Review Article

Insights into pathophysiology, management, and outcomes of near-hanging patients: A narrative review

Balaji Kannamani, Neeru Sahni[1], Anjishnujit Bandyopadhyay[2], Vikas Saini[1], Laxmi Narayana Yaddanapudi[1]

Department of Critical Care Medicine, Manipal Hospital, Dwarka, New Delhi, [1]Department of Anaesthesia and Intensive Care, Post Graduate Institute of Medical Education and Research, Chandigarh, [2]Department of Anaesthesiology, Pain Medicine and Critical Care, Jai Prakash Narayan Apex Trauma Center, All India Institute of Medical Sciences, New Delhi, India

Abstract

Hanging is suspension of a person by noose or ligature around the neck. Hanging has been used as a judicial execution method since medieval times and is still a common method of suicide in developing countries. The term "near hanging" is used for those who survive long enough to reach the hospital, and these patients still hold mortality as high as 45%. These patients can present with a wide array of complaints involving cardiovascular, neurologic, and pulmonary systems and local injuries to the airway and vascular structures. High serum lactate and cardiac arrest at arrival are some of the prognostic factors which portend a poor prognosis. Early admission and aggressive treatment are essential to provide best neurologic outcome. Due to paucity of definitive guidelines, therapy has to be based on clinical reports and expertise of the treating physician. Hence, near-hanging patients pose a great challenge in primary emergency care and intensive care management. This review aims to outline the pathophysiology, prognostic indicators, and recent evidence in the management of near-hanging patients.

Keywords: Aspiration pneumonia, hypoxic encephalopathy, near hanging, partial hanging

Introduction

Hanging is a commonly adopted mode of committing suicide worldwide.[1] Approximately 8 lakh people die every year due to suicide, with most of them occurring in low- and middle-income countries. The term "near hanging" is used for those victims who survive long enough to reach the hospital. Near-hanging victims present to the emergency department (ED) in dire need of specialized intensive care. They present with a wide gamut of clinical manifestations ranging from patients who are completely stable to those in shock; patients may present with hypoxic brain injury and/or respiratory failure.[2] Though the literature on postmortem findings in hanging patients is extensive, there is paucity of data on the clinical presentation, findings on neurologic imaging, as well as any guideline suggesting management and prognostication. When a victim is fully suspended, it is termed as complete hanging, and the term "incomplete" or "partial" hanging is used when some part of the body is in contact with the ground or any other surface beneath the victim.[3] External pressure on the neck, which is aggravated by suspension of the victim's own body weight, is the mechanism behind hanging.

Address for correspondence: Dr. Anjishnujit Bandyopadhyay, Department of Anaesthesiology, Pain Medicine and Critical Care, Jai Prakash Narayan Apex Trauma Center, All India Institute of Medical Sciences, New Delhi, India.
E-mail: docbando@gmail.com

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Pathophysiology

The axial traction generated from hanging leading to external pressure on the neck is the pathological basis of injury. The presence of a multitude of vital structures in an unprotected and confined area makes the anterior neck region extremely vulnerable to injury. Cervical spine is more mobile and less stable than thoracic and lumbar vertebrae, hence the spinal cord is more prone to injury at this level.

The major vessels present in the neck, the carotid body, the laryngeal cartilages, and the cervical spine are the main structures at risk. Early mortality is mainly due to secondary insults to the brain, whereas delayed mortality can be attributed to lung complications [Figure 1].

Injury to vascular structures

Jugular veins are very superficial and partially covered by sternocleidomastoid muscle, hence are prone to compression. Venous obstruction results in flow stagnation, leading to cerebral hypoxia, which may be the initial event in hanging. Loss of consciousness can occur as early as within 15 s after hanging; this is unlikely to be due to venous compression alone. Arterial spasm due to high external pressure has been noted to be a significant contributing factor. The weight needed to occlude carotid is only 3.5 kg (for jugular vein, it is only 2 kg), which is easily exceeded even in partial hanging.[4,5] Subsequently, hanging results in traction on the vessels, producing intimal tears resulting in dissection, thrombosis, and subsequent obstruction. Basal subarachnoid hemorrhage has also been reported as a result of these intimal tears.[6] Sudden cardiac arrest (CA) can also occur due to reflex bradycardia as a result of compression of carotid bodies.[6] Although the vertebral arteries are well protected throughout their course as they traverse through the transverse foramina, obstruction of these arteries can occur even if there is no cervical spine injury.[3]

