

Negative Pressure Pulmonary Oedema: Management in Resource-Challenged Hospital: Two-Case Reports

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Abstract

Negative Pressure pulmonary oedema (NPPO) is a medical emergency. It occurs when there is a strong inspiratory effort in obstructed upper airway. Laryngospasm is the main cause of postextubation negative pressure pulmonary oedema. Though it is life-threatening, early diagnosis and prompt treatment lead to rapid resolution with no residual respiratory complications. The mainstay management is to provide respiratory support, mostly in the intensive care unit. The recommended mode of respiratory support is to provide an invasive or non-invasive positive airway pressure. This requires the use of a ventilator. Most surgery centres in sub-Saharan Africa do not have intensive care unit or ventilators in their recovery wards. We report two cases of postextubation NPPO which occurred in a typical African hospital with no ventilator. All these two cases were successfully managed with a non-rebreather mask. The periods of resolution, both clinical and radiological, were 24 - 48 hrs. This is not significantly different from the resolution periods quoted in literature from cases managed in well-resourced centres with means of positive pressure ventilation. We therefore conclude that early detection and prompt initiation of management are important keys which can lead to good outcomes, even in low-resource centres.

Keywords

Pulmonary Oedema (PO), Negative Pressure Pulmonary Oedema (NPPO)

1. Introduction

Pulmonary oedema (PO) is the accumulation of fluid within the interstitium and air spaces of the lung. The causes of acute onset of perioperative pulmonary oe-

dema could be cardiogenic or noncardiogenic. The cardiogenic causes include heart failure. The non-cardiogenic causes in the perioperative period include neurogenic pulmonary oedema, acute respiratory distress syndrome (ARDS), anaphylaxis, aspiration pneumonitis, fluid overload and negative pressure pulmonary oedema (NPPO). Features of pulmonary oedema include dyspnoea, frothy sputum which may or may not be blood-stained, crackles and oxygen desaturation [1] [2].

NPPO occurs in patients with upper airway obstruction who make high inspiratory effort [1]. The cause of the obstruction could be a tumour, infection etc. [2] [3] [4]. The incidence of NPPO in general anaesthesia is 0.05% - 0.1% and the major cause is laryngospasm [2].

NPPO, just like any acute pulmonary oedema, is a medical emergency. Almost all the reported cases were managed in the Intensive Care unit. Most hospitals in Africa do not have intensive care units and also ventilators for postoperative ventilatory support. But when appropriately diagnosed, perioperative NPPO can be managed with a good outcome, even in a low-resourced centre. Appropriate diagnosis and timely intervention are essential for a good outcome. We present two cases of NPPO which occurred within a year apart at a level-II hospital in Ghana without an Intensive Care unit or a High Dependency Unit and no means of postoperative mechanical ventilatory support.

2. Case 1

A 17-year-old student with a reducible paraumbilical hernia had a hernia repair under general anaesthesia in a Level II hospital. Patient weighed 72 kg and was 1.76 m tall. Patient had no previous anaesthetic exposure. Patient was not on any medication, had no known allergy, did not take alcohol and did not smoke. He had no cough or history of cardiac disease. The chest was clinically clear. He had no known co-morbidity. He was ASA I.

The surgery was performed by a general surgeon and anaesthesia provided by a Certified Registered Anaesthetist.

Patient had an overnight fast, was induced with propofol and intubated with tube size ID 7.5 mm on suxamethonium. Anaesthesia was maintained with isoflurane, vecuronium (8 mg) and morphine (10 mg) under volume controlled ventilation. Intubation and intraoperative period were uneventful. Surgery lasted for two hours, blood loss was minimal and a total of 1 L of Ringer's lactate was given. At the end of surgery, muscle relaxants were reversed with neostigmine and atropine and patient was extubated and sent to the recovery ward.

Within 10 minutes of extubation, the patient's oxygen saturation (SPO₂) had dropped to below 70% despite the routine supplementary oxygen with nasal prongs at the recovery ward. The patient became restless and produced a frothy sputum. On review by a physician anaesthetist, the patient was restless, in respiratory distress, had tachypnoea and tachycardia and coarse crackles in both lung fields. Blood pressure was however stable. Patient was then becoming less

restless and more drowsy. A diagnosis of NPPO was made.

