Post-Obstructive Pulmonary Edema Following Accidental Strangulation: POPE - A Rare Medical Emergency

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ABSTRACT
Post obstructive pulmonary edema (POPE), a rare form of non-cardiogenic pulmonary edema, is a lesser known medical emergency which occurs due to acute upper airway obstruction (Type I) or following surgical relief of chronic airway obstruction (Type II). Though cases of POPE have been reported after various causes of upper airway obstruction but POPE developing after strangulation have not been reported widely. We report a 14 years old boy who developed POPE following accidental strangulation and complete recovery following prompt treatment with ventilator support and supportive care.

KEY WORDS: acute upper airway obstruction, NPPE, pulmonary edema, POPE, strangulation

INTRODUCTION:
Post obstructive pulmonary edema (POPE), also known as ‘Negative pressure pulmonary edema’ (NPPE) is a rare form of non-cardiogenic pulmonary edema. It generally manifests as potentially life threatening fulminant pulmonary edema that becomes apparent within seconds to minutes following acute upper airway obstruction (UAO) or relief of chronic UAO. Majority of POPE cases have been recognized postoperatively by anesthetists as a consequence of laryngospasm.[1] Other reported causes of POPE syndrome are croup, epiglottitis, mass effect secondary to tumor, choking,[2] strangulation and interrupted hanging.[3,4]

The association between UAO and pulmonary edema was described for the first time in animal experiments on dogs by Moore and Binger (1927),[5] in children by Capatanio and Kirkpatrick (1973)[6] and in adults by CE Oswalt et al (1977).[7] Very few cases have been published of POPE following strangulation.

CASE REPORT:
A 14 year old boy was brought in altered sensorium to emergency department by his relatives with alleged history of accidental strangulation while playing on a swing made from a saree. He was freed from the saree within minutes and rushed to our hospital within ½ hour of the incidence. On admission, he was very irritable with decerebrate posturing on painful stimuli. Blood tinged froth was coming out of his mouth and a ligature mark was noted around his neck (Figure 1). His vital signs revealed pulse rate 146/minute, blood pressure 80/60 mmHg, respiratory rate 44/minute, Oxygen saturation (SpO₂) 60% on room air which increased to 70% on 10 litres Oxygen/minute delivered by facemask. His pupils were semi-dilated and sluggishly reacting to light. Chest auscultation revealed bilateral coarse crepitations. He was immediately sedated with intravenous Midazolam, intubated and put on ventilator support with synchronized intermittent mandatory ventilation (SIMV) mode initially with tidal volume 350 ml, FiO₂ 100%, positive end expiratory pressure (PEEP) of 8 which was later increased to 10. Subsequently, Propofol infusion was
added to prevent patient fighting with ventilator and mode changed to intermittent positive pressure ventilation (IPPV). He required frequent endotracheal tube suctioning to clear his airways for initial 1 hour; more than 400 ml of blood stained frothy secretions were suctioned (Figure 2). His fundus examination was normal. Electrocardiogram showed sinus tachycardia. Laboratory tests for complete blood count, renal functions, liver functions, electrolytes, and urine routine examination were normal. Chest radiograph revealed bilateral fluffy opacities suggestive of pulmonary edema (Figure 3). Arterial blood gas (ABG) analysis done after putting patient on ventilator at FiO₂ of 100% revealed pH of 7.241, PaO₂ of 78.7 mmHg, PaCO₂ of 49.2 mmHg and HCO₃⁻ of 22.3 mmol/L, suggestive of hypoxemia with acute respiratory acidosis with metabolic acidosis. Considering the decerebrate posturing and pulmonary edema with PaO₂/FiO₂ ratio of < 100, a provisional diagnosis of ‘Hypoxic encephalopathy with acute respiratory distress syndrome (ARDS)’ was made. He was shifted to ICU and treated with intravenous diuretic, cerebral decongestants (Furosemide, Mannitol 20%) and Dexamethasone along with antibiotic and supportive care of unconscious patient. SpO₂ improved gradually to >90% over next 6 hours and repeat ABG showed pH of 7.383, PaO₂ of 110.8 mmHg, PaCO₂ of 44.2 mmHg and HCO₃⁻ of 23.8 mmol/L. His lungs were clear of crepitations by 24 hours. After 1½ days of admission, FiO₂ was slowly tapered to 40% and Propofol infusion was tapered and stopped in next 24 hours. Ventilator mode was changed to SIMV (F-14, TV – 350 ml, FiO₂ – 35%) and PEEP reduced to 5. At 2½ days of admission, Midazolam was also tapered and discontinued after which his consciousness started improving though he remained a bit irritable. His vitals were stable with pulse rate 90–106/minute, BP 100-110/60-70 mmHg, respiration rate of 18-22/minute maintaining SpO₂ of >95% at FiO₂ of 35%. As he was still disoriented, a T-piece trial was given; somehow patient self extubated in 4 hours.

