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Pulmonary Edema in Severe Bronchospasm

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TO THE EDITOR

I read the case report published in the Journal[1] with interest. In their report, acute pulmonary edema developed after relief of severe bronchospasm that was related to cardiopulmonary bypass, which was used for repair of atrial septal defect in 52-year-old woman. I have more interest in the pulmonary edema than bronchospasm. The explanation for the development of pulmonary edema in this case report provided by authors deserves some comments.

During severe bronchospasm, a large negative intrathoracic pressure is generated in the patient with asthma, while the patient is breathing spontaneously.[2] This large negative Intrathoracic pressure increases venous return to the right ventricle, leading to an increase in pulmonary blood volume. Intrathoracic negative pressure surrounding left ventricle and the descending and thoracic parts of aorta holds blood volume in the thorax, leading to an increase in left ventricular afterload. The increased pulmonary blood volume and left ventricular afterload elevate hydrostatic pressure in the pulmonary capillary. In addition, the negative intrathoracic pressure decreases pulmonary interstitial pressure. The combined effects would be an increase in the pressure gradient favoring filtration of edema fluid into the lung, leading to pulmonary edema. However, if the patient develops bronchospasm while the patient is on positive pressure ventilation (ventilated mechanically), then the intrathoracic pressure is largely positive rather than negative and the interstitial pressure is elevated. Therefore, pulmonary edema does not develop, when the severe bronchospasm is relieved in this situation. Moreover, the airway obstruction in asthmatic bronchospasm is an intrathoracic lower airway obstruction, which becomes less when lung volume increases or during inspiration. On the other hand, extrathoracic airway obstruction becomes more severe during inspiration, during which a large negative intrathoracic pressure is generated. Relief of airway obstruction in this situation leads to acute postobstructive pulmonary edema, also called as acute negative pressure pulmonary edema as authors referred.[3]

It is not likely that the pulmonary edema developed after relief of bronchospasm in the patient reported by authors was an acute postobstructive pulmonary edema. Chest radiograph is not consistent with acute postobstructive pulmonary edema which usually involves in a more diffuse homogeneous pattern without peripheral sparing. Chest radiograph is more consistent with an ischemia-reperfusion injury of the lung. During intrathoracic lower airway obstruction such as severe bronchospasm, lung is inflated by positive pressure ventilation but is not deflated, leading to overinflation of lung. In overinflated lung the high intraalveolar pressure compresses pulmonary capillary, leading to ischemia. In the case report by authors,[1] while struggling to improve ventilation, an enough time elapsed for the ischemia to develop, as evidenced by high PCO_2. Once the bronchospasm was relieved, reperfusion took place, resulting in an ischemia-reperfusion injury of the lung. Interestingly, it has been proposed that the phases of hypoventilation/hypoperusion with hypoxic-acidosis in severe asthma and reoxygenation upon relief of bronchospasm are similar to the phases of ischemia-reperfusion of the lung. Both phases involve interleukin-1 β mediated neutrophil activation.[4] If so, one can expect that ischemia-reperfusion injury of the lung can develop without a complete ischemia.

In summary, I think that the pulmonary edema after relief of intense bronchopasm that was related to cardiopulmonary bypass in the case reported by authors[1] is not an acute postobstructive pulmonary edema but an ischemia-reperfusion injury of the lung.

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