A bolus of IV furosemide 40 mg was given and subsequently needed to be continued. A supplementary oxygen which had been started with a nasal prong and subsequently with a simple face mask could not appreciably improve the patient's hypoxia and obtund, so a non-rebreather mask was obtained within two hours to continue with the supplementary oxygen. A chest x-ray showed bilateral diffuse opacification of both lung fields (**Figure 1(a)**). Over the subsequent 18 hours, all the parameters of the patient continued to improve and oxygen saturation could now be maintained at 100% with a nasal prong. By 24 hrs, the chest was clinically clear and the SPO₂ on room air was between 95% - 99%. A repeat chest x-ray on the second postoperative day showed clearing of the lung fields. (**Figure 1(b)**).

3. Case 2

A 25 year old male student (weight 70 kg, height 1.78 m) with a similar diagnosis of reducible paraumbilical hernia had hernia repair (component separation and overlay mesh repair) under general anaesthesia. The patient had not been exposed to anaesthesia before and did not have any known allergy. He had no cough, did not smoke and had no cardiovascular disease. His chest was clinically clear, had no features of difficult intubation and also had no co-morbidity. He was ASA I. Patient had an overnight fast.

Anaesthesia was induced with thiopentone and muscle relaxation achieved with suxamethonium. Endotracheal intubation was done with tube size ID 7.0 mm and was uneventful. Anaesthesia was maintained with isoflurane/vecuronium/morphine. The induction, intubation and the intraoperative course were uneventful. Patient was haemodynamically stable, SPO₂ was 98% - 100%. The surgery lasted for 2^{1/2} hrs and the blood loss was minimal. The total IV fluids given

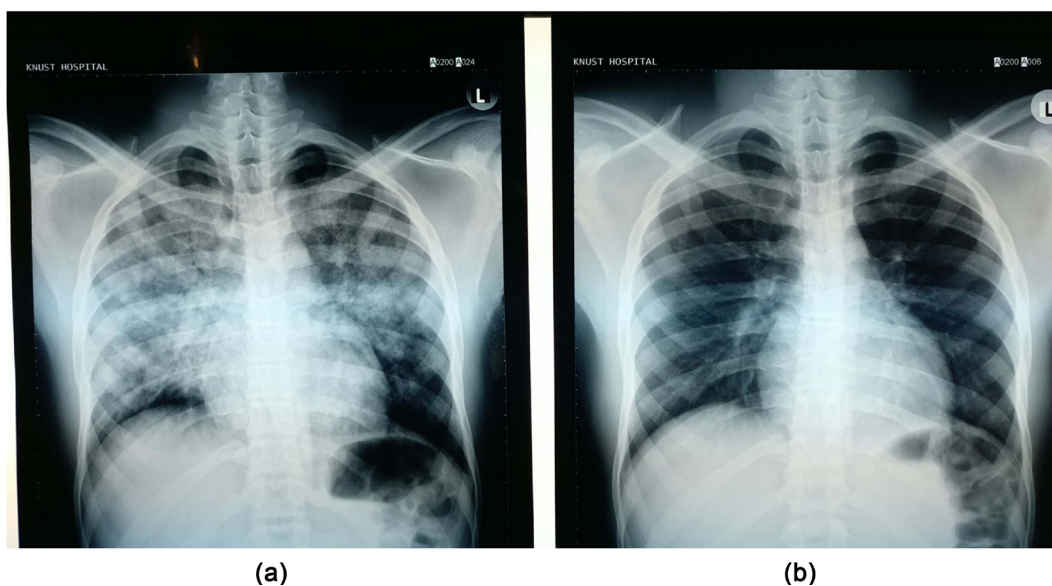


Figure 1. Beginning of treatment (a) and second posttreatmentday, (b) chest x-rays of case 1.

was 1.5 L of Ringer's lactate (500 mls preoperative and 1.2 L intraoperative). Residual muscle relaxants were reversed at the end of surgery with neostigmine and atropine. On extubation, patient developed inspiratory stridor which was managed with propofol (60 mg), manual ventilation 100% oxygen and CPAP with a face mask. Patient was then sent to the recovery ward. At the recovery ward, he began to cough out a pinkish frothy sputum. The SPO₂ then dropped to below 80% and patient became agitated, tachypnoeic and tachycardic. The blood pressure was however normal. Auscultation of the chest showed bilateral coarse crackles in both lung fields. A diagnosis of a NPPO was made. Oxygen supplementation was then changed from a simple face mask with a reservoir bag to a non-rebreather mask. A 40 mg bolus of iv furosemide was given and subsequently repeated 8 hrly. A chest x-ray showed a bilateral diffuse opacification of both lung fields consistent with pulmonary oedema (**Figure 2(a)**). Patient's clinical condition continued to progressively improve. Within 24 hours the SPO₂ could be maintained above 95% on room air and the crackles had cleared. A chest x-ray on the second postoperative day showed resolution of the bilateral diffuse opacification (**Figure 2(b)**)

