Post Obstructive Pulmonary Edema Following Attempted Suicide by Hanging

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Introduction

Most victims of non-lethal (suicidal) hanging are young men and survivors are uncommon. Laryngo tracheal injuries and spinal injuries, although reported in 20-50% of post mortem examinations, are infrequent in survivors. Pulmonary complications are implicated in most in hospital deaths. Initial neurological presentation is of limited prognostic value; a poor initial presentation does not exclude a good recovery if managed in time [1].

We present a case of a suicidal hanging that presented with features of cerebral anoxia and developed non cardiogenic pulmonary edema. Early resuscitation and aggressive management led to complete recovery without any residual neurological deficit.

Case Report

A 26 year old male patient was brought to the emergency department of a peripheral hospital with history of hanging. The duration of hanging was unknown. At presentation, he was frothing from the mouth, audible stridor was present and he was struggling against restraints in a semi-conscious state. The heart rate was 46/min, feeble peripheral pulse volume, blood pressure was not recordable. The oxygen saturation (SpO_2) was 68%. The pupils were dilated and fixed bilaterally. On auscultation chest had conducted sounds. Glasgow Coma Scale was E1V2M5. Advanced cardiac life support (ACLS) was commenced in the form of injection atropine, injection adrenaline, followed by mechanical ventilation with 100% oxygen (Bag and mask ventilation). Intravenous line was secured and fluids administered. The patient was endotracheally intubated after initial resuscitation and put on synchronized intermittent mandatory ventilation (SIMV) mode with positive end expiratory pressure (PEEP) on ventilator. The tidal volume (TV) delivered was 8ml/kg body weight at respiratory rate (RR) of 15/min. Fraction of inspired oxygen (FiO_2) prescribed was 1. The vital parameters post ventilation were heart rate of 170/min, blood pressure 170/98 mm of Hg,

SpO_2 94% on FiO_2 at 1. Prophylactic parenteral antibiotics and cerebral decongestants (mannitol) were administered. All hematological and biochemical parameters were normal. The next day patient developed low grade fever and had crepitations in the lung fields. Pupils were mid dilated, not reacting to light. Intake output and SpO_2 (on FiO_2 0.8) were maintained at 88-90%. Chest radiograph revealed bilateral fluffy opacities (Fig. 1).

Arterial blood gas (ABG) analysis revealed partial pressure of arterial oxygen (PaO_2) 62% (on FiO_2 0.8). $\text{PaO}_2/\text{FiO}_2$ ratio was calculated to be 77.5 suggestive of acute respiratory distress syndrome (ARDS). The PEEP was increased (08 cm of H_2O), while TV was decreased to 5ml/kg body weight and RR was increased to 18/min. Patient was kept sedated and paralyzed. Daily monitoring of electrolytes and periodic ABG analysis was done.

On day three, his lungs had improved clinically as well as radiologically, pupils were normal in size and reaction. Patient was hemodynamically stable and maintained SpO_2 (FiO_2 0.6). Weaning was commenced and patient was administered reversal (neostigmine and glycopyrrolate). Patient regained good spontaneous respiratory effort. Trial of continuous positive airway pressure (CPAP) alternating with SIMV mode was commenced.

On account of patient being fully conscious, presence of good respiratory effort and patient tolerating CPAP well, patient was extubated. Re-intubation was not required as he maintained good saturation levels with oxygen by Flexi-Mask (SpO_2 86-92%). Chest radiograph demonstrated clear lung fields. Nebulisation and chest physiotherapy was continued. Thereafter he had unremarkable recovery with no apparent residual lung or brain damage.

