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Response of Post-Thyroidectomy Laryngeal Oedema to Frusemide: A Case Report

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Abstract: Postoperative airway complications after thyroid surgery remain a nightmare for both the surgeons and anaesthesiologists. Recurrent laryngeal nerve palsy, hypocalcemia and haematoma are commonly encountered after total thyroidectomy. Laryngeal oedema remains a less known entity. We report a case of laryngeal oedema where we were saved of any airway instrumentation by the response of frusemide.
Keywords: Post-thyroidectmy, delayed, laryngeal oedema, stridor, fruesmide

INTRODUCTION

Thyroid surgery in recent years is considered quite safe. But the postoperative complications can be potentially life threatening. The two most common complications after thyroid surgery are recurrent laryngeal nerve palsy and permanent hypothyroidism[1]. Laryngeal oedema, although less reported, is a very serious complication as it can be fatal if not managed timely[2].

CASE REPORT

A 23 year old female weighing 50 kg was admitted in our surgery ward with thyroid carcinoma. She was posted for total thyroidectomy with bilateral radical neck node dissection. Preoperative examination revealed moderate sized thyroid swelling of about 5cm x 4cm confined to the neck with no retrosternal extension or pressure effects. Clinical examination and normal values of thyroid function tests suggested that the patient was euthyroid. She was not on any medications.

Preoperative indirect laryngoscopy showed normal vocal cord movements. Lateral and straight x ray of the neck did not reveal any compression or deviation of the trachea. Preoperative histopathology report was suggestive of malignancy. Other routine investigations were within normal limits.

The patient was adequately premedicated with Alprazolam (0.25 mg) and Ranitidine (150 mg). She was then shifted to the operation theatre and standard monitors attached. She underwent an uneventful surgery (duration of 6 hours). There was no significant blood loss. Before closure, suction drain was put and compression bandage applied. Vocal cord movements were examined by direct laryngoscopy and were found to be normal. Patient was observed in the recovery room for 30 min and then sent to the ward.

In post operative period she was advised IV calcium gluconate (10 ml 6 hourly), IV hydrocortisone (100 mg 12 hourly), antibiotics and analgesics. For the initial two days patient was asymptomatic though she had mild hoarseness of voice. On third post operative day, she was allowed sips of water. On ingestion of first sip of water, patient coughed and could not swallow. Within 2-3 min, she developed respiratory difficulty and inspiratory stridor. Immediately, oxygen was delivered via ventimask (FiO_2 0.4) and patient put in propped up position. She was given IV hydrocortisone 100mg and IV calcium gluconate, but the symptoms did not subside. Respiratory distress and inspiratory stridor progressively increased. As the patient did not respond to the medical treatment, surgeons decided to re-explore the surgical wound for any local hematoma.

Patient was shifted to operation theatre and examined by the anaesthesiologist. Her respiratory rate was 60/min, SpO2 88% in room air, heart rate 136/min and BP 128/70 mm of Hg. Surgical wound appeared healthy, without any local swelling and was not painful to touch. She was given oxygen at FiO_2 1.0 and was kept in propped up position. Within a minute, SpO_2 increased to 100%, heart rate settled down to 110/min and respiratory rate was 52/min but the stridor

persisted. Direct laryngoscopy was done (after lignocaine instillation).The movement of vocal cords were normal. Since the patient was stable on oxygen therapy and wound appeared healthy surgical intervention was deferred.

As other causes of airway complications were ruled out, IV frusemide 20 mg was given in anticipation of laryngeal oedema. She continued to receive 100% oxygen. Stridor began to decrease and within 10 min she showed marked improvement. The respiratory rate came down to 30/min and heart rate was 90/min. Symptoms were relieved within 20 min and patient was comfortable. Patient was observed for a period of 6 hours in the post operative room and advised frusemide (20mg) six hourly. Indirect laryngoscopy was done on fourth day and vocal cord movements were found to be normal. She was allowed oral fluids from fifth post operative day and was later discharged. There were no further episodes of respiratory distress.

DISCUSSION

Thyroid surgery poses a challenge in the postoperative period due to airway complications which are not only multifactorial but also threatening if diagnosis is delayed. We faced the above case of post thyroidectomy inspiratory stridor following water intake. It could have been due to haematoma, hypocalcemia or recurrent laryngeal nerve palsy[3]. These complications were ruled out as discussed below.

Hypocalcaemia:

Post thyroidectomy hypocalcaemia usually occurs after 72 hours[4]. But our patient did not complain of perioral tingling, neither did she develop Chovestok's sign, carpopedal spasm or Trousseau's sign. Moreover, parathyroid implantation was carried out and patient was given calcium gluconate six hourly. Even on appearance of stridor calcium gluconate administration did not relieve the symptom.

