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.

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Clinical record

A 53-year-old man underwent general anaesthesia for left total knee replacement. He had a history of depression and psoriasis, and was taking low-dose methotrexate long term. Preoperative physical examination and laboratory investigations gave normal results.

After induction of anaesthesia with propofol, rocuronium was administered for muscle relaxation; tracheal intubation was uneventful. The patient remained haemodynamically stable during surgery, and was moved to the recovery area and extubated. Within a few minutes of extubation, he developed a vigorous cough, followed by laryngospasm, hypoxia and hypertension. A laryngeal mask airway was inserted, aided by propofol (20 mg) administered intravenously. However, despite a patent airway, hypoxia persisted (oxygen saturation by pulse oximetry, approximately 80%), and frothy pink fluid was aspirated from the laryngeal mask airway. Intensive-care review was urgently requested, by which time fresh blood was evident in the laryngeal mask. Ventilation with continuous positive airway pressure (CPAP; 10 cmH_2O) was begun by face mask after removing the laryngeal mask, and the patient was transferred to the ICU.

On examination, the patient was conscious and oriented, but coughing pink frothy sputum. Systolic blood pressure was 190 mmHg, and bilateral coarse crackles were heard on chest auscultation. There were no petechiae, haematuria or mucosal bleeding. Initial assessment of arterial blood gases on CPAP with fraction of inspired oxygen (FIO_2) of 1 showed: pH, 7.32; Po_2, 151 mmHg; Pco_2, 59.8 mmHg; and HCO_3, 24.1 mmol/L. An electrocardiogram showed sinus tachycardia. Troponin T measurement and coagulation and other laboratory tests were unremarkable. Transthoracic echocardiogram was technically difficult, but suggested normal chamber size and function, with no regional abnormality of wall motion. Chest radiography showed bilateral perihilar interstitial and alveolar infiltrates, while upper-lobe air bronchograms suggested pulmonary oedema, although the lower zones were remarkably normal (Figure). Haemoglobin concentration was 178 g/L, compared to 164 g/L before surgery; the patient had received no blood intraoperatively.

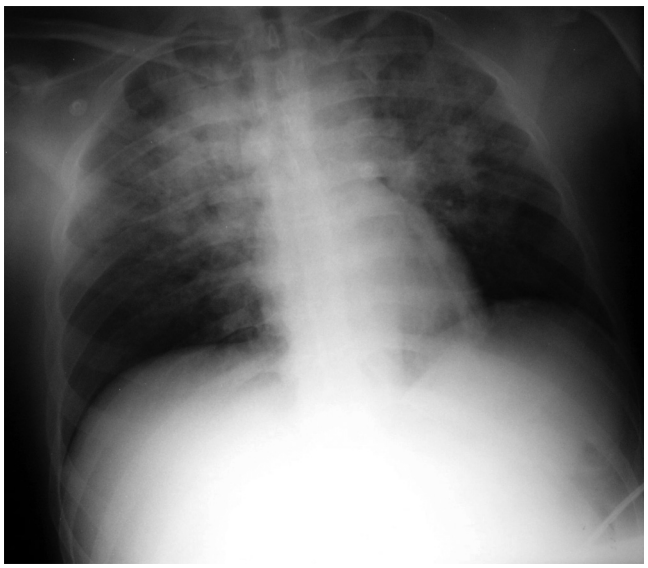
Despite CPAP ventilation by face mask, the patient's condition failed to improve, and he required reintubation. Laryngoscopy during intubation showed fresh blood at the larynx. He was then sedated and ventilated with a tidal volume of 450 mL, positive end-expiratory pressure of 15 cmH_2O, respiratory rate of 20 breaths per minute, and FIO_2 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/m^2. 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%.[1] 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,[2] and in children with epiglottitis. It resolves rapidly with restoration of a patent airway and positive-pressure ventilation; pulmonary haemorrhage is rare.



Chest x-ray in the ICU, showing bilateral mid- and upper-zone interstitial and alveolar infiltrates, with clear lower zones.

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.[3] This has been demonstrated in animal models.[4] The haemoconcentration seen in our patient during this episode favours this hypothesis.[5]

The radiographic findings of perihilar and upper-zone alveolar and interstitial infiltrates seen in our patient are typical in NPPE.[6] 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.[6]

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.[7-9] In one patient with pulmonary haemorrhage, bronchoalveolar lavage was suggestive of an alveolar origin of bleeding.[3] Some reports suggest bronchial vessel disruption as a cause of pulmonary haemorrhage. Bronchoscopy in one patient found punctate haemorrhage throughout the tracheobronchial tree.[10]

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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