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Case Report

A Healthy 36-Year-Old Woman with Hypoxia, Hemoptysis and Pneumomediastinum Immediate Post Extubation after Lower Segment Cesarean Section

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Received: August 27, 2020; **Published:** October 31, 2020

Abstract

A 36-year-old female (Mallampati class I), with no previous medical history, was transferred to the Surgical Intensive Care Unit following uneventful lower segment Cesarean Section (which lasted three hours for placenta accrete). The operative course was notable for minimal blood loss. She was intubated and mechanically ventilated and the plane was to extubate in the Surgical Intensive Care Unit (SICU).

In the SICU, the patient had return of spontaneous breathing but was severely agitated with aggressive cough and strong inspiratory efforts. In addition, she was continuously biting the endotracheal tube. As she was responding to verbal commands and maintaining good oxygen saturation, she was planned for extubation. No medications were given then. Immediate post extubation, the patient developed sub sternal chest pain, tachypnea and tachycardia requiring 100% oxygen to maintain oxygen saturation above 92%. Her vital signs were otherwise normal. Bilateral rhonchi were audible during auscultation with a frothy serosanguinous fluid being suctioned from the oropharynx. Within 2 min, this fluid became progressively bloodier. The patient soon coughed up around 200 ml of bright-red blood followed by another one in 5 minutes. Anteroposterior chest radiograph was performed showed perihilar interstitial and alveolar opacification consistent with pulmonary edema/hemorrhage. Air bronchograms and peribronchial cuffing were noted too. A repeat hemoglobin showed a significant drop from preoperative level of 13 g.dl to 10 g.dl−1 postoperatively. High-resolution computed tomography (CT) sections displayed a striking preferential central and nondependent distribution of ground-glass attenuation (edema/hemorrhage) consistent with negative pressure pulmonary edema/hemorrhage.

As the patient was maintaining her airway as well as her blood pressure, noninvasive positive pressure ventilation with use of high supplemental O2 as well as diuretic were used. No bronchoscopy was performed, as the patient was not intubated and was clinically improving over the course of the first few hours with complete resolution of chest X-ray. The patient was discharged from the SICU in 48 hours. The rapid onset of symptoms, transient hemoptysis and rapid and complete resolution were all consistent with a diagnosis of negative pressure pulmonary hemorrhage.

Keywords: *Postoperative; Extubation; Negative Pressure; Pulmonary Edema; Pulmonary Hemorrhage*

Abbreviations

SICU: Surgical Intensive Care Unit; ICU: Intensive Care Units; NPPE: Negative-Pressure Pulmonary Edema; DAH: Diffuse Alveolar Hemorrhage; CT: Computed Tomography; FiO_2: Fractional Inspired Oxygen Tension; CPAP: Continuous Positive Airway Pressure; ARDS: Adult Respiratory Distress Syndrome; AIMS: Australian Incident Monitoring Study; NIV: Non Invasive Ventilator; NPIP: Negative Pleural Inspiratory Pressure

Introduction

Diffuse alveolar hemorrhage (DAH) is an accumulation of blood in the alveoli originating from damaged alveolar microvasculature**.** Negative-pressure pulmonary edema (NPPE) secondary to upper airway obstruction - a potentially life-threatening emergency- often

encountered in the acute perioperative and critical care settings- proceeds DAH in the postoperative settings. Both NPPE as well as NPPE-related DAH are relatively rare but well-described life-threatening complication which was first reported many years ago [1]. Incidence is 0.1% of general anesthesia requiring tracheal intubation, most commonly caused by laryngospasm [2]. Strong respiratory efforts is the leading cause for generation of high negative pressure which is more prominent among young, athletic men than women with good thoracic musculature that allows them to maintain an extremely strong inspiration during a long period of apnea against an obstructed airway. Unlike the predominant occurrence of this entity in male patient, we discuss here a case of a female patient with no other risk factors presenting with NPPE related DAH associated with an impressive hemoptysis and acute respiratory failure.