Subsequently, his SpO₂ was monitored and managed with Oxygen by nasal prong at 2 litres/minute. Repeat X-ray chest done on 4th day showed complete resolution of pulmonary edema (Figure 4). By the end of 4th day, Oxygen was also
stopped and he was fully oriented to place and person but had amnesia for the strangulation event. His Echocardiography was normal. He was monitored for next 3 days and discharged with full recovery on 7th day of hospitalization.

DISCUSSION:

Though this patient was admitted with a provisional diagnosis of Hypoxic encephalopathy with ARDS, a possibility of POPE syndrome was suspected by the corresponding author based on his past experience and review of patient’s history. Rapid recovery of pulmonary edema within 2 days supported this diagnosis too. Pulmonary edema developing after non-fatal strangulation has been reported very infrequently; most probably due to a poor survival rate amongst victims of strangulation. Recommended management of POPE/NPPE includes use of Continuous Positive Airway Pressure (CPAP) or Positive End Expiratory Pressure (PEEP) in patients requiring mechanical ventilation ± diuretics. Clinical and radiographic improvement has been reported in 12 to 24 hours in majority of cases. Present case was also managed with standard recommendations.

POPE may occur within minutes to upto 6 hours in majority of patients, following either the development or relief of acute severe upper airway obstruction. However, delayed onset beyond 12 hours after the precipitating event has also been reported. Therefore, all patients should be observed and monitored for development of pulmonary edema after suffering from acute severe upper airway obstruction. Two subtypes of POPE/NPPE have been described-

POPE/NPPE Type I- develops following acute upper airway obstruction. Post-extubation laryngospasm is the most common cause. It may also be caused by hanging, strangulation, upper airway tumors, chocking due to foreign body, migration of Foley’s catheter balloon used to tamponade the nose in epistaxis and near drowning. Present case falls under this category.

POPE/NPPE Type II- develops following relief of upper airway obstruction - post operative patients with big tonsils, hypertrophic adenoids and hypertrophic redundant uvula.

The pathophysiology for development of pulmonary edema in POPE type I is attributed to many factors -

- As patient tries hard to breathe in against closed upper airway, it generates marked negative intrathoracic pressure. This reduces interstitial pressure causing shift of fluid from pulmonary capillaries to interstitium and alveoli.
- Hypoxemia as a result of UAO leads to peripheral vasoconstriction causing increased venous return to right heart and thus increased pulmonary circulation with increase in pulmonary capillary pressure favouring fluid shift to low-pressured alveolar spaces.
- Increased negative intrathoracic pressure [–50 to –100 cmH₂O] increases venous return to right heart leading to elevation of ventricular end-diastolic volumes; thus the left ventricular end-diastolic pressures get elevated contributing to formation of pulmonary edema.
- Capillary and endothelial damage may result from shearing forces generated during forced inspiration against a closed airway leading to diffuse alveolar injury and diffuse alveolar hemorrhage.

Pulmonary edema in POPE type II is attributed to sudden removal of auto positive end expiratory pressure (auto PEEP) and thus a sudden decrease in chronically elevated lung pressures. This process is characterized by a sudden change in the intrathoracic pressure, leading to a shift of fluid from the interstitium to the alveoli.
generates a negative intrapulmonary pressure causing transudation of fluid in lung interstitium.\textsuperscript{5,9}

**CONCLUSION:**

POPE/NPPE is often undiagnosed or misdiagnosed; hence an under-reported life threatening medical emergency. Anaesthesiists and critical care specialists encounter POPE mainly due to post-extubation laryngospasm. POPE secondary to strangulation is much lesser known condition and acute pulmonary edema in these cases can be life-threatening requiring mechanical ventilation with high PEEP to maintain oxygenation in addition to supportive treatment. Diagnosis of POPE syndrome is based primarily on clinical presentation-history suggestive of UAO followed by rapid development of agitation, respiratory distress, declining \textit{SpO\textsubscript{2}}, tachypnoea, tachycardia, bilateral crepitations \textit{+} rhonchi over chest with pink frothy sputum and chest radiograph suggestive of pulmonary edema. With early diagnosis and intervention, majority will recover completely within 24-48 hours. There is a need to increase awareness about this entity to all clinicians specially those working in emergency and critical care units and also to differentiate it from ARDS.

**REFERENCES:**