Haematoma:

It is a rare, but a serious complication post operatively[1]. It could be due to slipping of ligature, bucking, vomiting or increased blood pressure during recovery. We had a smooth neuromuscular blockade reversal. In our case, negative suction drain was put and strap bandage applied. There was no increase in the size of the wound or significant blood in the suction drain.

Recurrent laryngeal nerve palsy:

Though the glottic aperture is considerably narrowed in this palsy it does not cause significant respiratory obstruction unless laryngeal oedema is present as well. Usually there is biphasic stridor, dyspnoea and difficulty in coughing immediately after extubation[1].

Our patient had an indirect laryngoscopy before operation and another direct laryngoscopy after operation and during the stridor (after lignocaine spray instillation). Both the views suggested apparent normal vocal movements. Surgery was very meticulously performed by a team of highly experienced surgeons. But the patient had mild hoarseness of voice.

Laryngeal oedema:

After excluding the possible causes of airway obstruction we were left with a rare and less reported complication, laryngeal oedema, which may have been precipitated by stimulation of the airway. The airway in thyroid surgery(especially in total thyroidectomy) remains inflammed due to its close proximity with the procedure and handling of tissue. When the patient tried to take a sip of water she probably choked leading to stimulation/aspiration.

Wade JSH mentioned in his study that the narrowest part of the upper respiratory tract (apart from the glottic chink when the vocal cords are in adduction) is the subglottic area of the larynx just below the cords at the level of the cricoid cartilage. The mucosa here is extremely lax and, if congested, will rapidly swell and obstruct the lumen. The mucosa of the larynx above the cords is also lax but not to the same extent as that of subglottic region[5]. Swelling here is unlikely to cause obstruction after thyroid surgery, but it does obscure the vocal cords on laryngoscopy and makes intubation extremely difficult, even for an experienced anaesthesiologist. The vocal cords themselves do not become oedematous because the lining epithelium of the cord is bound down to the underlying vocal ligament and there is no submucosal layer. Normally the vocal cords abduct during inspiration and the glottic chink is widened. If one or both cords are paralysed the obstruction caused by subglottic oedema is accentuated. M Christor and Athanassaiades S extensively studied 400 post thyroidectomy patients in which only 13.5% reported laryngeal oedema[6]. They observed that the incidence of laryngeal oedema was higher in younger patients with toxic goitres who underwent radical surgery which was similar to our case. They further remarked that it occurred in first and second day of surgery and was mostly supraglottic in nature. Our patient developed oedema when allowed orally in the morning of third day but oedema was more in the subglottic region as supported by the result of Wade JSH.

There are two important causes of laryngeal oedema in thyroid surgery: a haematoma deep to the pretracheal muscles and trauma to the laryngeal mucosa[5]. Haematoma is unlikely as surgery was

meticulous and post operative recovery uneventful in the first two days. Laryngeal trauma was difficult to ascertain as we lubricated the endotracheal tube prior to intubation and there was no sign of bleeding or trauma during extubation.

The patient had inspiratory stridor which indicated large airway (larynx) obstruction. Our patient responded to frusemide. She was also on hydrocortisone postoperatively. Both these drugs reduce the hydrophilic and inflammatory component of laryngeal and pulmonary oedema[7]. The beneficial effect of frusemide in laryngeal oedema has been supported by the study of Netterville J et al.[8]. J Prandonta described that through inhibition of production and release of cytokines, interleukins 6,8 and TNF alpha frusemide has a beneficial effect on local inflammed tissue[9]. Besides the onset of action of frusemide is approximately 20minutes when given intravenously which matched with our case[10].

It was difficult to determine subglottic oedema (laryngoscopy does not reveal subglottic region) and mild vocal cord dysfunction (view during stridor was difficult and there was a change in voice). Aspiration of water in the background of inflamed and oedematous laryngeal mucosa with suspected mild vocal cord paresis accentuated the stridor. This fact could have been substantiated by a fibreoptic visualization which was not done as we feared sedation or topical application of local anaesthetics might not be sufficient for introduction of the scope as the patient was fully awake. Besides, a deep plane of anaesthesia was not desirable as the patient had just recovered from a hypoxic episode.

CONCLUSION

Laryngeal oedema may occur postoperatively after thyroid surgery. It is difficult to rule out among other causes of airway obstruction as it is not only rare but also difficult to diagnose. Before considering re-exploration, laryngeal oedema should be kept in mind as this may prevent airway manipulation (intubation may be both difficult and may also aggravate oedema). A commonly used drug like frusemide (besides hydrocortisone) may reduce laryngeal oedema and save any untoward complication.

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