Case Report

Pulmonary Oedema in a survivor of Suicidal Hanging

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Introduction

Post-obstructive pulmonary oedema (POPE), a rare form of non-cardiogenic pulmonary oedema, is generally characterised by fulminant pulmonary oedema that becomes apparent within seconds to minutes after the relief of severe upper airway obstruction. Development of POPE has been reported after various causes of upper airway obstruction but POPE developing after non-lethal hanging has not been reported widely. A case of POPE in a twenty year old girl, which developed after an attempted suicide by hanging, is reported.

Case Report

A 20 year old girl was brought to the hospital with an alleged history of hanging by neck. Patient was rescued and taken to the local Air Force Station Medicare Centre where she was found to be unconscious with peripheral cyanosis, pulse rate was 110/minute and blood pressure was 90/60 mm of Hg. On arrival in the intensive care unit of the hospital she had peripheral cyanosis, her pulse rate was 168/minute, blood pressure was 130/90 mm of Hg and respiratory rate was 40/minute. She had intermittent decerebrate movements and plantar reflexes were extensor on both sides. Air entry was equal bilaterally and coarse crepitations were auscultated on both sides. Copious pink frothy secretions were present in the oral cavity. Pulse-oxymetry revealed SpO₂ of 70%. Trachea was intubated orally using a cuffed, 7.5mm PVC endotracheal tube. A 'T Bird, VSO2' ICU Ventilator was used for artificial ventilation. Weight of the patient was approximately 50 kg. Ventilatory settings were as follows : Mode : Assist control mode (ACMV), FiO₂:1.00, PEEP:5cm of H₂O, Tidal volume : 350 ml, respiratory rate:18/min, inspiratory flow rate : 50 l/min (to achieve I:E of 1:2.5-3) and high pressure alarm limit : 50 cm of H₂O. The peak inspiratory pressure varied between 30-35 cm of H₂O with these ventilatory settings. ECG done in the ICU revealed no abnormality except for sinus tachycardia. Blood gas analysis could not be done due to nonavailability of such facility. In the next twelve hours patient was kept paralysed with pancuronium (5mg bolus and 1 mg supplements SOS) and sedated with midazolam, 1mg IV every four hours. X-ray chest done in the ICU revealed extensive

bilateral nonhomogeneous opacities while X-ray cervical spine did not reveal any bony lesion. Fundoscopy revealed no abnormality. After institution of mechanical ventilation, pulse rate stabilised at 130-140/min, blood pressure varied from 110-130/60-70mm of Hg and SpO₂ ranged from 90-92%.

Patient was conscious the next morning and was responding to verbal commands, pulse rate was 140/min and blood pressure 130/80mm of Hg. PEEP was gradually increased to 15cm of H₂O and FiO₂ was simultaneously decreased to 0.6, maintaining SpO₂ between 91% and 96%. Throughout the day dopamine at the rate of 3-5µg/Kg/min had to be infused intermittently to maintain systolic blood pressure above 90mm of Hg. On the third day of hospitalisation patient's general condition remained same. She was conscious and tolerated endotracheal tube well, pulse rate varied between 120-136/min and blood pressure varied between 85-90/50-70mm of Hg. Bronchial breath sounds were auscultated bilaterally with no adventitious sounds, but chest X-ray did not reveal any improvement over previous radiological findings. Hemogram and all biochemical parameters were within normal limits and tracheal secretions were sent for culture (which did not grow any organism after 48 hours of incubation). Amikacin and ceftazidime were instituted empirically. Dopamine was continued. Attempts to decrease PEEP keeping FiO₂ less than 0.6 failed and ventilatory parameters were left unchanged with SpO₂ varying from 91%-96%. Patient was sedated with morphine 3 mg IV every four hours. On the fourth day of hospitalisation PEEP was reduced to 10 cm of H₂O and FiO₂ to 0.5. Dopamine was gradually withdrawn and blood pressure during the day ranged between 90-100/60-70 mm of Hg. On the fifth day of hospitalisation, patient was found to be conscious with pulse rate of 88/minute, blood pressure of 100/60mm of Hg and SpO₂ of 99%. Chest was clinically clear but X-ray chest showed only minimal improvement. Weaning from artificial ventilation was attempted using SIMV mode with decreasing rate of ventilation followed by a spontaneous breathing trial with T-tube for two hours, but patient failed to maintain adequate SpO₂ and hemodynamic stability and ventilatory support was reinstituted with PEEP of 4 cm of H₂O and FiO₂ of 0.4. On the sixth day of hospitalisation patient was successfully weaned off the ventilator. Post extubation pulse rate was 104/min, blood pressure was 100/67mm of Hg, SpO₂ 98% and respiratory

