Pulmonary edema after Thyroidectomy: Case report and review

S Sharma, M Samplay, S Varshnay

Citation

S Sharma, M Samplay, S Varshnay. *Pulmonary edema after Thyroidectomy: Case report and review*. The Internet Journal of Anesthesiology. 2007 Volume 18 Number 2.

Abstract

Pulmonary oedema after extubation is a rare complication. But it has been described in adults as well as in healthy children. A case of a sixty years old gentleman who developed laryngeal stridor, vocal cord paresis and pulmonary oedema early after emergence from general anaesthesia is reported. Possible causes, management and a brief review of literature is discussed.

CASE REPORT

A 60-year-old, 73kg male presented with a history of neck swelling [right euthyroid thyroid nodule] for one-year. The nodule had rapidly increased in size during the last 3 months and was accompanied with hoarseness of voice for one month.

His Co-morbid conditions were Type –2 diabetes mellitus controlled on oral hypoglycemics and a well-controlled hypertension. The patient was euthyroid [T_3 _ 90 ng/dl, T4-7.0mcg/dl, TSH-1.84 mIU]. There was no history suggestive of a MEN syndrome.

The patient was conscious and alert with no pain in his neck. Vitals were normal. [HR 82/min., BP 125/74mm HGg, R.R.16/min.], CVS, Chest and abdomen were clinically normal. Echocardiography was normal, with LVEF 60%,

His neck examination revealed a diffuse thyroid swelling of size 8 cms x 6 cms on the right side of neck pushing trachea to the left, with no obvious retrosternal extension. Cervical nodes were impalpable

FNAC [Histopathology] report indicated medullary carcinoma thyroid.

On preoperative indirect laryngoscopy his vocal cords were bilaterally mobile.

On Chest X-ray a minimal left sided pleural effusion was suspected but Chest CT showed no underlying parenchymal lesion, lymphadenopathy or pleural effusion.

CT scan of the neck and thorax revealed a large right thyroid nodule [6.7cms in supero-inferior, 4.3cms in transverse and 5.4 cms in antero-posterior diameter] with marked tracheal shift without compression towards left [contra lateral] side, pushing the carotid artery laterally and compressing the internal jugular vein.

The patient was accepted as ASA Gr. II patient for Total thyroidectomy with central compartment node dissection .A consent for postoperative ventilation and tracheotomy [if required] was taken in view of the anticipated damage of the recurrent laryngeal nerve on the right side. The patient was premedicated with tab. alprazolam 0.25 mg and tab. ranitidine 150 mg P.O. two hours prior to surgery.

At induction of anesthesia, intravenous Fentanyl 125 ?g, Inj. Midazolam 1mg, Inj. ondansetron 4mg, Inj. Glycopyrrolate 0.2mg, Propofol 100mg, Inj.Vecuronium 6mg were administered with IPPV. The airway was secured with 8.5 size cuffed disposable endotracheal tube.

The vital parameters like HR, ECG, NIBP, SpO2, EtCO2, and temperature were monitored during the course of surgery.

The patient was explored through a collar incision. The strap muscles were densely adherent to the thyroid. The sternothyroid was dissected with the gland. There was densely adherent hard infiltrating nodule in the right lobe with dense fibrous adhesions to the anterior and lateral surface of the trachea. The esophagus was also adherent and was pulled up and medially. Parathyroid and recurrent laryngeal nerve could not be identified on the right side. The left recurrent nerve and parathyroid were identified and

preserved with a small sleeve of thyroid. There were no obviously enlarged lymph nodes in the central or lateral compartment so only a central compartment dissection was done. Serum Calcitonin sample was sent postoperatively.

The intraoperative course of anesthesia was essentially uneventful. The duration of surgery was 90 minutes. Intraoperative blood sugar was found to be 120 mg %. The estimated blood loss during surgery was around 200 ml. 1.5 litres of IV crystalloids were infused.

The neuromuscular blockade was reversed using neostigmine and glycopyrrolate at the end of surgery.

