Negative Pressure Pulmonary Edema: Case Report

S. M. Abbas, M. Q. Hoda
Department of Anaesthesiology, Aga Khan University Hospital, Karachi.

Introduction

Negative pressure pulmonary edema (NPPE) is a well-recognized phenomenon among the anesthesiologists. The concept of NPPE is not new. Moore[1] in 1927 hypothesized this. Before mid 80's very few cases were reported, however during the last 15 years increasing number of cases have been reported in literature.[2] Although causes of upper airway obstruction that lead to NPPE have been varied but postoperative laryngospasm is on the top of the list.[3] Other recognized causes include tumor, strangulation, hanging[4], bilateral vocal cords paralysis[5], foreign body aspiration[6], pseudomembrane formation in pharynx[7], saber sheath trachea[8] and severe episode of obstructive sleep apnea.[9]

We report the case of a 36 year old otherwise healthy female who developed acute upper airway obstruction as she had clenched her endotracheal tube towards the end of the procedure. Vigorous inspiratory efforts against a completely obstructed airway which was maintained for sometime (the modified Mueller maneuver)[10], led to the development of acute negative pressure pulmonary edema (NPPE).

Case Report

A 36 year old Gravida 5, para 3+1 presented with nine weeks history of amenorrhea with per vaginal bleeding and lower abdominal pain. She was 137.5 cm tall and weighed 88 kg. On examination her blood pressure was 130/90 mmHg, pulse 80/min and temperature 37ºC. Vaginal examination revealed a closed internal os and a bulky uterus. Uterine ultrasonography showed a very small intrauterine gestational sac and right ovarian cyst of 52x44mm. She was planned for laparoscopy and dilatation and evacuation under general anaesthesia.

She was classified as ASA IIE as she was slightly over weight. Twelve lead ECG was not performed since she was young and there was no history of cardiac illness on history/physical examination.

She was anaesthetized by using rapid sequence induction technique with thiopentone sodium (5 mg/kg) and suxamethonium (1.5mg/kg) and her trachea was intubated with PVC endotracheal tube (ETT) size 7.5 mm ID without difficulty. Analgesia was provided with 80 mg of pethidine and atracurium 25 mg was used for muscle relaxation.

The procedure lasted for approximately one hour. At the end of the procedure the inhalational anaesthetic was turned off and neostigmine 2.5 mg and atropine 1mg were administered to reverse the effect of neuromuscular blockade. Just before extubation she clenched her teeth on the ETT which resulted in complete airway obstruction. Her oxygen saturation on pulse oximetery decreased down to 54%. A Guedal oropharyngeal airway was introduced with some difficulty which relieved the obstruction. As the obstruction was relieved, copious amount of pink frothy fluid started flowing out of the ETT and oxygen saturation on pulse oximeter rose to 92% on 100% O_2. Chest auscultation revealed bilateral coarse crepitations.

At this stage working diagnoses included fluid over load, acute myocardial infarction or NPPE. Fluid overload was excluded as she was given only 500 ml of Ringers lactate solution and acute myocardial infarction was excluded on the basis of a normal 12 lead ECG. This left us with a strong possibility of NPPE.

Figure 1. Chest X-ray showing pulmonary edema and normal size heart.

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A decision was taken to electively ventilate this patient. In the mean time an arterial line and CVP line was inserted which showed a CVP of +4 cm of H_2O. Chest Xray revealed pulmonary oedema with normal sized heart (Figure 1). After urinary catheterization, injection frusemide 80mg was given. She was then shifted to the recovery room where she remained on pressure support ventilation for the next 4 hours.

During her stay in the recovery room, she remained hemodynamically stable with 700 milliliters urine output in 4 hours. Her oxygen saturation (SpO_2) improved gradually which allowed us to reduce FiO_2 to 0.4 with a SpO_2 of 98% while breathing spontaneously. After a repeat chest X-ray, which also confirmed radiological improvement and clearance of pulmonary edema (Figure 2). She was then extubated and sent to the special care unit where she remained stable for 24 hours and was then discharged home. Postoperative follow up did not show any residual effect .

Figure 2. Chest X-ray after treatment showing clearance of lung fields.

Discussion

Although many cases of NPPE have been reported but in most of them pulmonary oedema was associated with serosanginous fluid rather than alveolar hemorrhage. Frank alveolar hemorrhage was found only in three cases reported by Schwartz[11], Dolinski[12] and Devys.[13]

Biting the laryngeal mask as a cause of NPPE has been reported by Devey[13] while clenching on the endotracheal tube, to our knowledge, has been reported by Dispinigaitis[14] for the very first time and then by Liu.[15]

The pathogenesis of NPPE is multi factorial and includes changes in starling forces, hemodynamic changes secondary to markedly increased negative intrathoracic pressure[7], alveolar hypoxia, increased catecholamines levels diverting the systemic blood to pulmonary system and failure of lymphatics.[10]

During normal tidal inspiration intrapleural pressure becomes negative from normal -2cm of H_2O to -10cm of H_2O. But during strong inspiratory effort this reduction can be even down to - 50 to -100cm of H_2O.[16] This negative intrapleural pressure results in drawing of fresh air from the atmosphere and increase in venous return.

During expiration elastic recoil of the lung creates a positive intrapulmonary pressure which expels the air out of the lung. The intrathoracic dynamics during the inspiratory and expiratory effort with an obstructed airway is basically an exaggerated response of normal phenomenon. Due to the complete airway obstruction all the driving forces in the thorax cancel each other till the obstruction is relieved.[10] The sudden fall in intrathoracic pressure not only drives the air into the lungs from the atmosphere but also causes sudden increase in venous return, reduction in right atrial pressure, with a concomitant increase in pulmonary arterial and pulmonary capillary hydrostatic pressure, complicated by a reduction in perivascular interstitial hydrostatic pressure.[17,18] This causes an increase in transcapillary pressure gradient, favoring the transudation of fluid into the interstitial spaces. In addition increased central venous pressure impedes passive lymphatic blood flow.[18,19] Pulmonary blood volume is augmented by increase in systemic pressure secondary to the release of norepinephrine resulted from hypoxia, hypercapnia and anxiety.[18,20] The increase in right ventricular volume lets the intraventricular septum shift towards the left causing reduction in left ventricular diastolic compliance.

The catecholamines induced increase in afterload and negative intrathoracic pressure mediated increase in preload, severely affect the left ventricular function contributing to the formation of pulmonary edema.

Furthermore negative pulmonary pressure increases the mechanical stress on pulmonary capillaries. This results in mechanical disruption of the alveolar-capillary membrane with subsequent impairment of barrier function, a process termed "stress failure."[11,21]

Recommended management of NPPE in almost every reported case is the same and includes use of Continuous Positive Airway Pressure (CPAP)[8] or Positive End Expiratory Pressure (PEEP)[11] in patients[3] requiring mechanical ventilation[16] as we did with our patient. Use of

diuretic[8-10,16], is also a common therapeutic measure in every case, while use of hydrocortisone has been recommended in cases where laryngeal edema was suspected.

This case report emphasizes on the importance of using bite block/oropharyngeal airway at the end of surgery in order to avoid NPPE as this is the crucial time which can lead to tube biting during emergence from anaesthesia.

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