Case Report

An otherwise healthy 36-yr-old woman underwent elective lower segment Cesarean Section for placenta accrete. She had previously undergone general anesthesia without complications and had no family history of difficulties with anesthesia. The patient's height was 165 cm, she weighed 75 kg. Her physical examination revealed no abnormalities. After premedication, anesthesia was induced and maintained. The surgery lasted three hours and was uneventful. The plane was to extubate in the Surgical Intensive Care Unit (SICU) because of a busy OR, therefore she was shifted intubated and mechanically ventilated to the SICU.

In the SICU, the patient initiated spontaneous ventilation but was severely agitated with aggressive cough and strong inspiratory efforts resulting in chest wall retraction. In addition, she was continuously biting the endotracheal tube. As she was responding to verbal commands, maintaining good oxygen saturation, and had reached for the endotracheal tube she was extubated. No medications were given then. She was then noted to have no apparent air movement for approximately 35 seconds. Immediately and post extubation, the patient developed sub sternal chest pain, tachypnea and tachycardia Oxygen saturation measured by pulse oximetry (SpO_2), decreased to approximately 80%. 100% oxygen were needed to increase SpO_2 to 92%. Her vital signs were otherwise normal. Bilateral rhonchi were audible during auscultation with a frothy serosanguinous fluid being suctioned from her oropharynx. Within 2 min, this fluid became progressively bloodier. The patient soon coughed up around 200 ml of bright-red blood followed by another 200 ml blood in 5 minutes. Upon further suctioning neither gastric contents nor more blood was present in the oropharynx. Continuous positive airway pressure (CPAP) with 100% oxygen was then applied using a face mask. An anteroposterior chest radiograph was performed which showed perihilar interstitial and alveolar opacification consistent with pulmonary edema and possibly alveolar hemorrhage. Air bronchograms and peribronchial cuffing were noted too. A small pneumomediastinum was also seen (Figure 1).

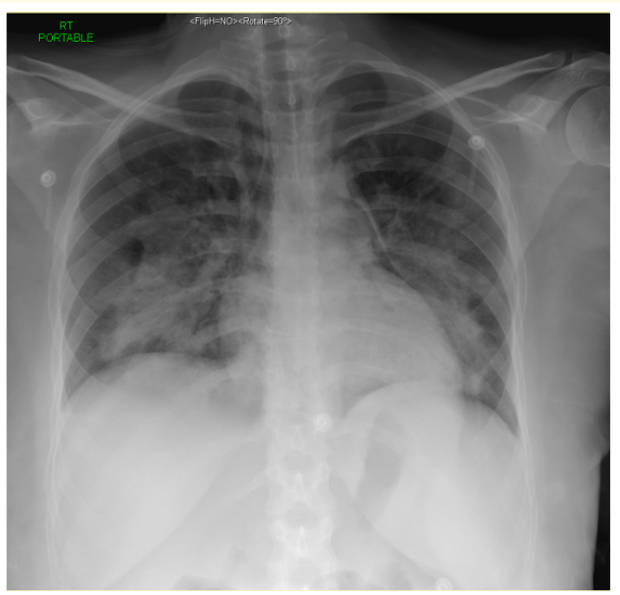

Figure 1

The first arterial blood gas measurement obtained within 5 minutes of the initial event showed a pH of 7.34, an arterial carbon dioxide tension ($PaCO_2$) of 32 mmHg and an arterial oxygen tension (PaO_2) of 70 mmHg, with a fractional inspired oxygen tension (FiO_2) of 1.0.

Over the course in the SICU, repeat hemoglobin showed a significant drop from preoperative level of 13 g.dl to 10 $g.dl^{-1}$ postoperatively. Transthoracic echocardiography showed preserved ejection fraction with normal chamber size. High-resolution computed tomography (CT) sections displayed a striking preferential central and nondependent distribution of ground-glass attenuation (edema/hemorrhage) consistent with negative pressure pulmonary edema and hemorrhage (Figure 2). Serologic workup for systemic disorders including vasculitis and connective tissue diseases was found later to be negative.

