ISSN 2515-8260 Volume 10, Issue 1, 2023

A RARE CASE OF NEGATIVE PRESSURE PULMONARY EDEMA

Type of article: Original Article

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1) Dr.Shahbaz Hasnain(Professor)

Second author

Department of Anaesthesiology and Critical Care

Dr.Dy Patil Medical College,Hospital,Research Centre

Pimpri,Pune 411018

Maharashtra, India

E-Mail: shahbazhsnn@gmail.com

2) Dr.Waseema Kabeer (Second Year Resident)

Corresponding author

Department of Anaesthesiology and Critical Care

Dr.Dy Patil Medical College,Hospital,Research Centre

Pimpri,Pune 411018

Maharashtra,India

E-Mail:waseema040194@gmail.co.in

3) Dr.Surya Teja (Second Year Resident)

First author

Department of Anaesthesiology and Critical Care

Dr.Dy Patil Medical College,Hospital,Research Centre

Pimpri,Pune 411018 Maharashtra,India

E-Mail:suryajteja@gmail.com

Department(s) and institution(s)- Department of Anaesthesiology, Dr. D.Y. Patil Medical College, Hospital and Research Centre, Pimpri, Pune Dr. D.Y. Patil Vidyapeeth, Pune, Maharashtra 411018 India.

Corresponding Author:

Name: Dr. Waseema Kabeer

Address: Department of Anaesthesiology, Dr. D.Y. Patil Medical College, Hospital and Research Centre, Pimpri, Pune Dr. D.Y. Patil Vidyapeeth, Pune, Maharashtra 411018 India.

Phone numbers: 9884028248

Facsimile numbers: NIL

E-mail address: waseema040194@gmail.com

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HIGHLIGHT

Negative pressure pulmonary edema (NPPE) or post obstructive pulmonary edema occurs due to forced inspiratory efforts against an obstructed airway. It is dangerous and life threatening if not promptly diagnosed and treated. Patients generate a high negative airway pressure and hydrostatic forces imbalance, which increases transvascular fluid infiltration and precipitates interstitial and alveolar edema. Recovery is rapid with adequate supportive measures, endotracheal intubation or cricothyroidectomy and oxygen. We have discussed about the steps on how to relieve laryngospasm, extubation, treatment and appropriate ventilator settings and investigations. We have focused on its early identification and management to prevent mortality and further complications which is informative for all anaesthesiologists.

KEY WORDS: Negative pressure Pulmonary edema, Upper airway obstruction, post extubation laryngospasm, anaesthesia, ICU.

CASE PRESENTATION

NPPE is a type of non cardiogenic PE that occurs due to increased negative intra-thoracic pressure after inspiration against a closed glottis. Due to this pressure the pulmonary capillaries are affected which changes the equilibrium of the hydrostatic pressure causing extravasation of fluid into the lung parenchyma and alveoli[1]. It causes severe hypoxia and PE[2]. In anaesthesia, post extubation laryngospasm and upper airway obstruction can cause this complication. This occurs in 0.05% to 0.1% of cases as a serious complication of general anesthesia with tracheal intubation[3], and more than half of these cases are related to post-anesthetic laryngospasm[4]. The true incidence, however, is not known and may be higher, since many cases may have been misdiagnosed because of a lack of familiarity with the syndrome. The incidence of NPPE is more than 50% among men following laryngospasm[5] during intubation or in the postoperative period after anesthesia.

Other than upper airway obstruction, there may be other pathophysiological disorders making patients more vulnerable to PE. Based on these disorders, upper airway obstruction is the trigger to induce NPPE.

This is a case of a 48 year old male posted for endoscopic retrograde cholangiopancreatographic (ERCP) stent removal under general anaesthesia. Pre operative routine investigations were done and within normal limits.Patient was a known case of type 2 diabetes mellitus;non compliant with medication. However, his blood sugar level and HbA1C was normal. General and systemic examination were

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normal. Baseline blood pressure was 130/80mmHg, Pulse rate 78/min, SpO_2 98% on room air and fitness was given under ASA2.

Intra-operatively,basic monitors were attached and baseline readings were within normal limits. Patient was pre-oxygenated with 100%O_2 and premedicated with 1mg midazolam,0.2mg glycopyrrolate and120mcg fentanyl. Induced with Propofol 2mg/kg and after confirming ventilation succinylcholine 2mg/kg was given and intubated with 8.5mm cuffed endotracheal tube.Vecurronium 0.1mg/kg was given and maintained with Sevoflurane on Volume control mode. The short and uneventful procedure lasted about 1 hour15 minutes. Post procedure, after achieving adequate respiratory efforts and tone patient was reversed with Inj Neostigmine 0.05mg/kg and Inj Glycopyrrolate 0.4mg/kg. And extubated after oropharynx suctioning.

