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CASE REPORT

NEGATIVE PRESSURE PULMONARY EDEMA AFTER GENERAL ANESTHESİA

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ABSTRACT

Negative pressure pulmonary edema (NPPE) due to upper airway obstruction has been reported in several clinical situations. The increase in negative intrathoracic pressure is the main cause of NPPE. We present a case of NPPE that occured after general anesthesia for migraine surgery. We wanted to share our experiment and to review the literature.

INTRODUCTION

Negative pressure pulmonary edema (NPPE) following upper airway obstruction has been reported in several clinical situations including postanesthetic state (1-3)

Laryngospasm is the main cause of NPPE. NPPE has a complicated pathogenesis. Inspiratory effort to overcome airway obstruction can be result in negative intrathoracic pressure. It causes decreased pulmonary capillary perivascular pressure favoring hydrostatic transudation of fluid into the interstitiel tissue. Normal pleural inspiratory pressure is between -2 to -5 cm H2O. This pressure level may drop to -50 cm H2O and negative pressures as of -100 cmH2O, during airway obstruction.

Treatment includes: variable regimens of diuretics, digoxin, corticosteroids, morphine, and fluid restriction (4).

Case Report

A 41-year old woman underwent an elective migraine surgery under general anaesthesia. She was evaluated in the class 1 ASA (American Soceity of Anesthesiologists) before surgery. She was 170 cm tall and weighted 74 kg. There was no abnormality in the laboratory and physical examination before the surgery. The chest X-ray was normal (Figure 1).

Her Mallampati grade was assessed as class 1. General anesthesia was induced with propofol (200 mg) and fentanyl (100 microgram). The endotracheal intubation was faciliated easily with a 7,5 mm cuffed tube after iv rocuronium bromide (40 mg) administration in three minutes. Anaesthesia was maintained with sevoflurane (1-2 %) and 4 L/min oxygen /nitrous oxide (40/60 %). 1000 cc 0.9 % NaCl was given to the patient during the surgical procedure.

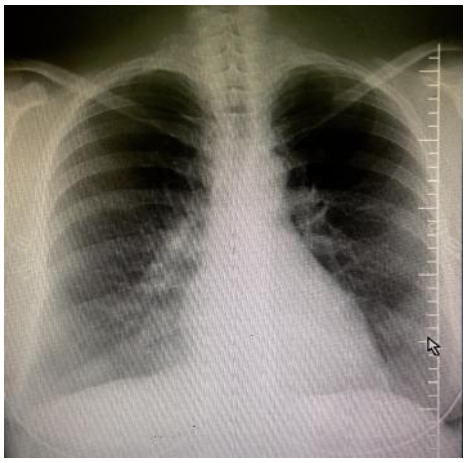

Figure 1 Chest X-ray showing a normal preoperative examination of the patient

The planned surgical procedure was completed in 1 hour, 30 minutes. After the anaesthetic gases were discontinued spontaneous breathing started and the exubation was performed after iv 3 mg neostigmine and 1 mg atropine administration. Following the extubation (1-2 minutes later) the patient developed hypoxemia. Sp02 was 30%. The hypoxemia was determined as laryngospasm and to overcome the hypodynamic response, 1,5 mg/kg 2 % lidocaine and 100 % oxygen were administrated. 80 mg iv prednisolone was added against the traumatic effect of the cuff. About 3-4 minutes later, the respiratory distress dissappeared and the oxygen saturation got better. After the recovery, the patient was transported to the postanesthesia care unit to follow up closely. Then patient developed hypoxemia. The oxygen saturation was 88 %. At this time arterial blood gas analysis performed. The results were: ph: 7.39, PaO2: 63 mm Hg and Pa CO2: 36 mm Hg. Bilateral end-inspiratory crepitations were heard anteriorly and posteriorly, especially in the basal segments of the lungs. The chest X-ray showed diffuse

bilateral infiltrates (Figure 2).

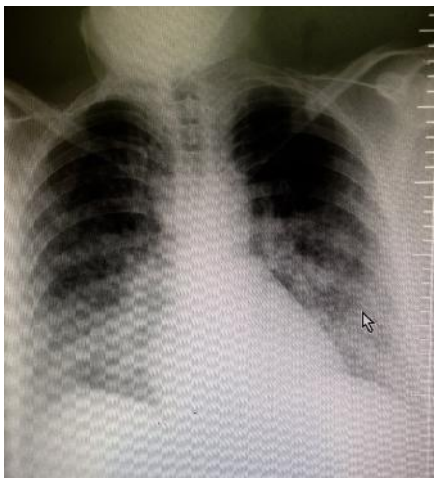

Figure 2 Chest X-ray showing bilateral inferior infiltrates, suggestive of pulmonary edema

