

Negative pressure pulmonary oedema after routine elective surgery

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ABSTRACT

Negative Pressure Pulmonary Oedema (NPPE) is known to occur in healthy subjects in the early post anaesthesia period, in the absence of fluid overload or left ventricular dysfunction. This type of non cardiogenic pulmonary oedema is also reported in literature following upper airway obstruction. We report two such cases of negative pressure pulmonary edema. Both the patients developed NPPE during postanaesthesia period due to persistent laryngospasm leading to upper airway obstruction. Both the patients were treated with diuretics, bronchodilators & antibiotics in intensive care unit. Within 24 hours the coarse crepitations disappeared and the patients were weaned off the ventilator. Awareness, early recognition and prompt treatment of negative pressure pulmonary oedema could be life saving.

Key words: Fluid extravasation, negative pressure pulmonary oedema, post anaesthesia, laryngospasm

Introduction

Negative pressure pulmonary edema (NPPE) or post obstruction pulmonary edema (POPE) is a clinical condition of great importance in anesthesiology and intensive care. The presentation of NPPE can be immediate or delayed, and hence necessitates immediate recognition and treatment by person directly involved in the perioperative care of a patient. [1, 2] The incidence of NPPE has been reported to be 0.05%–0.1% of all anesthetic practices; however, it is suggested that it occurs more commonly than is generally documented. [2] According to one review, NPPE develops in 11% of all patients and patients usually requiring active intervention for acute upper airway obstruction. [3] The Australian

monitoring study of 4000 incidences of laryngospasm during anesthesia showed that NPPE occur in up to 4% of all incident reports of laryngospasm. [4] This disorder is classified as Type I and Type II. [5, 6] Type I NPPE develops usually with acute upper airway obstruction or after manipulation of the airway surgically. It is also described as laryngeal spasm-induced pulmonary edema by some authors in the literature. [7] On the other hand Type II NPPE usually results after relief of upper airway obstruction caused by big tonsils, hypertrophic adenoids. The incidence of developing Type I NPPE associated with acute postoperative upper airway obstruction is 9.6–12%, whereas the incidence of developing Type II NPPE is 44%. [8] In adults

about 50% of NPPE occurrences are due to postoperative laryngospasm.^[9] Negative pressure pulmonary oedema is though uncommon is a serious life threatening problem. This type of non cardiogenic pulmonary oedema usually occurs due to upper airway obstruction secondary to persistent laryngospasm which can lead to forced inspiration against a closed glottis and results in rapid changes in intrathoracic, alveolar and interstitial pressures leading to extravasation of fluid from the pulmonary capillaries into the interstitial and alveolar spaces causing negative pressure pulmonary oedema. We report here two such cases of negative pressure pulmonary edema.

Case 1

50 year old female (weight 50 Kg, height 160 cm) presented with the history of abdominal pain along with vomiting off and on for 3 months. She was diagnosed as a case of Cholelithiasis and posted for Laproscopic Cholecystectomy. Patient was a known case of diabetes on regular human insulin and was well controlled. There was no relevant history of hypertension, tuberculosis, jaundice and oedema feet. Her vital signs were stable. X- ray chest, ECG and blood investigations were all normal. Cardiovascular and respiratory systems were normal on examination.

Premedication consisted of Inj. Ondansetron 4 mg, Inj. Midazolam 1.5 mg, Inj. Pentazocine 15 mg, Inj. Glycopyrrolate 0.2 mg given IV 10 minutes before surgery on Operation table after peripheral venous access was secured. She was put on routine monitoring which consisted of ECG, Pulse Oximeter, NIBP (Non-invasive blood pressure), End tidal CO₂. Induction was facilitated with propofol 100mg IV just

sufficient to loss of verbal commands. The trachea was intubated with a sterile cuffed endotracheal tube of size 7.5 mm internal diameter after paralyzing the patient with succinylcholine 100 mg. Anaesthesia was maintained with nitrous oxide, oxygen, intermittent doses of vecuronium and 0.6 to 0.8 per cent isoflurane. Additional 10 mg of inj.pentazocine 15 mg was given for intraoperative analgesia. The procedure lasted for 60 min. during which her vital parameters were within normal limits. She was infused with one litre of crystalloids during the surgery. At the end of procedure the patient was reversed with neostigmine 2.5 mg and atropine 1.2 mg. with the return of spontaneous respiration and reflexes patient was extubated after thorough tracheobronchial toilet. Ten minutes after extubation patient developed laryngospasm and got desaturated. This was managed with bag and mask ventilation with 100 per cent oxygen. Injection hydrocortisone hemisuccinate 100 mg was given intravenously. As the laryngospasm was persisted patient was sedated with injection midazolam 2mg IV and reintubated after giving 100mg succinylcholine. It was then noticed that frothy secretions were welling up though the endotracheal tube. The secretions became pinkish and increased in quantity so repeated tracheobronchial toilet was done. On auscultation of the chest revealed bilateral coarse crepitations. At this stage working diagnosis included fluid over load, acute myocardial infarction and NPPE (Negative Pressure Pulmonary Oedema). Fluid overload was excluded as she was given only one litre of Ringers lactate solution and acute myocardial infarction was excluded on the basis of a normal 12 lead ECG and normal level of serum cardiac

enzymes. This left us with a strong possibility of NPPE which was probably induced by the development of negative pressure due to the persistent laryngospasm. Chest x-ray reveals presence of alveolar & interstitial infiltrates in both lung fields.