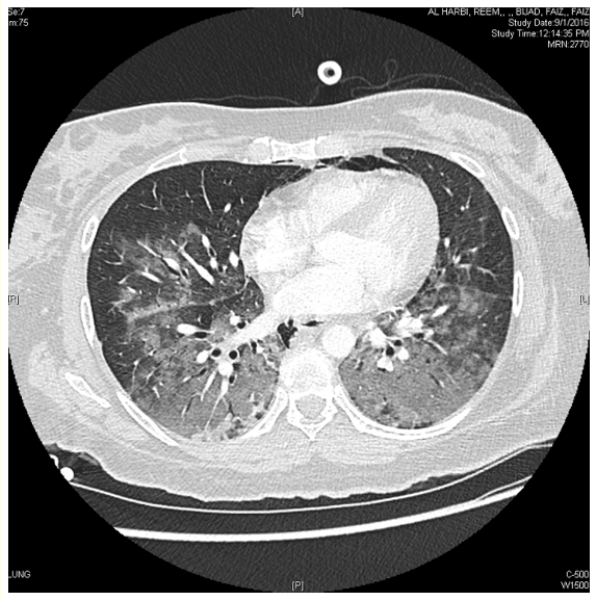

Figure 2

During the next few hours, respiratory condition stabilized, necessitating weaning oxygen requirements gradually. Blood pressure remained stable all through. On the second ICU day, the patient's condition began to show marked improvement. Oxygenation improved dramatically, and FiO_2 was 0.3. Bronchoscopy was thought to be unnecessary because of the patient's obvious clinical improvement and was not intubated. Forty eight hours from the initial event, the patient was transfers to the ward and thereafter was discharged from the hospital after 2 days, with normal results of chest X-ray. Follow-up at six months showed no functional deficit.

Discussion

Negative-pressure pulmonary edema (NPPE)-related diffuse alveolar hemorrhage (DAH) is an underdiagnosed clinical entity seen with alveolar capillary damage. The pathophysiology of NPPE is generation of a negative pleural pressure against an upper airway obstruction.

NPPE itself is a rare but well-described complication of upper airway obstruction and has been of particular interest to clinicians [3]. It is a dangerous clinical complication during the recovery period after general anesthesia. It occurs more frequently than reported in studies, which should draw the attention for anesthesiologists and Intensivist.

NPPE usually progresses rapidly. The time from the onset of airway obstruction to the development of pulmonary edema could range from few minutes to within first few hour after surgery without specific time window [4]. Although negative intrapleural pressure is the main component of NPPE pathogenesis, other factors also play an important role. Other risk factors for NPPE include obesity, short thick neck, Mallampati score III, history of obstructive sleep apnea and non-utilization of laryngotracheal topical anesthetics during intubation and intravenous administration of lidocaine before extubation. Obviously, the overt straining, strong inspiratory efforts and endotracheal tube biting were the causative factors in the case we had.

In the perioperative period, NPPE could be life-threatening complication without timely diagnosed and treated. Therefore, it is essential to notice the potential causes, make a rapid differential diagnosis, and determine the effective treatment before disease aggravation occurs. In this case, pulmonary edema associated with aspiration pneumonia was first excluded. The patient underwent strict fasting with no history of aspiration. She was otherwise healthy young woman with no history of cardiovascular disease, with Echocardiography showing no abnormalities, thus cardiogenic pulmonary edema was also ruled out. In addition, there was no drug-associated allergy during the entire perioperative period. Considering the above factors as well as patient undergoing acute airway obstruction after extubation followed subsequently by flash pulmonary edema and rapid recovery thereafter after use of assisted ventilation via CPAP, the diagnosis of NPPE and NPPE related DAH was made.

NPPE has was estimated to have incidence of 0.1% in all anesthetic practices. More recently, an Australian Incident Monitoring Study (AIMS) reported an incidence of NPPE in up to 3% of all incidence reports of postextubation laryngospasm [5]. Literature review reveals mortality ranging from 11% up to 40%, more in elderly patients with respiratory dysfunction and cardiac disease. Failure of early recognition may lead to progression to unnecessary and potentially deleterious iatrogenic complication of NPPE 'adult respiratory distress syndrome (ARDS) through damage of the pulmonary capillaries by mechanical disruption of the alveolar-capillary membrane, contributing to increase risk of mortality.