Discussion

Pulmonary complications including acute lung injury (ALI), pulmonary edema, bronchopneumonia, ARDS and cerebral edema have been implicated in most hospital deaths [1,2]. The pulmonary edema may be from a neurogenic origin or secondary to negative intra-thoracic

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Fig. 1 : Chest radiograph (PA view) shows bilateral fluffy opacities

pressures generated as victim attempts inspiration through an obstructed airway. Negative pressure pulmonary edema (NPPE) has been described to be an imitator of ALI/ARDS and failure to consider NPPE in the differential diagnosis of acute clinical, physiological and radiographic changes that fit the criteria of ALI/ARDS may lead to unnecessary and potential deleterious complications. While early recognition and precipitating factors may herald NPPE or post obstructive pulmonary edema (POPE), an obvious underlying cause may be absent after emergence from unintentional upper airway obstruction or anesthesia [3]. Majority of POPE was recognized post operatively as a consequence of laryngospasm [4]. Neurogenic pulmonary edema results from a centrally mediated, massive, sympathetic discharge [5]. This discharge produces intense, generalized, but transient, vasoconstriction with a resultant shift of blood from systemic to pulmonary system. The pulmonary vasoconstriction also increases permeability by disrupting the permeability barrier. Neurogenic pulmonary edema is often recognized after lethal brain injury [5]. Fishman et al [6], has described two cases of ARDS following attempted suicide by hanging. Both had bilateral pulmonary edema and were managed with tracheal intubation and positive pressure ventilation. Both survived without any neurological deficits.

The original report of POPE included cases of upper airway obstruction due to tumor, strangulation and near hanging [7]. The initiating event is negative intra-pleural pressure generated by a forceful inspiratory effort (as high as -100 cm water) against an obstructed extra thoracic airway. The incidence of pulmonary edema associated with airway obstruction has been estimated as 11 % in adults requiring active airway intervention

for acute airway obstruction of varying etiology [6]. The hypoxia induced hyper adrenergic state causes translocation of blood from systemic to pulmonary circulation and an increase in both pulmonary vascular resistance and pulmonary capillary permeability [8].

Prognosis for patients with negative pressure pulmonary edema after strangulation injury is not as grave as that for patients with neurogenic pulmonary edema. The criteria for ARDS were met in this case but the rapid response and pre morbid condition may suggest POPE as the diagnosis.

Preventing re-obstruction and providing adequate ventilation and oxygen are mandatory after relief of upper airway obstruction. Treatments include supplemental oxygen and support cares, but positive end expiratory pressure and mechanical ventilation may be required for a prolonged period [9].

Recent data show that about 60% of patients recover. Thus, mortality rate has improved from 70% in past, to 40%. Better understanding of the disease process and protective ventilation therapy is responsible for an improved survival rate [10]. To conclude, the hopelessness of presenting features should not deter an energetic and practiced response to emergencies.

Conflicts of Interest

None identified

References

1. Kaki A, Edward T Crosby, Anne CP Lui. Airway and Respiratory management following non lethal hanging. *Can J Anaesth* 1997; 44: 445-50.
2. Jennifer A Davidson JA. Presentation of near hanging to an Emergency department in Northern Territory. *Emergency Medicine Australia* 2003; 15: 28-31.
3. Ackland GL, Mythen MG. Negative pressure pulmonary edema as an unsuspected imitator of acute lung injury/ARDS. *Chest* 2005; 127: 1867-8.
4. Alb M, Tsagogiorgas C, Menhardt JP. Negative pressure pulmonary edema(NPPE). *Anaesthesiol Intensivum Notfallmed Schmerzther* 2006; 4: 64-78.
5. Theodore J, Robin ED. Pathogenesis of neurogenic pulmonary edema. *Lancet* 1975; 2:745-51.
6. Fishman CM, Goldstein MS, Gardener LB. Suicidal Hanging, An association with Adult Respiratory Distress Syndrome. *Chest* 1977; 71: 225-7.
7. Timby J, Reed C, Zeilender S, Glauser FL. "Mechanical" causes of pulmonary edema. *Chest* 1990; 98: 973-99.
8. Lang SA, Duncan PG, Shephard DAE, Ha HC. Pulmonary edema associated with airway obstruction. *Can J Anaesth* 1990; 37: 210-8.
9. Dolinsky SY, MacGregor DA, Scudari PE. Pulmonary hemorrhage associated with negative pressure pulmonary edema. *Anesthesiology* 2000; 93: 888-90.
10. Amato MBP, Barbas CSV, Medeiros DM. Effect of a protective ventilation strategy on mortality in acute respiratory distress syndrome. *N Engl J Med* 1998; 338: 347-54.