She was sedated and connected to ventilator on volume controlled ventilation-tidal volume- 500ml, respiratory rate- 14/min, PEEP of 10 cm of H₂O, FIO₂ of 0.6. A central venous line and an arterial line were placed for monitoring of central venous pressure, arterial blood pressure and for estimation of arterial blood gases. The urine output was also monitored. She was treated with diuretic frusemide, bronchodilators and antibiotics in the ICU. Patient had fall in BP due to diuresis. Inj. Dopamine infusion was started which was later tapered off as patient vital becomes stable. Within 48 hours the coarse crepitations disappeared, the chest X-ray and blood gases returned to normal. The patient was then weaned off the respirator and the endotracheal tube was removed.

Case 2

A 38 yrs old male patient (weight-60kg, height-170 cm) with fracture both bones forearm was scheduled for plating under general anesthesia. He had no relevant history of hypertension, diabetes mellitus, jaundice, oedema feet. General physical examination showed short neck with no other significant findings. Vital parameters were normal. Chest had bilateral equal air entry and cardiovascular examination was normal. Preoperative investigations including Chest X ray and ECG were within normal limits. The patient was accepted for anesthesia as ASA I physical status and was premedicated with Inj. ondansetron 4 mg,

Inj. pentazocine 15 mg and Inj. midazolam 1.5 mg given intravenously on operation table after peripheral venous access was secured. After adequate preoxygenation anesthesia was induced with titrated dose of Inj. propofol 140 mg just sufficient to loss of verbal commands. Neuromuscular blockade was achieved with Inj. succinyl choline 100 mg. After 1.5 mins of assisted-controlled ventilation, the trachea was intubated with 8.5 sized cuffed portex endotracheal tube. Anesthesia was maintained in usual sequence with Isoflurane, oxygen and nitrous oxide with controlled ventilation through closed circuit. Neuromuscular blockade was maintained with Inj. vecuronium bolus dose and further incremental doses. . Additional 15 mg of inj.pentazocine 15 mg was given for intraoperative analgesia. At the end of surgery inhalation anesthetics were stopped and residual neuromuscular blockade was reversed with 2.5 mg neostigmine and atropine 1.2mg. The patient was extubated uneventfully and shifted to post operative recovery unit. Three hours post operatively; patient complained of difficulty in breathing along with fall in oxygen saturation and had froth containing blood tinted sputum. Patient was given high flow oxygen but saturation was not coming up. He was shifted to ICU, intubated with ETT no.7.5 cuffed after paralysis with 75 mg succinyl choline. ETT suctioning revealed pink frothy secretions and on auscultation bilateral coarse crepitations were present. ABG revealed low SaO₂ and PaO₂. Chest x ray revealed presence of alveolar & interstitial infiltrates. He was connected to ventilator on volume controlled ventilation with PEEP of 10mm of Hg, respiratory rate 15/min, tidal volume - 500ml and FiO₂ of 100. Central venous line

was inserted. FiO_2 was de escalated based on peripheral oxygen saturation. He was investigated for other causes of pulmonary edema including ECG, cardiac enzymes, and echocardiography, which were all normal. Patient was thus diagnosed as NPPE and ventilated overnight. Patient was treated with diuretic frusemide, bronchodilators and antibiotics in the ICU. Next day his chest was improved, FiO_2 was furthur de escalated to 40% and PEEP decreased to 8mm of Hg. Patient was weaned off from the ventilator on second day and shifted to ward. Patient was discharged from the hospital on seventh day after removing the sutures.

Discussion

The exact mechanism of development of NPPE is not very clear but literature reveals that it usually occurs due to persistent laryngospasm and upper airway obstruction. Persistent upper airway obstruction results in development of negative intrathoracic pressure due to forced inspiration against closed glottis. Due to this negative intrathoracic pressure venous return to right heart and pulmonary arteries is increased and consequently this results in transudation of fluid from the pulmonary capillaries into the interstitium which leads to interstitial & alveolar edema.

The patients usually develop NPPE immediately after obstruction or it can be delayed by 2-3 hours. The patients usually presents with stridor, difficulty in breathing and pink frothy sputum with consequently fall in the oxygen saturation. On auscultation bilateral coarse crepitations are heard in both lung bases. Chest radiograph reveals alveolar and intestinal infiltrates in both lungs.

