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Acute dyspnea during the general anesthesia recovery period: do not overlook negative pressure pulmonary edema

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TO THE EDITOR:

Negative pressure pulmonary edema (NPPE) is a type of noncardiogenic edema that may occur after an abrupt increase in negative pressure within the pleural space secondary to contraction of the inspiratory muscles in an attempt to overcome an acute obstruction in the upper airway. NPPE is a potentially serious complication during the recovery period from general anesthesia, occurring in up to 0.1% of patients undergoing orotracheal intubation, also accounting for 5-10% of all episodes of upper airway obstruction.[1,2]

During anesthetic practice, NPPE is more common in young and healthy men, who have greater muscle mass and therefore generate greater variations in intrapleural negative pressure.[2] Although NPPE usually develops shortly after extubation, this phenomenon can eventually occur after a few hours. The major symptoms are dyspnea and cough with foaming serosanguineous secretion. Upon physical examination, tachycardia, snoring, rales, and stridor may be present. In most severe forms, bradycardia, paradoxical breathing, and cyanosis can be observed.[1-5]

We herein report the case of a 26-year-old man with no previous comorbidities who underwent elective bilateral flexible ureterorenoscopy and lithotripsy with the insertion of a double J catheter for the treatment of right renal and left ureteral stones. The procedure was performed under balanced general anesthesia with laryngeal mask airway (LMA) use. SpO_2 and blood pressure before anesthetic induction were 99% and 130/80 mmHg, respectively. There was no sign of aspiration of gastric contents during the procedure. Fluid balance remained close to zero, and no significant blood pressure changes were observed. The patient evolved with an acute episode of respiratory distress (tachydyspnea, cough, and frothy, bloody sputum; SpO_2 dropped to 86%) in the recovery room while waking up from general anesthesia 15 min after LMA removal. Pulmonary auscultation showed crackling rales predominantly at the base of the right hemithorax. There was no stridor at that time. Arterial blood gas analysis revealed partial pressure of oxygen of 41 mmHg and arterial oxygen saturation of 74% in room air. Oxygen supplementation was started with a non-rebreathing mask with a flow of 5 L/min.

Bedside chest radiography was performed and showed subtle ill-defined opacities more evident in the lower right lung field (Figure 1A). The patient underwent assisted ventilation with 10 cmH_2O CPAP and 100% oxygen. After 30 min, there was an improvement in SpO_2 (98%), with reduction of respiratory distress. Intubation was not considered due to the clinical improvement. Then, he underwent pulmonary CT angiography, which was negative for pulmonary embolism and showed several small, sometimes coalescing, consolidative opacities associated with ground-glass opacities affecting all of the lobes in both lungs, predominantly in peribronchovascular regions, more extensive in the lower lobes (Figure 1B-1D). Laboratory tests revealed 9,600 cells/μL leukocytes, 13.5 g/dL hemoglobin, and normal platelet count. Serum electrolytes, renal function, echocardiogram, and electrocardiogram were normal. The patient was referred to the ICU, and CPAP was maintained in 60-min sessions every 8 h, with weaning from oxygen therapy after 24 h. He was discharged after two days with complete resolution of symptoms and radiological abnormalities.

In summary, the clinical scenario was an acute episode of respiratory distress in a young male patient during the general anesthesia recovery period with LMA use. The main differential diagnoses, including pulmonary embolism and cardiogenic edema, were ruled out. Due to the clinical course of rapid resolution of pulmonary edema combined with a clear causative factor and exclusion of differential diagnoses, it was concluded that the negative pressure mechanism after a transient laryngospasm was responsible for the clinical presentation. Thus, the presumptive diagnosis was NPPE, a form of noncardiogenic pulmonary edema.

NPPE has been subdivided into two types. Type 1 is related to vigorous inspiratory effort in case of acute airway obstruction, such as post-extubation laryngospasm or epiglottitis, and type 2 occurs after the relief of chronic partial airway obstruction, such as after adenoidectomy.[6-8] The case presented herein was type 1 NPPE.