With this findings the patient was transferred to the medical intensive care unit. (ICU) for further management. Serial electriocardiograms were taken and all were normal. Echocardiogram showed normal ejection fraction with normal valves and no wall motion abnormality. The patient was diagnosed with post-extubation negative pressure pulmonary edema and was treated with intravenous furosemide (20 mg 2x1), iv ranitidine (50 mg 2x1), oxygen theraphy and ipratropium bromide (20 mcg) and salbutamol (100 mcg 4x1) and budesonide 0,5 mg in nebule. The oxygen saturation became 95-96 % in the monitore. Arterial blood gas analysis was performed again and the results were: ph:7,40, Pa02 67 mm Hg and PaCO2 43 mm Hg. On the second day in ICU, the patient was better with normal vital signs. The control chest X-ray showed marked resolution of the pulmonary infiltrates (Figure 3). And the patient was transferred back to the to be followed up. The patient was followed up for 24 hours there and there was no abnormality and discharged

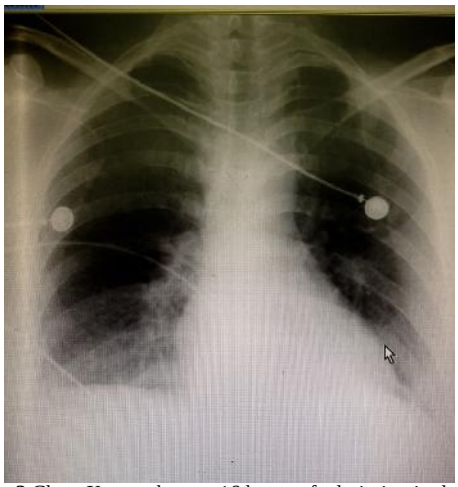

Figure 3 Chest X-ray taken on 16 hours of admission in the ICU showing marked resolution of the pulmonary edema

DISCUSSION

Negative pressure pulmonary edema (NPPE) is a clinical entity of great relevance in anesthesiology and intensive care. NPPE can be immediate or delayed. The incidence of NPPE has been reported to be 0,05 %-0,1 % of all anesthetic practices (5).

The NPPE pathophysiology is defined as four major mechanisms: disturbances of pulmonary fluid homeostasis can be induced by four pathways that can lead to increase interstitial fluid, increased hydrostatic pressure in the pulmonary capillary bed, decreased osmotic pressure of plasma, increased permeability of the membrane, and decreased return of fluid to the circulation via lymphatics. The high negative intrathoracic pressures cause a dramatic and abrupt increase in systemic venous return to the heart with a simultaneous drop in cardiac output associated with the reduced pulmonary venous drainage to the left atrium. Pulmonary capillary pressures increase while intra alveolar pressures drop, and alveolar cell junctions are disrupted. Fluid moves rapidly into interstitial and alveolar spaces, and the pulmonary edema remains even after the airway obstruction is relieved. NPPE usually develops immediately after extubation, rarely it may develop a few hours later in the postoperative period. This delayed manifestation is the result of a positive pressure, created by forceful expiration against a closed glottis, opposing fluid transudation. When airway obstruction relieves, increased venous return causes blood shift from peripheral to central circulation and hydrostatic transudation (5).

The patient, in our case report, has no risk factors for NPPE. In clinical presentation; symptoms include; decreased oxygen saturation, stridor, suprasternal and supraclavicular retractions, urgent use of accessory muscles of inspiration, panic in the facial expression. Pulmonary edema causes both impaired diffusion of oxygen and ventilation/ perfusion mismatching, leading to sudden and possibly severe hypoxemia. In the chest radiograph a diffuse interstitial and alveolar infiltrates is typical (5).

Diagnosis of NPPE bases on a detailed history and symptoms. Agitation, tachypnea, tachycardia, frothy pink pulmonary secretions, rales, and proggressive oxygen desaturation suggest the diagnosis of NPPE. Relief of the airway obstruction and correction of hypoxemia are the first steps of the treatment. The next step is to address the pulmonary edema with a diuretic unless the patient is hypovolemic. If the airway obstruction is due to the patient biting down on the endotracheal tube, a dose of succinylcholine (0.1-0.2 mg/kg) may be needed to relax the jaw muscles, although controversial use of steroids in NPPE has been reported. Continuous positive airway pressure is required in 9% - 18% of all cases and 34 % -46 % of the patients require controlled mechanical ventilation via orotracheal intubation (6,7).

Most patients who develop NPPE do well with only oxygen and furosemide, as in our case, ICU staying and monitoring may be required in a few cases. Some patients may also require reintubation. Short acting muscle relaxants such as succinylcholine can be an alternative to overcome laryngospasm, ıf the normal ventilation is ineffective (6). Intraoperative muscle relaxants, topical or spray lidocaine, and steroids should decrease the NPPE incidence. Steroids may be useful for patients who are at risk (8).

CONCLUSIONS

With prompt diagnosis and intervention most patients can be

treated without incident. The patients who experience NPPE should be monitored for longer than usual postoperative period.

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