The patients presented here is one in which laryngospasm resulted in the development of Non Cardiogenic Pulmonary oedema. As the oxygen saturation decreased markedly the patient's trachea was reintubated and maintained on ventilatory support in the ICU. She was monitored and treated with diuretics, bronchodilators, steroids, and antibiotics. The patient recovered within 48 hours. The trachea was extubated when patients were awake, breathing well spontaneously and blood gas levels returned to normal. Post-operative recovery of the patients was otherwise uneventful.

The management of our case with NPPE was similar to that reported by other authors in the literature. The important aims in the management of NPPE are to maintain oxygenation, ventilation and to reduce the preload. Initially 100 per cent oxygen can be given by a facemask.^[10, 11, 12] If the PO_2 cannot be maintained at 60 mmHg or if there is progressive hypercapnia the patient should be intubated and mechanically ventilated. Application of PEEP of 5-20 cm of H_2O is beneficial in these patients as it can decrease the preload and after load thereby improving cardiac function. This also results in redistribution of the fluid in the lungs from the intra alveolar to the extra spaces where it does not interfere significantly in gas exchange and also prevents atelectasis.^[13] These advantages of PEEP should be weighed against the risk of inducing barotraumas and furthur reduction of cardiac output. Reduction of preload can also be done by the loop diuretic frusemide, bumetamide. Fursemide is commonly used as it is venodilator and can reduce the preload rapidly.^[12]

Morphine also acts as transient venodilator and helps to reduce the preload in addition to relieving dyspnea and anxiety. These measures can decrease stress leading to reduction of catecholamine levels, tachycardia and ventricular afterload in pulmonary oedema. In our study reported here, the treatment was on similar lines with oxygenation, diuretics, sedation and mechanical ventilation. Steroids and bronchodilators were given in our cases to reduce the laryngeal oedema and to improve ventilation. To conclude one must have understanding of NPPE as cause of non cardiogenic pulmonary oedema as early perception and prompt treatment results in reversal of the condition and can be life saving.

References

1. Timby J, Reed C, Zeilender S, Glauser FL. "Mechanical" causes of pulmonary edema. *Chest Care* 1990;98:973–9.
2. McConkey PP. Postobstructive pulmonary oedema: A case series and review. *Anaesthesia Intensive care*. 2000 Feb;28(1):72-6.
3. Tami TA, Chu F, Wildes TO, Kaplan M. Pulmonary edema and acute upper airway obstruction. *Laryngoscope* 1986;96:506–9.
4. Visvanathan T, Kluger MT, Webb RK, Westhorpe RN. Crisis management during anaesthesia: laryngospasm. *Qual Saf Health Care* 2005;14:e3.
5. Dicipinigaitis PV, Mehta DC. Postobstructive pulmonary edema induced by endotracheal tube occlusion. *Intensive Care Med* 1995;21:1048–50.
6. Anderson AF, Alfrey D, Lipscomb AB, Jr. Acute pulmonary edema: An unusual complication following arthroscopy: A report of three cases. *Arthroscopy* 1990;6:235–7.
7. Guinard JP. Laryngospasm-induced pulmonary edema. *Int J Pediatr Otorhinolaryngol* 1990;20:163–8.
8. Lathan SR, Silverman ME, Thomas BL, Waters WC. Postoperative pulmonary edema. *South Med J* 1999;92:313–5.
9. Goli AK, Goli SA, Byrd RP, Jr, Roy TM. Spontaneous negative pressure changes: An unusual cause of noncardiogenic pulmonary edema. *J Ky Med Assoc* 2003;101:317–20.
10. Moore RL, Binger CA. The response to respiratory resistance: A comparison of the effects produced by partial obstruction in the inspiratory and expiratory phases of respiration. *J Exp Med* 1927;45:1065–80.
11. Holmes JR, Hensinger RN, Wojtys EW. Postoperative pulmonary oedema in young athletic adults. *Am J Sports Med* 1991;19(4):365-71.
12. Sharma ML, Beckett N, Gormley P. Negative pressure pulmonary oedema following thyroidectomy. *Can J Anaesth* 2002;49:215.
13. Hochman JS, Ingbar D. Cardiogenic shock and pulmonary oedema. In: Dan L Longo, Anthony S Fauci, Dennis L kasper, Stephen L Hauser, J Larry Jameson, Joseph Loscalzo, editors. *Harrison's Principles of Internal Medicine*. 18th ed. USA: McGrawHill; 2012.p.2232-2236.
14. Szucs R, Floyd H. Laryngospasm induced pulmonary oedema. *Radiology* 1980;170:446.
15. Van Kooy MA, Gargiulo RF. Post obstructive pulmonary oedema. *Am Fam Physician* 2000;62:401-4.

16. Jackson FN, Rowland V, Corssen G. Laryngospasm induced pulmonary oedema, *Chest* 1980;78:819-21.
17. Weissman C, Damask MC, Yang J. Non cardiogenic pulmonary oedema following laryngeal obstruction. *Anesthesiology* 1984;60:163-65.

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