

Acute respiratory distress syndrome in a child with cerebral palsy

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Citation

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Abstract

Background: We report a case of pulmonary edema following laryngospasm in a child with cerebral palsy. Even though the initial presentation was suggestive of negative pressure pulmonary edema, a diagnosis of acute respiratory syndrome was made later. We discuss the need to differentiate acute lung injury or acute respiratory distress syndrome from simple negative pressure pulmonary edema. **Case report:** An 8-year-old-girl with cerebral palsy developed laryngospasm shortly after extubation. Serious oxygen desaturation ensued, treated with 100% Oxygen and continuous positive airway pressure. Within minutes patient developed clinical features resembling negative pressure pulmonary edema. Blood stained secretions appeared in the tracheal tube following re-intubation. Chest X-ray showed bilateral 'fluffy' infiltrates with normal heart size and $\text{PaO}_2:\text{F}_i\text{O}_2$ remained <200 for more than 24 hours, suggestive of acute respiratory distress syndrome. Child recovered after few days after intensive management in critical care unit. **Conclusion:** Differentiating acute lung injury or acute respiratory distress syndrome from simple negative pressure pulmonary edema will help initiate aggressive management and improve outcome.

INTRODUCTION

Pulmonary edema can occur following airway obstruction. These reports are usually put under the category of negative pressure pulmonary edema (NPPE)¹⁻⁷. This report describes a case of pulmonary edema following laryngospasm in a child with cerebral palsy. Early diagnosis of acute respiratory distress syndrome (ARDS) lead to intensive management and complete recovery.

CASE REPORT

A 8year old girl, weighing 8 kg (malnourished) with spastic cerebral palsy was scheduled for hip adductor release. She had past history of recurrent chest infections and tonic posturing. On examination drooling of saliva, mild scoliosis and spasticity were present. Chest X-ray showed apparently normal lungs.

Child was premedicated with Famotidine 10mg, Metoclopramide 5mg and Midazolam 4mg p.o. Anaesthesia was induced with Sevoflurane (upto 6%) in 100% Oxygen. Fentanyl 20 µg was given i.v. after securing intravenous line with difficulty. Trachea was intubated with 5.5 mm cuffed tracheal tube by direct laryngoscopy. Neuromuscular blockade was achieved with Atracurium 5 mg i.v. and anaesthesia maintained with N₂O:O₂ (2:1) and Isoflurane

(upto 1%). The surgical procedure lasted for an hour with minimal blood loss and Ringer's lactate 250 ml was administered. Paracetamol 250 mg was administered per rectum for post-operative pain relief. Neuromuscular blockade was reversed with Neostigmine and Atropine. Shortly after extubation the patient developed laryngospasm with severe intercostal retractions and absent breath sounds. Arterial oxygen saturation (SpO₂) decreased to 70% with bradycardia. It was relieved by CPAP with 100% oxygen within a minute and SpO₂ improved to 92%. SpO₂ remained at 92% despite 100% oxygen for more than five minutes. Fine crepitations appeared on auscultation over the entire chest. Negative pressure pulmonary edema was suspected and trachea was re-intubated with 5.0 mm cuffed tracheal tube after Morphine 3mg i.v. Bright red coloured frothy secretions appeared in the tracheal tube. Lungs were ventilated with 100% oxygen and positive end expiratory pressure (PEEP). Blood pressure was within normal limits. Chest X-ray showed bilateral diffuse 'fluffy' infiltrates and normal heart size suggestive of acute lung injury (ALI) or ARDS (Figure-1). Central venous pressure, electrocardiogram and echocardiogram were normal. $\text{PaO}_2:\text{F}_i\text{O}_2$ remained <200 for more than 24 hours.

Figure 1

Figure 1: Chest X-ray showing bilateral 'fluffy' lung infiltrates suggestive of ARDS.



The child had recurrent episodes of arterial oxygen desaturation with appearance of bright red frothy secretions in the tracheal tube. High PEEP (10 to 12 cmH₂O) was required to maintain arterial oxygen saturation in the next few days. Laboratory investigations showed normal renal and liver functions including normal plasma proteins. The child improved over the next few days and was successfully extubated on the seventh postoperative day.

DISCUSSION

There have been many reports of pulmonary edema following upper airway obstruction, usually put under the category of NPPE. It is often self-limited, resolving within hours with or without positive pressure ventilation^{3,8}. But there are few reports describing severe pulmonary edema or even bleeding after airway obstruction⁹⁻¹². The above patient developed pulmonary edema following laryngospasm which could have been triggered by muscle spasticity, slow recovery from inhalational anaesthesia and postoperative pain^{13,14}. The high negative intrapleural pressure caused by vigorous inspiratory attempts against a closed glottis, could have triggered pulmonary edema. Rapid progression to ARDS could be because of pre-existing alveolar damage indicated by recurrent chest infections. Patients with cerebral palsy develop silent pulmonary aspirations due to gastroesophageal reflux leads and poor coordination of pharyngeal and laryngeal muscles. Respiratory complications are the most frequent cause of death in these

patients^{13,14}.

The high negative intrapleural pressure is the main precipitating event in the development of edema following upper airway obstruction. Damage to alveolar-capillary membrane by hypoxia, acute aspiration of gastric contents or pre-existing lung pathology could worsen the problem leading to ARDS⁸. It is essential to differentiate ALI/ARDS from simple negative pressure pulmonary edema, since the management has to be more aggressive with the former. Even though the initial clinical picture is similar, presence of blood in the lung secretions, bilateral diffuse 'fluffy' infiltrates on chest radiography and persistently low PaO₂:F_iO₂ should help differentiate ALI/ARDS from negative pressure pulmonary edema. Early detection and aggressive management may help reduce the morbidity and mortality in these patients.

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