Pulmonary haemorrhage associated with negative-pressure pulmonary oedema: a case report

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ABSTRACT

Negative-pressure pulmonary oedema caused by upper airway obstruction after tracheal extubation is well recognised, but extensive pulmonary haemorrhage is rare. We report a case of post-extubation, laryngospasm-induced pulmonary oedema with associated pulmonary haemorrhage. The patient required mechanical ventilation with high positive end-expiratory pressure.

450 mL, positive end-expiratory pressure of 15 cmH₂O, respiratory rate of 20 breaths per minute, and FIO₂ of 1, which was weaned to 0.4 over the next 12 hours. A pulmonary artery catheter inserted 3 hours after ICU admission showed a pulmonary artery occlusion pressure of 12 mmHg, central venous pressure of 12 mmHg, pulmonary artery systolic pressure of 32 mmHg, and cardiac index of 2.19 L/min/m². The patient's clinical course was consistent with non-cardiogenic, negative-pressure pulmonary oedema (NPPE).

The next day, repeat laboratory tests showed a drop in haemoglobin concentration to 140 g/L. This was associated with the ongoing appearance of frank blood on suctioning via the endotracheal tube. Repeat chest radiography showed a slight decrease in pulmonary infiltrates. Blood tests for vasculitis and serum urea and creatinine levels were normal. No organisms were cultured from the tracheal aspirate.

Over the next few days, the pulmonary haemorrhage abated, and chest radiography showed progressive resolution of infiltrates. The patient was successfully extubated on the fifth postoperative day.

Discussion

Negative-pressure pulmonary oedema is well defined in the literature, with a reported incidence as high as 11%. It is reported more commonly in young patients after surgery when laryngospasm complicates extubation. It is also described in ICU patients following endotracheal tube occlusion, and in children with epiglottitis. It resolves rapidly with restoration of a patent airway and positive-pressure ventilation; pulmonary haemorrhage is rare.
A proposed mechanism for the development of pulmonary oedema in this situation is the generation of extreme negative intrathoracic pressure, leading to more negative interstitial pressure. This increases the hydrostatic pressure gradient across the pulmonary microcirculation and increases transudation of fluid into the lungs. This has been demonstrated in animal models. The haemoconcentration seen in our patient during this episode favours this hypothesis.

The radiographic findings of perihilar and upper-zone alveolar and interstitial infiltrates seen in our patient are typical in NPPE. They may be explained by the more negative pleural and interstitial pressure in central and non-dependent regions, creating a larger gradient across the alveolar–capillary membrane in these zones compared with the dependent regions of the lung.

The mechanism for pulmonary haemorrhage associated with NPPE is not clear, but disruption of the alveolar–capillary membrane caused by large negative pressure swings is most likely. In one patient with pulmonary haemorrhage, bronchoalveolar lavage was suggestive of an alveolar origin of bleeding. Some reports suggest bronchial vessel disruption as a cause of pulmonary haemorrhage. Bronchoscopy in one patient found punctate haemorrhage throughout the tracheobronchial tree.

In conclusion, NPPE may present as pulmonary haemorrhage and require positive-pressure ventilatory support for some time. The radiographic finding of perihilar and upper-zone alveolar and interstitial infiltrates may help in its diagnosis.

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