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rate 24/min. 2500ml of crystalloids were infused daily during the period on mechanical ventilation. Chest physiotherapy was instituted and patient was encouraged to cough out secretions, which were copious and blood tinged. Patient remained asymptomatic over the next three days but for hoarseness of voice, which improved gradually. X-ray chest done on the 10th day of hospitalisation was normal and she was discharged from the hospital the same day.

Discussion

POPE was first described in humans in 1973 [1]. There are two recognized forms of POPE [2]. Type I follows a sudden, severe episode of upper airway obstruction and can be seen in association with any cause of acute upper airway obstruction such as post-extubation laryngospasm, endotracheal tube occlusion, epiglottitis and croup and choking. Pulmonary oedema developing after non-lethal hanging has been reported very infrequently. One reason for this may be a poor survival rate amongst victims of suicidal hangings [3]. Survivors of suicidal hanging have been found to suffer morbidity due to upper airway obstruction caused by ligature strangulation [4]. Type II POPE develops after surgical relief of chronic upper airway obstruction and has been reported after tonsillectomy [2].

Anaesthesia related studies have found that incidence of POPE varies from 0.05%-0.1% of all anaesthetics [5]. In a series of 176 children with severe upper airway obstruction, Galvis AG reported an incidence of 9.6% for POPE I and 44% for POPE II [6].

The initiating event in POPE is the markedly negative intrapulmonary pressure generated by a forceful inspiratory effort against an obstructed extrathoracic airway and hence the synonym, "negative pressure pulmonary oedema". The importance of vigorous inspiratory efforts in the development of POPE I is supported by the apparent increase in susceptibility to this condition in young and healthy patients [7]. An exaggerated negative intrapleural pressure results in increased venous return to the right ventricle and increased pulmonary blood flow and elevated capillary hydrostatic pressure. These changes favour the transudation of fluid from the pulmonary capillaries into the interstitial and alveolar spaces. Some investigators have theorized that the pulmonary oedema associated with airway obstruction may be because of permeability defects in capillaries, a "capillary leak syndrome". This is supported by the finding of alveolar fluid containing high protein levels and inflammatory cells, usually not present in transudative pulmonary oedema [8]. Capillary and endothelial damage may result from shearing forces generated during forced inspiration against a closed airway. Also, hypoxia may be partly responsible for

pulmonary oedema. The genesis of POPE II is not clear.

Type I POPE generally occurs within 60 minutes of a precipitating event but the onset has been delayed for up to six hours in some case reports [9]. Patients with risk factors for upper airway obstruction, including those with difficult intubations, should be monitored closely for at least 60 min after extubation. The presence of agitation, tachypnea, tachycardia, frothy pink pulmonary secretions, rales and progressive oxygen desaturation suggest the diagnosis of POPE in the appropriate setting. Chest radiograph findings of pulmonary oedema support the diagnosis. A history of normal cardiac function and particularly the occurrence of such symptoms in a healthy young person, in the presence of upper airway obstruction, makes the diagnosis of POPE more likely. Treatment consists of supplemental oxygen and ventilatory support. Intubation and the application of low levels of PEEP have been employed in most reported cases. The role of diuretics in management of POPE is unclear. The prognosis for these patients is excellent and most patients respond rapidly to this management.

To conclude, upper airway obstruction is a recognised mechanism that can produce non-cardiogenic pulmonary oedema. Airway obstruction is the main cause of morbidity and subsequent mortality in the survivors of suicidal hangings. Pulmonary oedema may develop subsequently in such patients. In most instances, post-obstructive pulmonary oedema is a reversible process once recognized and properly treated.

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