level of the hyoid bone.

Comprehensive contingency plans were developed preoperatively involving a cardiothoracic surgeon, otorhinolaryngologic (ORL) surgeon and anesthesiologists emphasizing on the management of difficult airway and acute cardiorespiratory decompensation following induction of anesthesia[3]. A written anesthetic consent was taken after explaining the procedure of Awake Flexible Fiber-optic intubation along with explanation focused on

risks of difficult airway, consequences of hypoxia and mediastinal mass syndrome following induction of anesthesia.

A preoperative fasting of eight hours for solid food and two hours for clear fluid was followed. Premedication consisted of nebulized lignocaine (2%, 60 mg) in sitting position, 2 drops of Otrivin nasal drops and 2% Lignociane jelly in both nostrils was applied. 10 % lignocaine spray was sprayed twice to the posterior pharyngeal wall to achieve adequate airway anesthesia and intravenous (IV) glycopyrrolate (200 mcg) as an antisialagogue to prepare for awake flexible fiberoptic intubation (FOI). No sedative premedication was given. The right radial artery was cannulated under local anesthesia and the patient was put on American Society of Anesthesiologists standard monitoring. Awake FOI was performed under infusion of dexmedetomidine titrated at 0.2 -0.3 mcg/kg/h without the administration of a prior loading dose.(6) Once the glottic structures were identified, 4% lignocaine (40 mg) was sprayed directly onto the glottic inlet. Another 40 mg of 4% lignocaine was sprayed below the vocal cords to provide further airway anesthesia via **spray as you go** technique. Tracheal intubation was done with 8.0 mm cuffed PVC endotracheal tube (fig.4)

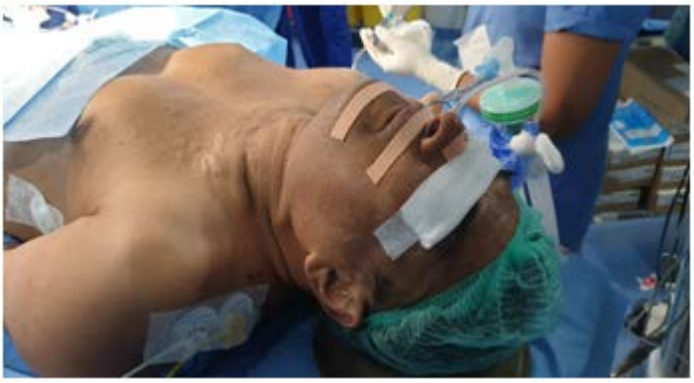

Figure 4: Positioning of patient after intubation.

No deterioration in hemodynamics or difficulty in delivering positive pressure ventilation was demonstrated following administration of propofol (100 mg, IV) and sevoflurane at minimum alveolar concentration of 1.0 after intubation. His blood pressure and heart rates were maintained within 15% of the baseline values.

Maintainance of anesthesia was done with sevoflurane + Oxygen + nitrous oxide. Injection vecuronium bromide muscle relaxant was given adequately for the procedure. Surgical manipulation did not lead to any tracheal compression and inadequate ventilation or complication of desaturation. The total thyroidectomy specimen (fig. 5) altogether measuring 17x13x6.8 cm was successsfully removed after sternotomy (one lobe was measuring 12x7x5cm and other lobe measuring 11x6x4.5cm) with minimal blood loss and the trachea was patent, negating the need for elective tracheostomy. Direct laryngoscopy performed at the end of surgery demonstrated a Cormack and Lehane grade one view of the glottic inlet. The patient was extubated with all due precautions. No respiratory compromise was noted after extubation and he had an uneventful recovery.

