Case Report

Postoperative Acute Pulmonary Edema: An Obstructive Sleep Apnea Syndrome

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Abstract

Pulmonary edema can occur in the immediate postoperative period, and this may reflect intraoperative cardiac events resulting in cardiogenic pulmonary edema. The predominant mechanism is increased negative intrathoracic pressure, although hypoxia and cardiac and neurologic factors may contribute. We report a case of a patient with postextubation pulmonary edema who had no obvious risk factors for the development of this syndrome. This syndrome can occur in any patient undergoing general anesthesia and that this syndrome may develop following lapses in anesthetic technique, especially extubation.

Keywords: Pulmonary oedema; Obstructive sleep apnea; Blood pressure; Anesthesia

Introduction

Pulmonary edema can occur in the immediate postoperative period, and this may reflect intraoperative cardiac events resulting in cardiogenic pulmonary edema. The predominant mechanism is increased negative intrathoracic pressure, although hypoxia and cardiac and neurologic factors may contribute. They usually require immediate attention in the postanesthesia recovery room. In most series these patients have risk factors for this syndrome. However, not all patients have identified risks, and the pathogenesis in these patients remains unclear. We report a case of a patient with postextubation pulmonary edema who had no obvious risk factors for the development of this syndrome.

Case Presentation

This is a 31-year-old patient, weight 65 kg, height 1.70 m, with a BMI of 22.49 kg/m^2; history of surgery for fracture of the nasal bones 15 years ago, admitted to the operating room for management of a herniated disc L5S1. Patient seen in pre-anaesthetic consultation and Classified ASA I.

The preoperative blood test are NFS, ionogram, blood sugar, creatine urea returned normal. The electrocardiographic tracing showed a normal sinus rhythm.

Standard general anesthesia was proposed, with standard routine monitoring (electrocardiogram, noninvasive blood pressure, pulse oximetry and capnography). On the day of the operation, the patient was admitted to the operating room. Induction of anesthesia was achieved by titrated doses of propofol, and curarization was achieved using 50 mg of rocuronium. Fentanyl 200 microgram intravenously was administered for analgesia.

Maintenance of anesthesia was performed using an oxygen/air mixture and sevoflurane. The volume-controlled ventilation mode was used intraoperatively, and a carbon dioxide target of 35 mm Hg-38 mm Hg was maintained. After anesthesia and intubation of the patient and the setting of artificial respiration, the patient was placed in the genupectoral position with a cushion under the thorax, abdomen free. Vital hemodynamic parameters remained stable and surgery could begin. At the end of the operation the patient was returned to the supine position. Once fully awake and conscious, he was extubated. Furthermore, during the patient's stay in the post-intervention monitoring room, shortly after extubation, he developed respiratory distress with increased oxygen requirements to maintain Spo2 above 95%. The patient was lethargic, BP=90/40 mmHg FC=110 bpm, polypneic at 34 cycles/min with bilateral crackles on auscultation and pink foam from the mouth, the diagnosis of acute pulmonary edema was placed, hence his emergency reintubation and administration of boluses of furosemide. The chest X-ray showed bilateral alveolar infiltrates. While a transthoracic ultrasound was performed showing apical hypertrophic cardiomyopathy with preserved left ventricular ejection fraction. Patient subsequently admitted to intensive care, PAO episode controlled with furosemide and ventilatory treatment. patient extubated after 6 hours

Finally, the patient was transferred to the service for a continuation of his care, the patient did not keep any neurological deficit.A transthoracic ultrasound was redone the next day, showing a slightly hypertrophied left ventricle realizing the appearance of an athlete's heart, fraction of ejection of left ventricle preserved without other abnormalities. A cardiac MRI was performed and returned without abnormality. Moreover, given the absence of causes explaining the occurrence of PAO, a polysomnography was requested which noted the presence of a ranchopathy associated with a syndrome of severe obstructive sleep apnea (hypopnea apnea index50, 42/hour), non-positional and responsible for significant nocturnal desaturations greater

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than 4%: 43.41/hour).Thus, the analysis of the heart rate and the RR spaces is without abnormality. The diagnosis of obstructive sleep apnea syndrome was made and the patient was fitted with a continuous positive pressure device.

Results and Discussion

Sleep Disordered Breathing (SDB) is a continuum from habitual snoring through upper airway resistance syndrome to Obstructive Sleep Apnoea (OSA). Obstructive Sleep Apnea (OSA) is characterized by recurrent episodes of partial and complete airwayobstructions during sleep with repetitive apneas and hypopneas as a result [1]. The disease severity is measured using the apnea-hypopnea index (AHI), i.e., the mean number of apneas and hypopneas per hour of sleep. OSA is defined when the AHI is ≥5 and OSA syndrome when AHI ≥5 is accompanied with daytime sleepiness Obstructive events impair the balance in the Starling forces that govern transcapillary fluid transudation, while simultaneously impeding the lymphatic drainage of the lung fluids, which leads to extracellular and alveolar edema [2].