Direct laryngoscopy done before extubation revealed feebly mobile vocal cords on both sides. Post extubation the laryngoscopy was repeated, it showed the approximation of both the vocal cords in mid line with minimal movement.

The patient's respiration was observed and intermittently assisted manually with 100% oxygen, in preparation for a probable reintubation. A few minutes after extubation the patient's SpO2 started falling and reached upto 78%, with tachycardia [134/min] and hypertension [BP 180/110mm of Hg.] He showed marked tracheal tug and intercostals indrawing. The patient could not be maintained further on IPPV through facemask,

His spontaneous respiratory efforts reduced. He was reintubated and put on IPPV after IV propofol 40mg and succinylcholine 50mg. At this stage, there was no laryngeal oedema .The airway pressure was high [42cms. of H_2O] with a SpO_2 of 94%, and tachycardia. [H.R. 112-120/min.]

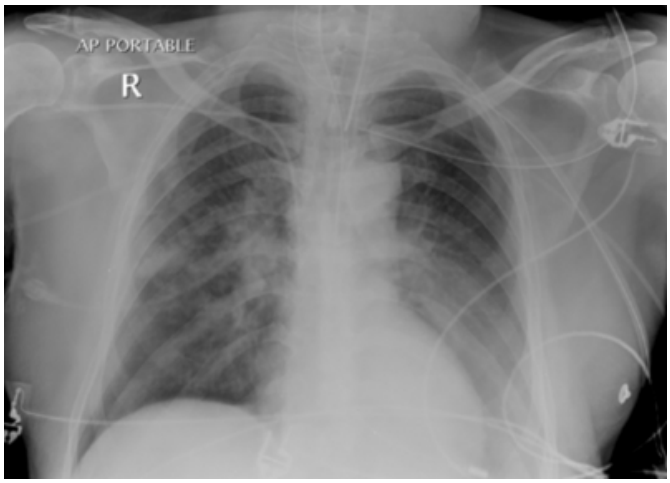
After 5-6 breaths of IPPV, pinkish frothy sputum was noticed in the endotracheal tube. There was an associated fall in B.P to 68/40 mm of Hg. On auscultation, air entry was found to be bilaterally equal with coarse crepitations all over the chest.

IPPV with O2, Inj. Frusemide 40 mg I.V, Hydrocortisone 100mg and Morphine 3 mg was given suspecting pulmonary oedema, Infusion of Inj.Dopamine @ 8 microgram/Kg /min. was also started. . PEEP 10cm H2O was added once BP stabilized to 140/90 mmHg. The patient was shifted to MICU on IPPV for observation and further management. Patient was catheterized postoperatively and 150 ml of high colored urine output was recorded.

Immediate Postoperative X-ray chest showed bilateral lung infiltration and minimal left pleural effusion. [Fig.1].

Figure 1

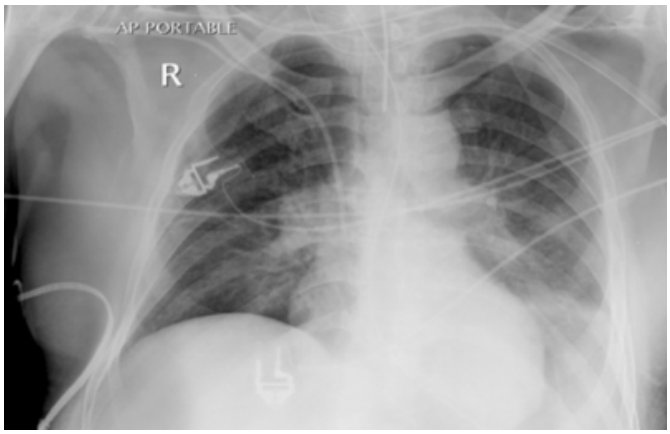
Figure 1: Immediate Post operative X-Ray Chest



On Day 2: X-ray Chest showed clearing of pulmonary infiltrates. [Fig.2]