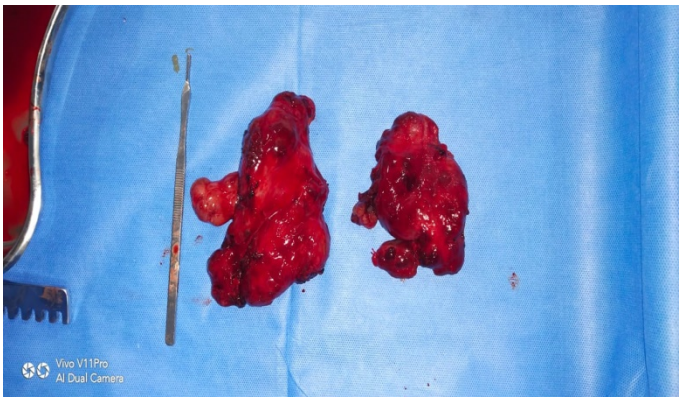

Figure 5: Total thyroidectomy specimen

Discussion

Mediastinal mass syndrome (MMS) describes a clinical condition caused by a mediastinal mass in patients undergoing anesthesia[4]. It can occur at every stage of anesthesia up to the postoperative period[5] or even simply by a change of posture[6]. Acute respiratory and/or cardiovascular decompensation can occur. The tumor may

lead to total airway occlusion by direct mechanical compression of the trachea, main bronchi or both whereas may result in cardiovascular catastrophe which can be potentially fatal by external compression of major vessels (i.e. the pulmonary artery and superior vena cava) or even the heart. In our case, there is extension of mass into retro-tracheal area almost encasing it upto the level of tracheal bifurcation. The inferior most part of the mass is almost at the level of the ascending aorta. It is in between the trachea, ascending aorta and superior venacava. Superior venacava compression is seen. There is severe narrowing of the airway at the level of hyoid bone.

A review of 98 adult patients with anterior or middle mediastinal masses who had undergone over one hundred anesthetics demonstrated perioperative cardiorespiratory complications 14.3% of the time[5]. The cases mainly comprised of lymphoma (21.0%), followed by thymoma (15.3%). Only 8.2% of these patients presented with an intrathoracic goiter and thyroid carcinoma. The authors concluded the presence of cardiorespiratory symptoms and signs at presentation are associated with perioperative complications. Other high risk factors are the presence of pericardial effusion or tracheal compression of greater than 50% as well as mixed restrictive and obstructive patterns on pulmonary function tests. However, none of the patients experienced airway obstruction during anesthesia despite some cases having severe compression of the tracheobronchial tree or peak expiratory flow rates of less than 40% of predicted values. We did not subject our patient to dynamic tests like flow-volume curves in relation to positional change as the patient was asymptomatic despite CT evidence of critical airway narrowing. In fact, Slinger and Karsli[7] proposed that the assessment is not beneficial as flow-volume loops have been shown to be poorly correlated with the degree of airway obstruction and have not demonstrated usefulness in management. Nevertheless, the challenges of anesthetizing this patient could not be underestimated.

Comprehensive contingency plans on airway management had been developed with the involvement of anesthesiologists, a general surgeon, an ORL and cardiothoracic surgeons. The availability of rigid bronchoscopy with jet ventilation would offer rescue in the event of loss of airway control[8].

On the other hand, positioning change may help to relieve the mass effect of tumors[6]. Induction was done on an operating table that enabled immediate changes in the patient's position. It is vital to identify the most 'comfortable' position in terms of respiration and hemodynamics in those symptomatic patients prior to surgery[4]. A tiltable operating table also allows checking for obstruction of airways in relation to positioning changes via the bronchoscope[3]

In our case, Awake FOI was performed under sedation with dexmedetomidine. Awake FOI and awake tracheostomy are the recommended approaches in managing anticipated difficult airways[3]. We provided adequate airway anesthesia via nebulized lignocaine, induced with propofol after securing the airway and maintained with sevoflurane, oxygen, nitrous oxide and muscle relaxantion with injection vecuronium and reversed with combination of neostigmine and glycopyrolate.

Conclusion

A comprehensive preoperative planning and a close working relationship among multidisciplinary medical teams were prerequisite for successful delivery of anesthesia and uneventful recovery of this patient. A structured in-house clinical protocol should be developed and deployed in order to ensure safe management of such patients in the future[3]

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