4. Discussion

NPPO is a form of noncardiogenic pulmonary oedema. It occurs when excessive inspiratory effort is made to overcome an obstructed upper airway [1]. So theoretically, any form of upper airway obstruction in a spontaneously breathing patient can result in NPPO. The incidence of NPPO in upper airway obstruction is 12% [1]. In fact, the first reported cases of NPPO in literature was in 1977 and were not related to endotracheal intubation (hanging, tumour, and strangulation) [3]. The incidence of perioperative NPPO is 0.1% with majority occurring after extubation (74%) though can also occur during the initial airway management and postintubation [4]. Laryngospasm accounts for 50% of perioperative NPPO. NPPO can also occur in intubated patients in the ICU due to patient-ventilator-asynchrony [5].

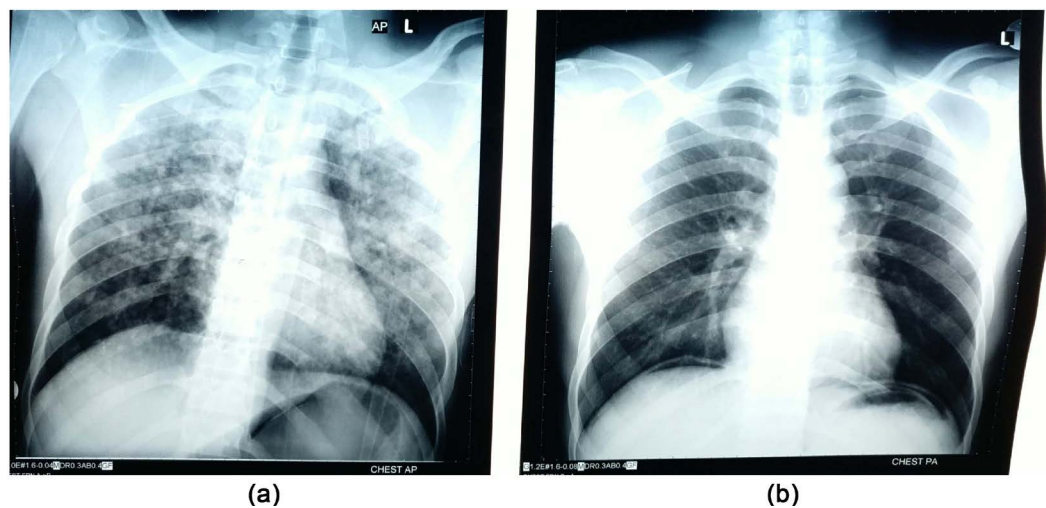


Figure 2. Beginning of treatment (a) and second posttreatment day (b) chest x-rays of case 2.

Both cases were young healthy males. The pathophysiology of NPPO stems from the generation of a high negative intrathoracic pressure induced by a strong inspiratory effort against an obstructed upper airway. Understanding the pathophysiology of NPPO would help in the early suspicion and tailored management. Starling's equation governs fluid homeostasis in the lung.

$$Q = K[(P_{mv} - P_i) - \sigma(\pi_{mv} - \pi_i)]$$

where Q is net fluid flux from the capillary lumen to the alveolar interstitium; K is the coefficient of capillary permeability; P_{mv} is the capillary lumen hydrostatic pressure; P_i is the alveolar interstitial hydrostatic pressure; s is the reflection coefficient (the effectiveness of the vascular barrier in preventing diffusion of protein); π_{mv} is the microvascular protein osmotic pressure; and π_i is the interstitial protein osmotic pressure.

In the normal steady state, there is a net movement of fluid from the capillaries into the interstitium, which is subsequently removed by the lung lymphatics and eventually returned into the systemic circulation [6]. When the rate of accumulation of interstitial fluid is higher than the rate of lymphatic removal, the oedema fluid may eventually flood the alveoli and appear as frothy sputum (which may or may not be pink) and opacities on chest x-ray [5] [6].