Figure 2

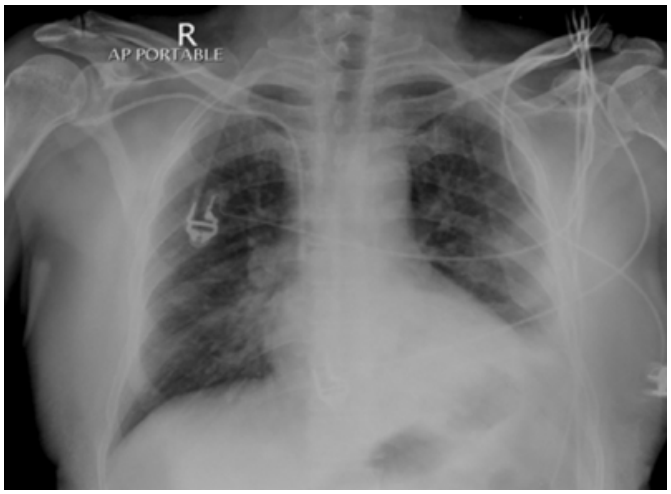
Figure 2 : Day 2 ,X-Ray Chest



By Day 3, a marked improvement in the X-ray chest was present.[fig.3]

Figure 3

Figure 3 : Day 3 –X-Ray Chest



Arterial Gasometry [ABG] had shown an acute respiratory acidosis with normal electrolytes in the early postoperative period. Repeat ABG, an hour later showed normocapnia. On day 2 before check bronchoscopy, drop in oxygen saturation was noted [Table-1] which improved after the procedure.

Figure 4

Table 1: ABG reports

	Time	pH	PaCO2	PaO2	Lac.	SPO2 [%]	HCO3	BE
1.	Day 1 [Post op.]	7.217	59.9	204	0.8	98.6	23.5	-4.9
2.	Day 1 [One hour later]	7.39	40.5	95	0.7	96	20.2	–5.1
3.	Day 2 [Pre- bronchoscopy]	7.506	40.5	66	1.0	94.9	21.5	-0.4

Bronchoscopy performed on Day 2 showed slight tracheomalacia with moderate laryngeal oedema and sluggish movement of vocal cords. The endotracheal tube was removed under bronchoscopic guidance and a trial of BIPAP through facemask was given. The patient was observed overnight in ICU and shifted to ward next day.

The Serum calcium level was noted to be low [8.1 gm %], so he was also put on calcium replacement therapy.

A day later the patient developed a significant stridor inspite of BIPAP, Direct laryngoscopy showed marked laryngeal oedema with sluggish movement of the cords with no apposition suggestive of bilateral abductor palsy.

Tracheostomy was performed. Patient was on spontaneous respiration after that and was discharged on the 11th day postoperatively.

He received radioactive iodine subsequently. He was off tracheostomy after 3 months of the operative procedure.

DISCUSSION

Pulmonary oedema after general anaesthesia, a rare complication has been earlier reported in literature subsequent to an acute airway obstruction [1,2,3,4], bilateral vocal cord paralysis [5] and also post Thyroidectomy. [6]

Most cases of negative pressure pulmonary oedema present within minutes after the relief of the obstruction [1,7] as occurred with our patient also.

The fall in SPO2 on extubation in our case can be attributed to mild tracheomalacia, bilateral abductor palsy, inadequate breathing efforts and development of pulmonary oedema.

Pulmonary oedema can be explained by the occurrence of persistent inspiratory efforts against closed/partially-closed glottis, creating a markedly negative intra-pleural pressure. Thus labeled as negative pressure pulmonary oedema [NPPE}.

Pathophysiologic mechanisms such as increased venous return to the right heart, interventricular septal shift, reduced left ventricular compliance, hyperadrenergic state due to hypoxia, and increased afterload, have also been suggested.

Haemodynamic changes in combination with the high negative hydrostatic pressure in the pulmonary interstitium change the starling forces of the pulmonary circulation favoring the transudation of fluid to the pulmonary interstitium and then into the alveoli.

