



Negative pressure pulmonary edema

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Dear Editor,

We read with great interest the reporting of a case with recurrent, acute negative pressure pulmonary edema.¹ The case is didactic in underlying that when the main causative factor is still present (external compression of the larynx in combination with inspiratory negative pressures in the airway) the syndrome cannot stop from resurging repetitively.

We would like to point that according to the reporting we believe that the true etiology is a combination of NPPE type I and II. It seems that this patient had already developed the pathophysiology of type II NPPE due to progressive, though not critical (as flexible laryngoscopy revealed) narrowing of the larynx due to the external compression from the growing goiter. So, the patient was on the edge on manifesting a type II NPPE (recent onset of exertional dyspnea) when something critical ensued - an undetected infection that led to mucosal edema or the sudden onset of narrow complex tachycardia - as the authors wisely detected and states. So, the presenting episode of NPPE is presumably of type II, following the pathophysiology that the authors elegantly explained.

The following two episodes could be explained as type I NPPE. The institution of positive pressure ventilation (after each episode) in combination with diuretic treatment stabilized patient's respiratory and circulatory status to normal patterns that permitted clinicians to wean the patient off the ventilator. To our

opinion the switch from positive pressure ventilation to negative pressure spontaneous ventilation was the trigger of the subsequent episodes of desaturation and type I NPPE. The softening of the trachea, discovered later, in combination with the inspiratory efforts of an obese patient produced typical negative pressure pathophysiology in the setting of a functional upper airway obstruction.

In an older publication, we reported the presentation and treatment of a case of type I NPPE in a young athlete that forcefully bit the laryngeal mask while emerging from anesthesia.² The negative airway pressures elevate right ventricular volume and produce displacement of the ventricular septum towards the left ventricle, thus reducing its compliance. Reduced compliance further elevates the wall tension of the left ventricle (of this hypertensive, diabetic patient). Catecholamine secretion (either from the circulatory stress or hypoxia) increases systemic vascular resistance (and might also have produced the reported SVT). These concomitantly acting imbalances decrease the ejection fraction of the left ventricle and produce a typical pulmonary edema. Certainly, these swift and completely reversible derangements can reverse rapidly, when the triggering factor is withdrawn.

Nevertheless, we applaud the authors that managed to effectively manage the patient this series of critical and life-threatening situations and successfully restored his neck anatomy close to normal.

REFERENCES

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