There are two widely accepted mechanisms for the development of NPPO secondary to an upper airway obstruction [7]. These are the: generation of negative intrathoracic pressure leading to substantial fluid shift and generation of mechanical stress in the lungs [3] [8]. In the first mechanism, when a substantial inspiratory effort is made against an obstructed airway, there is a generation of a high negative intrathoracic pressure which leads to an increase in the venous return to the right side of the heart and a subsequent increase in the pulmonary venous pressures. This increases the transpulmonary hydrostatic gradient which results in increase movement of fluid from pulmonary capillaries into the interstitium and alveolar spaces. There is also a hyperadrenergic state associated with the upper airway obstruction which causes peripheral vasoconstriction, increase in venous and therefore further contributing to the pulmonary oedema.

The second mechanism suggests that when there is respiration against an obstructed upper airway, there is generation of mechanical stress which disrupts the alveolar epithelium and pulmonary microvascular membranes, leading to an increase in capillary permeability and alveolar fluid.

There were similar characteristic in both case 1 and case 2. These were young, otherwise healthy patients with no previous chest or cardiac pathology or any other comorbidity. The NPPO also occurred after extubation. Though case 2 had a laryngospasm, the antecedent for case 1 was not detected. NPPO is more common in the young due to their ability to generate a high inspiratory effort, have less cardiac abnormality and also has a high sympathetic drive [7].

Differential diagnosis of pulmonary oedema (PO) detected after extubation could be cardiogenic or non-cardiogenic. The cardiogenic cause could be a new-onset left ventricular dysfunction caused by severe arrhythmia or an ischaemic

episode. The non-cardiogenic causes include NPPO, fluid overload, aspiration pneumonitis and anaphylaxis.

There was no echocardiography service in the hospital to investigate the PO as a cardiogenic cause. However, the absence of a pre-existing co-morbidity and any significant perioperative arrhythmia in these young patients made it unlikely. Also the total volume of Ringer's lactate given during both the intraoperative and postoperative period could not cause fluid overload to precipitate a PO. The triggers of perioperative anaphylaxis PO include latex, antibiotics, benzylisoquinolone muscle relaxants, and some opioids and intravenous induction agents. However, anaphylaxis is also associated with features like early urticaria rash and anaphylaxis significant enough to cause PO would have been associated with a haemodynamic collapse and none of these occurred in the patient. Though aspiration pneumonitis can produce a picture close to that NPPO, the rapid onset and resolution is not a feature of aspiration pneumonitis [9].

Various investigations have been performed in the differential diagnosis of postextubation PO. These include analysis of the oedema fluid, bronchoscopy, chest x-ray and echocardiography. In NPPO, the findings have always been a high protein content in the oedema fluid, enforcing the concept of disruption of capillary integrity rather than simple rearrangement of Starling forces [4]. Cardiac filling pressures too have not been high, suggesting non-cardiogenic origin. The common investigative tool available in most hospitals in developing countries would be a chest x-ray. Chest x-ray has consistently shown an early diffuse opacification in the lung fields which rapidly resolve within 48 hrs after treatment.

A variety of treatment modalities have been used in the management of post-extubation NPPO but the mainstay has been the use of invasive and non-invasive positive airway pressure ventilatory support. This has been in form of CPAP, BiPAP or PEEP. Most hospitals in developing countries, just like the hospital where these two cases occurred, do not have intensive care units/high dependency units and also ventilators capable of delivering these modes of support in their recovery wards. In both cases, simple face mask could not treat the NPPO. The use of non-rebreather mask was able to manage both patients within similar times as those treated in well-resourced centres with means of positive pressure ventilation. Diuretics are often administered, though its use has been questioned by others [4] [8] [10]. In the absence of haemodynamic instability, the patients were safely given diuretics.

Other additional therapies that have been used in managing NPPO include β -agonists which may increase the rate of alveolar fluid clearance via increased active cation transport. Prone ventilation and extracorporeal exchange membrane oxygenation has also reportedly been used [11].

5. Conclusion

In conclusion, NPPO may be uncommon, but can be encountered. The preoperative characteristics of patients, type of surgery, time course of onset and resolu-

tion of features are adequate to help in differential diagnosis of post-extubation PO even in low-resourced centres. It is an important cause of morbidity, unplanned intensive care admission and occasionally mortality in young and otherwise healthy patients. Prevention and early relief of upper airway obstruction should decrease the incidence. In low-resourced centres without means of positive pressure respiratory support, early recognition and the use of non-rebreather mask can be used and the outcomes are comparable. Therefore all hospitals must be encouraged to have non-rebrather mask as part of their oxygen delivery devices.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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