Many authors insist that hypoxia plays the major role in the loss of pulmonary capillary integrity (increasing capillary permeability) and the subsequent pulmonary edema rather than the negative intrapleural pressures although recent publications indicate that this type of pulmonary oedema has a hydrostatic etiology without increased alveolo-capillary membrane permeability [8,10]

The pulmonary oedema in our patient responded well to diuretics, inotropes, endotracheal intubation IPPV and PEEP (with invasive followed by non invasive mode)

Radiological characteristics in this patient were typical of centralized oedema due to mechanisms described above.

Absence of peripheral edema on X-ray signified absence of capillary leak due to hypoxia.

Hydrostatic pulmonary edema can also be distinguished from increased permeability pulmonary oedema by measurement of the ratio of total protein concentration between pulmonary oedema fluid and plasma. A ratio of less than 0.65 is characteristic of hydrostatic pulmonary edema, whereas patients with increased-permeability pulmonary oedema have a ratio between 0.75 and 1.0.

We could not measure the protein concentration of the oedema fluid in our patient but the radiologic features were suggestive of hydrostatic pulmonary oedema.

CONCLUSION

We faced an unanticipated occurrence of pulmonary oedema in a sixty year old gentleman early after general anaesthesia, which required management with diuretics, inotropes and mechanical ventilation with PEEP for 24 hours.

In our opinion, the cause of this pulmonary oedema was mainly the strong negative inspiration pressure during emergence from anaesthesia, complicated by tracheomalacia and bilateral abductor paresis of vocal cords. The patient was tracheotomised for three months and treated with radioactive iodine. At present; the patient is doing well without tracheostomy and has no vocal cord paresis.

CORRESPONDENCE TO

Dr. Sunila Sharma, MD Senior Consultant and Head of the department, Fortis Flt. Lt. Rajan Dhall Hospital, Vasant Kunj, New Delhi 110070. India Email: sunila.sharma@fortishealthcare.com

References

1. Willms D, Shure D: Pulmonary Edema Due To Upper Airway Obstruction in
Adults. Chest 1988; 94:1090
2. Stuth EAE, Stucke AG, Berens RJ: Negative-pressure Pulmonary Edema in a
Child with Hiccups during Induction. Anesthesiology 2000; 93:282-4
3. Cascade PN, Alexander GD, Mackie DS: Negative pressure pulmonary edema after
endotracheal intubation. Radiology 1993; 186:671-5.
4. Kallet RH, Daniel BM, Gropper M, Matthay MA: Acute pulmonary edema
following upper airway obstruction: case reports and brief review. Respiratory Care
1998; 43:476-80
5. Dohi S, Okubo N, Kondo Y: Pulmonary edema after airway obstruction due to
bilateral vocal cord paralysis. Can J Anaesth 1991; 38:492-5
6. Manohar Lal Sharma, Neal Bekett, Paul Gormley," Negative pressure pulmonary edema following thyroidectomy."Canadian journal of Anesthesia 49:215(2002)
7. Lang SA, Duncan PG, Shepard DA,et al: Pulmonary oedema associated with
airway obstruction. Can J Anaesth 1990; 37:210-8.
8. Devys JM, Balleau C, Jayr C, Bourgain JL: Biting the laryngeal mask: an unusual
cause of negative pressure pulmonary edema. Can J Anesth 2000; 47:176-8.
9. A.Petrou, K.Valmas, E.Svarna, G.Chortaria, A.Giamarelou, G.Papadopoulos," Negative pressure pulmonary oedema in a patient ventilated with laryngeal mask," The greek E- Journal of perioperative medicine 2003,1-69-73[ISSN 1109 -6888] www.anesthesia.gr/ejournal.
10. Kallet RH, Daniel BM, Gropper M, Matthay MA: Acute pulmonary edema
following upper airway obstruction: case reports and brief review. Respiratory Care
1998; 43:476-80

Author Information

Sunila Sharma
Senior Consultant and Head of the department

Mukesh Samplay
Associate Consultant, Fortis Flt. Lt. Rajan Dhall Hospital

Sanjiv Varshnay
Attending Consultant, Fortis Flt. Lt. Rajan Dhall Hospital