Assuming that oncotic pressure and capillary membrane conductance remain unchanged, the main determinant of transmembrane fluid transudation is transcapillary hydrostatic pressure. During obstructive apnea, intravascular hydrostatic pressure should increase [3,4].

In the presence of OSA, substantial negative intrathoracic pressure is generated against the closed upper airway. The increase in negative pressure increases transmural pressure in all intrathoracic structures, including the pulmonary interstitial pressure of the perivascular space. Consequently, perivascular hydrostatic pressure decreases, further favouring excess fluid filtration [5,6]. Prone position can also increase the negative intrathoracic pressure. The absence of left ventricular dysfunction and the sequence of the described symptoms make the diagnosis of acute Negative Pulmonary Pressure Edema likely (NPPE).

It is a relatively uncommon intra-operative and post-operative complication and is still a diagnosis by exclusion of other factors. Generally, NPPE occurs when inspiratory effort against an obstruction creates a large intrapulmonary negative pressure which can increase venous return with subsequent increase in the pulmonary capillary hydrostatic pressure. The transudation of fluid from the pulmonary vasculature causes edema formation into the alveolar space [7,8].

We also emphasize that OSA-related hypoxia contributes to increased pulmonary microvascular hydrostatic pressure resulting from the constriction of pulmonary postcapillary venules. These events further alter Starling forces, favouring fluid filtration into the lung [9].

Pulmonary edema can occur in the immediate postoperative period, and this may reflect intraoperative cardiac events resulting in cardiogenic pulmonary edema. It can also develop after other intraoperative and postoperative events, including aspiration and laryngospasm.

Laryngospasm also occurs in patients who are extubated in Stage 2 anesthesia rather than in Stage 1 (fully awake) . Our patient may probably have laryngospasm as a consequence of extubation or secondary to an unknown risk factor(s) that causes laryngeal hyperreactivity.

In most series patients also have other risk factors which contribute to the upper airway obstruction [10-14]. Lorch and Sahn reported that 50% of the patients in their review had a predisposition to upper airway obstruction secondary to obesity and/or sleep apnea syndrome [12]. Our patient did not have complicated anesthetic courses,laryngospasm, and did not have obesity or a known diagnosis of sleep apnea.The OSA was discovered during the explorations carried out to seek the probable causes of the OAP.

However, increased pulmonary blood flow can also cause transient injury to the pulmonary endothelium which would increase permeability and could contribute to edema formation. The pulmonary edema develops quickly (in less than 10 min). In some patients it follows reintubation and relief of the obstruction suggesting that the positive end-expiratory pressure during laryngospasm retards the formation of edema [4,15,16]. This is the case of our patient, It has been observed consistently that the pulmonary edema occurs following relief of the obstruction by reintubation.

Patients who are predisposed to airway obstruction may have an increased risk of airway complications upon extubation after general anesthesia. Prior to extubation, it would be prudent to ensure that these patients are completely awake, thus ensuring optimal upper airway muscular tone (Figure 1).

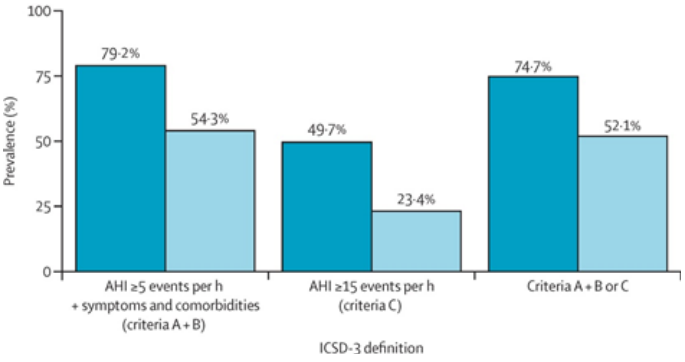

Figure 1. Prevalence of sleep aponea syndrome in the middle to old age general population. **Note:** (■) Men (■) Women.

Conclusion

In summary, postextubation pulmonary edema continues to occur. It usually develops in healthy young men who may not have any risk factors for its development. This suggests that this syndrome can occur in any patient undergoing general anesthesia and that this syndrome may develop following lapses in anesthetic technique, especially extubation .

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