Immediately after extubation,patient was unable to talk and was seen gasping,agitated with drop in SpO_2 upto 75% and PR152/min,BP150/90mmHg,respiratory rate 35/min with paradoxical breathing noted. Laryngospasm was quickly identified and 100%O_2 with manual positive pressure ventilation was given using anatomical face mask. Succinylcholine 50mgIV and Hydrocortisone 100mg IV given while ventilating.On auscultation there was no air entry in bilateral basal lung zones so patient was desaturating on room air, so re-intubated with 8mm ETT and put on pressure control mode. Salbutamol and Budecort MDI were administered via ETT. Inj.Furosemide 20mg IV and Adrenaline 0.1 mg/kg IM was given. He was paralyzed with Vecurronium 5mg.Etophylline 85mg and Theophylline 25mg IV and second dose of Hydrocortisone 100mg given. On rechecking air entry, mild improvement noted. He was shifted to surgical intensive care unit after stabilisation for further management.

In ICU, he was conscious and oriented with PR 130/min, BP 140/90mmHg, room air saturation of 89% with tachypnoea. He was ventilated with pressure support/continuous positive airway pressure (PS/C-PAP) with higher positive end expiratory pressure (PEEP) of 8-10 cmH_2O. On auscultation, air entry reduced with infrascapular region basal crepts present.Arterial blood gas analysis showed respiratory alkalosis.Chest x-ray showed haziness suggestive of ?NPPE, ?mucous plug or ?aspiration. Correct placement of ETT confirmed and endobronchial intubation ruled out that may show a similar picture on x-ray. Plan of treatment was over night ventilation,IV antibiotics,diuretics,bronchodilators,steroids,nebulization with bronchodilators,adrenaline and steroids,4^{th} hourly ABG,fluid restriction,chest x-ray coming morning followed by extubation. 2d Echocardiography was normal and repeat chest x-ray showed reduction in haziness.ABG was normal. Room air saturation was 87%. A negative cuff leak test with coughing,difficulty in breathing when cuff was deflated suggested persistent vocal cord edema. Hence the plan to extubate was

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postponed.Steroids and antibiotics were stepped up and 4th hourly nebulization given with BSL monitoring.Next day cuff leak test was repeated and it was positive suggesting reduction in the vocal cord edema. Rapid shallow breathing index was 49 predicting successful weaning off and extubation. A good cough reflex to ETT suctioning, strong ETT blast and well tolerated t-piece trial for over 8 hours on room air without desaturation noted. Repeat chest x-ray had vast reduction in haziness.ABG was normal and auscultation was clear.100%O_2 given for 2 minutes and he was extubated with positive pressure during inspiration on post operative day 1. Nebulization with adrenaline was repeated and the patient was comfortable and vocalizing.

He was observed in ICU upto POD 3 and did not show any desaturation, tachycardia and breathing difficulty.

Once the patient was maintaining saturation on room air he was shifted to ward with stable vitals.