Type 1 NPPE most commonly occurs in young patients during the recovery period from general anesthesia with orotracheal intubation. However, with the accelerated adoption curve of LMA in clinical practice, several cases of NPPE with LMA use have been reported.[8-10]

The pathogenesis of type 1 NPPE is multifactorial. Inspiratory effort to overcome the obstruction generates

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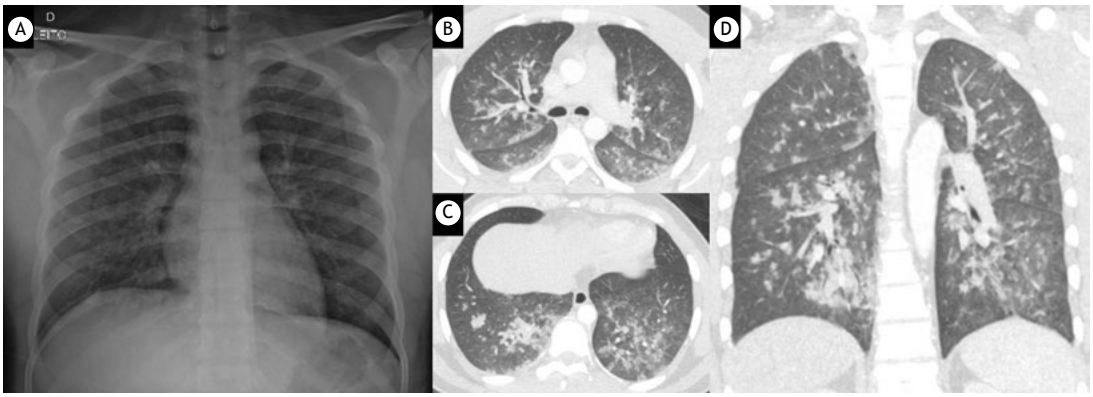

Figure 1. In A, Anteroposterior chest radiograph (bedside) demonstrated subtle ill-defined opacities, more evident in the lower right lung field. In B, C, and D, pulmonary CT angiography images (lung window) in axial (B and C) and coronal (D) planes revealed several small, sometimes coalescing, consolidative opacities associated with ground-glass opacities affecting all of the lobes in both lungs, predominantly in peribronchovascular regions, more extensive in the lower lobes. These findings are consistent with pulmonary edema (in this case, negative pressure pulmonary edema). It is worth mentioning that the exam was negative for pulmonary embolism.

an increase in intrapleural negative pressure. The "siphon effect" within the rib cage increases venous return to the right heart chambers, which produces an increase in hydrostatic pressure in the pulmonary capillaries and causes fluid to move out of the vessels into the interstitial and alveolar space, causing edema, imbalance between ventilation and perfusion, and, consequently, hypoxemia. It may trigger hypoxic pulmonary vasoconstriction and raise pulmonary vascular resistance. By increasing the right ventricular volume, the interventricular septum may shift leftward, with reduced left ventricular diastolic compliance. Increased cardiac afterload and myocardial hypoxia lead to decreased left ventricular function, with increased pulmonary venous pressure. Hypercapnia, acidosis, hyperadrenergic response, and loss of capillary integrity may be contributing factors in the pathophysiology.[1-3,6,8]

NPPE has a quick onset, commonly within minutes, and a relatively rapid resolution. If recognized and treated early, NPPE is usually a self-limited condition and, thus, reversible, with relevant clinical and radiological improvement, frequently in 12-48 h. The diagnosis of NPPE can be made on the basis of the presence of a precipitating situation and compatible symptoms. Chest radiograph and/or CT findings of pulmonary edema support the diagnosis.[1,4,6,8]

The management of NPPE is mainly supportive and includes its early recognition followed by upper airway clearance, supplemental oxygen therapy, and use of noninvasive ventilation (including CPAP). In extreme cases, (re)intubation with a certain level of PEEP may be required. Muscle relaxation with low doses of succinylcholine can relieve laryngospasm.[1-6,8] The role of steroids and diuretics is still controversial, and there is no clear recommendation regarding their use.[1]

In conclusion, medical practitioners should be aware of NPPE, and a correct understanding of pathophysiological mechanisms behind this condition is essential for early diagnosis and proper management.

AUTHOR CONTRIBUTIONS

BBL, BLM, and DRC: conception, study planning and design, data collection, selection of the images, and literature review. BLM, DRC, and FMC: writing of preliminary drafts and final version of the manuscript. BLM, FMC, and PRPS: revision of the manuscript. All authors read and approved the final version of the manuscript.

CONFLICTS OF INTEREST

None declared.

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