The normal negative pleural inspiratory pressure (NPIP) ranges from -2.5 to -10 cm H2O, whereas the NPIP during acute airway obstruction in adults may reach -50 to -80 cm H_2O (10 times or more that of normal breathing). In adults, an inspiratory effort against a closed upper airway (Muller maneuver) can generate up to negative 140 mm H_2O pressure with vigorous inspiratory efforts [6]. After laryngospasm, the patient continues inspiring against a closed glottis, generating a marked negative intrathoracic pressure leading to an increase in venous return to the right heart and pulmonary arteries. This volume expansion causes high capillary and arteriolar pressure, which favors the accumulation of transudate in interstitial and alveolar spaces. With the increase in hydrostatic pressure, erythrocytes might diffuse through alveolo-capillary membrane, resulting in pink frothy sputum, while blood (hemoptysis) could occur when higher NPIP is generated resulting from rupture of alveolocapillary membrane and diffuse alveolar hemorrhage (DAH) [7]. Hemoptysis is caused by generating extremely high negative inspiratory pressure reaching more than -150 cm/H_2O [8]. This degree of high negative pressure affects the central airways much more than the distal alveolar-capillary system (as in NPPE) elevating the bronchial vascular pressure resulting in rupture and hemorrhage. In addition, the increased stress in the pulmonary capillary wall may cause mechanical rupture of the alveolar-capillary membrane with subsequent damage to the barrier function, in a process called "stress failure" with a consequent development of free air in the pleural space (pneumomediastinum) as seen in our patient).

NPPE may be more common in intensive care units (ICU) patients than is thought [9]. Reported causes of NPPE among adults in the ICU have included inspissated tracheal secretions, hiccups, difficult intubation, oropharyngeal surgery, biting the endotracheal tube and severe patient-ventilator asynchrony. A clench of the endotracheal tube lasting 20 seconds to 1 - 2 minute appears sufficient to produce NPPE.

NPPE treatment is supportive, aimed at restoration and maintenance of a patent airway, use of appropriate positive pressure breathing and supplemental O_2. Administration of diuretics may sometime be used. If these measures are inadequate, mechanical ventilation with

positive end-expiratory pressure may be required. Although intubation and conventional mechanical ventilation remains the mainstay of treatment for severe hypoxemia and respiratory distress, noninvasive positive pressure ventilation (NIPPV) has a great role. The good pre-existing health condition of many patients who develop NPPE/NPI explains the rapid resolution of this syndrome by supportive treatment in most of the cases.

Prevention and early relief of upper airway obstruction should decrease the incidence. In patients at risk of developing NPPE/NPI, preventive measures (mostly practiced in Perioperative period) should be considered. These include clearing the airway of the retained blood or secretions, using lidocaine topically applied on the larynx, instilled through the endotracheal tube prior to extubation or administered intravenously, preoxygenating with 100% oxygen and administering steroids for upper airway obstruction post extubation. Although it is not common to use steroids, it has been used in patients who are at risk. When a patient is biting on the endotracheal tube, muscle relaxants can be used to stop laryngospasm. Delaying extubation until full wakefulness decreases risk of laryngospasm. None of these measures were taken in our case.

Conclusion

In conclusion, we presented a case of a healthy young female who developed postoperative hemoptysis, diffuse ground-glass opacity and infiltrates on computed tomography (CT) of the chest, anemia, and hypoxic respiratory failure, based on which a diagnosis of DAH was made. NPPE was the most likely triggering mechanism. Though hemoptysis is mostly self-limiting, it may be massive, warranting aggressive treatment. Although NPPE and NPPE- related DAH is a life-threatening and well described postanesthesia complication, it remains an underdiagnosed event. Increased vigilance among the providers is essential to prevent the morbidity associated with this condition.

Source of Funding

None.

Conflicts of Interest

None.

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Volume 6 Issue 11 November 2020