Post Obstructive Pulmonary Edema in a Child who Attempted Suicidal Hanging

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ABSTRACT

Postobstructive pulmonary edema occurs rarely in children. We describe here a child who attempted suicide by hanging and developed postobstructive pulmonary edema and was successfully managed. There was a rapid response to management with morphine and supportive care, enabling extubation by 30 hours of ventilatory support. The case highlights an unusual cause of postobstructive pulmonary edema. [Indian J Pediatr 2008; 75 (10) : 1075-1077] E-mail: rakesh_lodha@hotmail.com

Key words : Postobstructive pulmonary edema; Children; Hanging

Postobstructive pulmonary edema (PPE), also termed negative pressure pulmonary edema (NPPE), is a relatively rare cause of pulmonary edema occurring at the time of or following the relief of an upper airway obstruction, usually as a complication of laryngospasm post-extubation. However, PPE may occur in a large spectrum of pathologies associated with upper airway obstruction, and awareness of this condition is crucial during daily clinical practice. We report a case of postobstructive pulmonary edema in a child following attempted suicidal hanging to highlight this condition.

CASE REPORT

A 12-year-old boy with poor scholastic performance and frequent school absenteeism got upset with his mother when refused a toy that he demanded after a satisfactory result in a school examination. He went to another room, and half an hour later, father found the child hung by a ‘duppatta’ (a long scarf worn by women) from a door’s latch, in a kneeling position, with his neck extended, unconscious, and not breathing well. Child regained consciousness after stimulation. En-route to the hospital, child started breathing laboriously and appeared disoriented. Breathing difficulty worsened with time, with increased cough and expectoration of blood stained frothy secretions.

When seen in the emergency room about ninety minutes after the event, the child was drowsy but responsive to painful stimuli, afebrile, with heart rate of 105/min, respiratory rate of 44/min, feeble pulses and blood pressure of 96/60 mm Hg. The SaO₂ was 90% on room air. The child had chest indrawing and chest auscultation revealed bilateral crepitations. Cardiovascular and per abdomen examination were unremarkable. GCS was E2M5V2, pupils were 2-3 mm but reactive, bilateral deep tendon jerks were brisk and the plantars were down going. Initial diagnosis considered was that of attempted hanging with either cervical spine injury or intracranial bleed, with pulmonary edema/ aspiration.

In view of severe respiratory distress, child was intubated; pink frothy secretions were noted below the glottis. The child was mechanically ventilated with volume controlled mode of ventilation using Drager Evita 2 ventilator, with a tidal volume of 250 ml (7 ml/kg), rate 20/min, inspiratory time of 0.9 second, FiO₂ 100%, and PEEP 12 cm H₂O. Child was given one dose of morphine 0.1 mg/kg for pulmonary edema, and shifted to the pediatric intensive care unit (PICU).

In the PICU, vital parameters were similar to that recorded earlier; SaO₂ was 82%. Endotracheal secretions were profuse, pink, and frothy, suggesting pulmonary edema. Child was administered morphine again. Thereafter, the child was administered dopamine 10 µg/kg/min and dobutamine 10 µg/kg/min. The secretions decreased significantly though crepitations persisted,
more on the right side of chest.

In the ICU, initial arterial blood gas analysis revealed a pH 7.283, PCO₂ 45.3 mm Hg, PO₂ 201.7 mm Hg, HCO₃⁻ 20.8 mEq/L, and a base deficit 5.6. Serum electrolytes were normal; arterial blood lactate was 1.6 mmol/L, and blood sugar 155 mg/dl. Hemoglobin was 12.8 g/dl and white blood cell count was of 31,600/μL. Renal and liver function tests were normal.

Chest radiograph at admission was suggestive of pulmonary edema and showed bilateral, diffuse infiltrates, more on the right, fluid in the horizontal fissure, and a normal cardiac silhouette (Fig. 1). A standard 12-lead ECG showed sinus tachycardia without any other abnormality. Cardiac troponin I (Enzyme linked fluorescent assay, VIDAS, Biomerieux, France) was slightly elevated at 1.26 µg/L (normal value < 0.16 µg/L), while CK-MB fraction was normal at 22 IU/L against a CK value of 395 IU/L (normal: up to 6% of Creatine Kinase). Transthoracic echocardiography revealed normal biventricular function.

Hemodynamic stability was achieved and dopamine and dobutamine were discontinued after 23 hours and 50 hours of infusion respectively. By 24 hours of ICU stay, chest crepitations had resolved and ventilatory settings could be brought down. Child was extubated at 30 hours of ICU stay and repeat chest radiograph was normal (Fig 2). Child received oxygen by face mask at 3 liters per minute for 4 hours, and subsequently maintained saturation above 95% in room air. Neurological examination was normal. Post extubation arterial blood gas analysis was normal.

At admission, non-contrast computed tomography of brain had been normal, while that of cervical spine suggested grade I anterolisthesis of C2 over C1. Injection methylprednisolone was given over 24 hours in view of suspected cord compression. However, an MRI of the neck subsequently revealed no abnormality, and neurological examination being normal, IV steroids were discontinued.

Sensorium was normal post extubation; the child was shy, not forthcoming with details of what prompted him to attempt suicide. A psychiatry opinion was taken; the act seemed to be impulsive and not premeditated. In the absence of past personal or family history of psychiatric illness or suicidal attempts, and lack of obvious familial dysfunction or conflicts, the risk of suicide was adjudged as being low. The child was discharged after four days of stay and advised to follow up regularly.

The Institute Ethics Committee waived off the need for consent to report this case.

**DISCUSSION**

We have described here post-obstructive pulmonary edema in a child with unsuccessful hanging. This is an uncommon occurrence.

Post-obstructive pulmonary edema (PPE) or negative-pressure pulmonary edema (NPPE), an entity of chiefly anesthesiologic relevance, occurs usually perioperatively due to laryngospasm.¹ It develops acutely, immediately after onset of acute airway obstruction like laryngospasm or epiglottitis (PPE type I) or after the relief of chronic upper airway obstruction such as adenotonsillar hypertrophy (PPE type II).² However, literature suggests that it is more common than recognised, occurring in 0.05 to 0.1% of all anaesthesia administrations, being often misdiagnosed.³

Other than with laryngospasm, PPE type I in children may occur with anatomical anomalies, epiglottitis, croup, impaction by foreign body, postextubation subglottic