Delayed pulmonary oedema following suicidal hanging

S Ahmad1, S Kamal2, K Jamal3
Senior Resident1, Department of Anaesthesiology and Critical Care, J N Medical College, Aligarh
Muslim University, Aligarh, Uttar Pradesh, India. Senior Resident2, Department of Anaesthesiology,
Paras Global Hospital Darbhanga, Bihar, India.

Corresponding author: kamalshadab@gmail.com

Suicidal hanging usually leads to death within a few minutes. The patient, if rescued, may develop respiratory distress, pulmonary oedema, convulsions, raised intracranial pressure and unconsciousness immediately after the incident. We report a case of suicidal hanging in a young female, brought to hospital in a conscious state with restlessness and anxiety. She developed pulmonary oedema after twelve hours, and was resuscitated successfully.

Keywords: Suicidal hanging; hypoxia; pulmonary oedema; acute respiratory distress syndrome; raised intracranial pressure

Introduction
The majority of victims of suicidal hanging are young and survivors are uncommon. Death usually occurs within few minutes of hanging. In survivors, respiratory and neurological complications develop immediately in most patients. Pulmonary complications are a cause of mortality in most hospital deaths. Pulmonary oedema, one of the most common complications occur in patients immediately following their rescue from acute airway obstruction or suicidal hanging.1

We present a case of suicidal hanging in which pulmonary oedema developed 12 hours after the incident. She presented in a conscious state with restlessness and features of cerebral anoxia and later developed non-cardiogenic pulmonary oedema. Early resuscitation and aggressive management led to complete recovery without any residual neurological deficit.

Case report
An 18 year old female was brought to the casualty department of our hospital with an alleged history of suicidal hanging by her cloth shawl (dupatta). The patient was rescued by her parents within a few minutes and brought to hospital within 20 minutes. On examination she was conscious, her pulse rate was 124/min and blood pressure was 110/60 mmHg. Pupils bilaterally reacted normally to light. On auscultation chest was bilaterally clear with adequate air entry, but patient was tachypnoeic with a respiratory rate of 30/min. Upper airway reflexes were present. Pulse-oximetry revealed SpO2 90% and arterial blood gas (ABG) analysis showed PaO2 80mmHg, PaCO2 29mmHg, pH 7.26, base excess (BE) 9.2mEq/L and SpO2 90% on Hudson mask with oxygen flow rate of 6L/min. Chest X-ray and CT scan head were normal and X-rays of cervical spine (AP and lateral view) revealed no bony injury. Conservative management done in ICU included propping up the patient and providing oxygen with Hudson mask with reservoir bag. After 12hrs patient developed respiratory distress with coughing frequently and gradually her SpO2 dropped to 74%. On auscultation bilateral crepitations were present in chest, Glasgow Coma Scale was E1V2M5, blood pressure was 82/54 mmHg and pulse rate was 132/min. Chest X-ray showed bilateral diffuse infiltrates consistent with pulmonary oedema.

Immediately, her trachea was intubated orally with 7.0mm ID cuffed endotracheal tube after intravenous ketamine 100mg and succinylcholine 75mg. Patient was attached to a ventilator with pressure controlled ventilation mode with respiratory rate of 12/min, inspiratory pressure 14cm of H2O, PEEP 8cm of H2O and FiO2 of 1.0 after administration of vecuronium bromide 4mg and dexamethasone 8mg intravenously. The vital parameters post ventilation were pulse rate of 120/min, blood pressure 124/80mmHg, SpO2 94% on FiO2 of 1. Prophylactic antibiotics were administered.

© 2017. Ahmad et al. This is an Open Access article distributed under the terms of the Creative Commons Attribution License (http://creativecommons.org/licenses/by/4.0), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly credited.
After one hour of therapy, SpO₂ further dropped to 70% and patient developed frank pulmonary oedema with extensive fine crepitations all over the chest and pink frothy secretions appeared in the breathing system. Controlled ventilation was continued with PEEP of 10cmH₂O. She was given furosemide 40mg intravenously, repeated 8 hourly. After 2 hours patient’s ABG revealed PaO₂ 90 mmHg, PaCO₂ 32mmHg, SpO₂ 96% on FiO₂ at 1. Her chest condition markedly improved after 14hrs of ventilation. ABG analysis showed PaO₂ 110mmHg, PaCO₂ 38mmHg, pH 7.45, SpO₂ 98% on FiO₂ at 0.5. She regained consciousness, and was put on synchronized intermittent mandatory ventilation (SIMV) mode with FiO₂ at 0.4 for the next 12 hrs. After 24 hours (2nd day) she was fully conscious. Her SpO₂ was 99% on FiO₂ at 0.4. Chest X-ray AP view was normal. ABG analysis revealed PaO₂ 112mmHg, PaCO₂ 40mmHg, pH 7.40 and SpO₂ 99% on FiO₂ of 0.4. As her condition had improved, she was extubated. Post extubation her SpO₂ was 97% on FiO₂ of 0.4 by facemask. Chest radiograph demonstrated clear lung fields. Nebulisation and chest physiotherapy was continued. Thereafter she made an unremarkable recovery with no apparent residual lung or brain damage.

**Discussion**

The incidence of hanging in India is approximately 25% of total cases of suicide.³ Pulmonary complications like pulmonary oedema, bronchopneumonia, acute respiratory distress syndrome (ARDS) and cerebral oedema have been reported in most hospital deaths.³ The pulmonary oedema may be of neurogenic origin or secondary to negative intra-thoracic pressures generated as victim attempts inspiration through an obstructed airway. Negative pressure pulmonary oedema (NPPE) has been described to be a cause of ARDS and failure to consider NPPE in the differential diagnosis of ARDS may lead to unnecessary and potential lethal complications. While early recognition may herald NPPE or post obstructive pulmonary oedema (POPE), an obvious underlying cause may be absent after emergence from unintentional upper airway obstruction.³ Neurogenic pulmonary oedema results from a centrally mediated, massive, sympathetic discharge which produces intense, generalized, but transient vasoconstriction with a resultant shift of blood from systemic to pulmonary system.³ The pulmonary vasoconstriction also increases permeability by disrupting the permeability barrier.

The incidence of pulmonary oedema associated with airway obstruction has been estimated as 11% in adults requiring active airway intervention for acute airway obstruction of varying aetiology.³ 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.⁷

The strategy of management should target to get adequate oxygenation and cerebral perfusion.¹ In the presence of respiratory distress (hypoxia) with or without pulmonary oedema tracheal intubation and mechanical ventilation are indicated. PEEP (positive end expiratory pressure) has its definite role in the treatment of pulmonary oedema, but at the same time it also lead to increased intracranial pressure, so one has to weigh its use according to the patient’s condition.⁸

Re-establishment of a patent airway may not provide the proper oxygenation even with a clear chest as occurred in our patient. Such patients may require aggressive oxygen therapy for adequate oxygenation. Pulmonary oedema is reversible in most of the cases once recognized and treated.

To conclude, patients rescued from suicidal hanging or strangulation even with a clear chest must be treated with aggressive oxygen therapy and monitored closely in ICU as they may develop pulmonary oedema even after 12 hours following the event.

**References**