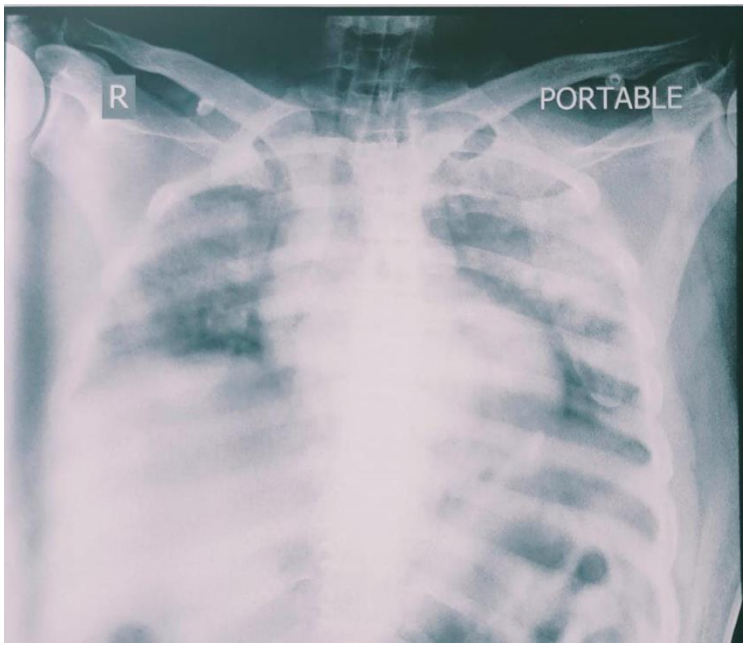

Fig 1-Immediate post op chest x-ray

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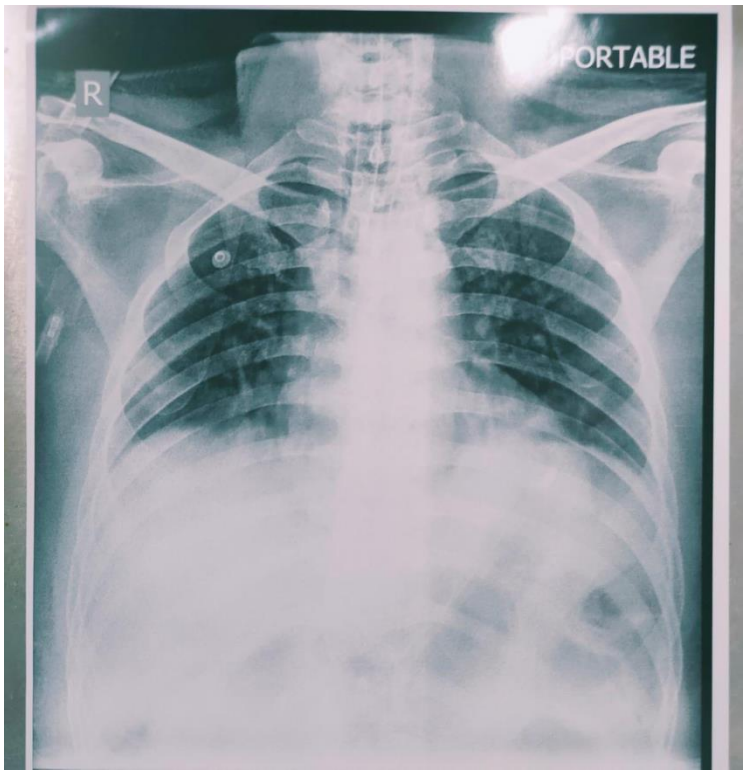

Fig 2- chest x-ray on POD 2

There are two main mechanisms that seem to be responsible for NPPE.

NPPE is more common in young patients who can generate a high amount of intrathoracic pressure of upto -140 cmH_2O. This high negative inspiratory pressure is transmitted to the intrapleural spaces resulting in increased venous return; and pulmonary venous pressure; decreasing the perivascular interstitial hydrostatic pressure causing movement of fluid from capillaries to interstitium and alveoli. UAO causes the patient to be in a hyperadrenergic state which causes peripheral vasoconstriction and an increase in venous return further increasing pulmonary blood flow and edema. NIP increases the left ventricle afterload causing increase in LV wall tension and decreases the ejection. The resulting hypoxemia decreases myocardial contractility and increases pulmonary arterial resistance. The fall in left ventricular ejection fraction increases the end-diastolic pressure, left atrial pressure, and pulmonary venous pressure, further increasing the pulmonary capillary hydrostatic pressures that promote the formation of PE. This causes a disturbance in Starlings equilibrium causing PE.[6]

The second mechanism is a break in the alveolar epithelium and pulmonary micro vasculature due to mechanical stress of respiration against an obstructed airway resulting in increased pulmonary capillary permeability and protein-rich PE.

In our case PE associated with aspiration was excluded as the patient followed strict NBM prior to surgery. Mucous plug was excluded on High resolution computed

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tomography (HRCT) of the thorax. Drug associated allergy was also ruled out by taking a thorough pre-anaesthetic history. Residual paralysis was also ruled out by confirming the presence of swallowing reflex and good breathing attempts. NPPE usually progresses rapidly within minutes from the onset of airway obstruction to the development of PE. It is more common in healthy ASA 1 males as they are capable of generating more negative intrathoracic pressures.[7] Other common causes of NPPE could be ETT biting, foreign body aspiration, epiglottitis, upper airway tumours, post tonsillectomy and adenoidectomy.[8]

CONCLUSION

Prompt diagnosis of NPPE due to post extubation laryngospasm, management by a skilled anesthetist and experienced intensivist led to the successful outcome of this rare complication. It can be prevented by gentle management of airway, minimal tracheal irritation, and extubation in an adequate plane of anaesthesia to prevent the occurrence of laryngospasm. NPPE usually resolves in 12 to 48 hours with supportive treatment without the need of prolonged ICU stay.

REFERENCES

1. Bhattacharya M, Kallet R, Ware L, Matthay M. Negative-Pressure Pulmonary Edema. Chest. 2016;150(4):927-933.

2. Xiong J, Sun Y. Negative pressure pulmonary edema: a case report. BMC Anesthesiology. 2019;19(1).

3. Albergaria VF, Soares CM, Araujo RdeM, et al. Negative-pressure pulmonary edema after transsphenoidal hypophysectomy. Case report. Rev Bras Anestesiol 2008;58:391–396.

4. Liu R, Wang J, Zhao G, Su Z. Negative pressure pulmonary edema after general anesthesia. Medicine. 2019;98(17):e15389.

5. Lee J, Lee J, Lee M, Cho H, Park S. Postoperative negative pressure pulmonary edema following repetitive laryngospasm even after reversal of neuromuscular

ISSN 2515-8260 Volume 10, Issue 1, 2023

blockade by sugammadex: a case report. Korean Journal of Anesthesiology. 2017;70(1):95.

6. Lemyze M, Mallat J. Understanding negative pressure pulmonary edema. Intensive Care Medicine. 2014;40(8):1140-1143.

7. Tebay A, Bouti K, Tebay N. Œdème pulmonaire à pression négative après une cholécystectomie – à propos d'un cas. Revue de Pneumologie Clinique. 2017;73(5):267-271.

8. Silva L, Guedes A, Filho M, Chaves L, Araújo F. Negative pressure pulmonary edema: report of case series and review of the literature. Brazilian Journal of Anesthesiology (English Edition). 2019;69(2):222-226.