Airway injury

Though not present universally, hanging often causes fractures of the bony and cartilaginous framework of the neck, leading to laryngotracheal disruption. Narrow ligatures more commonly lead to airway injuries compared to broad ones, with thyroid cartilage being the most commonly fractured structure. Significant soft-tissue injury to the larynx occurs inconsistently, both with and without fracture.[7] Laryngotracheal trauma can lead to complete disruption of the airway and subcutaneous emphysema. Hematomas/swelling in retropharyngeal, pharyngeal, and paratracheal spaces have also been reported as a late presentation, resulting in airway compromise and hypoxia.[8]

Cervical spine injury

Cervical spine injuries are common in judicial hangings. The length of drop is equivalent to the victim's height and the suspension is complete. In partial hanging, the suspension is incomplete, and hence, the axial traction component is very less. This explains the less than 1% incidence of cervical spine injury in near-hanging cases in the literature.[9]

Pulmonary complications

Pulmonary edema, acute respiratory distress syndrome (ARDS), and aspiration pneumonia are the commonly occurring pulmonary complications. Pulmonary edema after relief of acute airway obstruction has an incidence of approximately 11%.[10] The trachea of an adult is easily compressed by a 15 kg weight and this is easily exceeded in

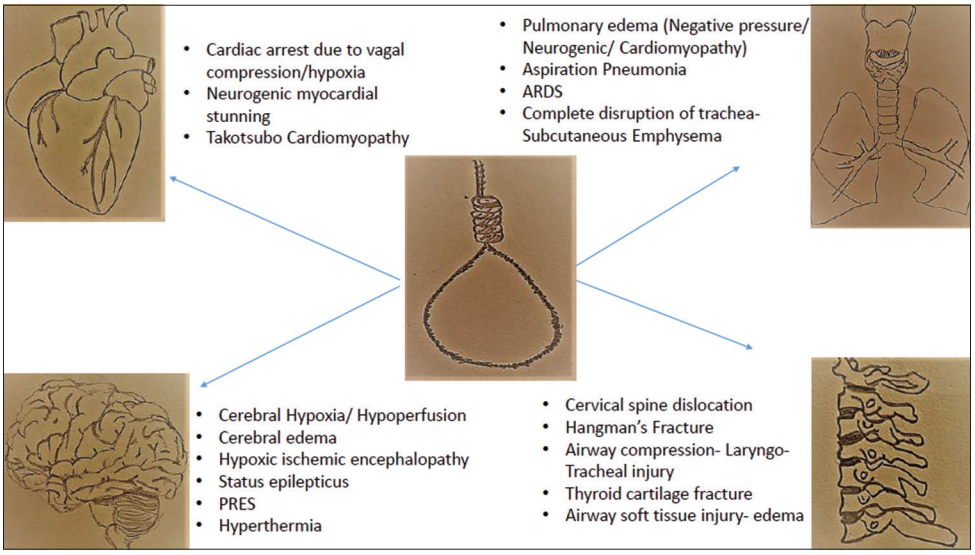

Figure 1: Pathophysiological effects of hanging

near-hanging cases.[3] As a result of generation of excessive negative intrathoracic pressure (−100 cm of H_2O) during airway obstruction, pulmonary hyperemia and, subsequently, edema develop. Cerebral hypoxia leads to release of vasoactive substances like histamine, serotonin, and kinins, causing neurogenic pulmonary edema.[10]

Aspiration of gastric contents is a usual occurrence with loss of consciousness, and this might lead to aspiration pneumonia and, subsequently, ARDS in partial hanging.

Neurologic complications

Neurologic outcomes in hanging vary from death, permanent hypoxic brain damage to complete recovery. Uncommon features include posterior reversible encephalopathy syndrome (PRES), amnesia, Korsakoff's psychosis, and hyperthermia.[11] Cerebral edema is common after partial hanging, due to venous compression leading to venous stagnation in the brain. Luxuriant cerebral perfusion of an already hypoxic brain following relief of obstruction may also contribute to cerebral edema. Poor Glasgow Coma Scale (GCS) score at arrival to emergency usually signifies a poorer outcome. Hypoxic ischemic encephalopathy (HIE) is a dreaded but delayed manifestation of being hung for a long time. Incidence of anoxic encephalopathy is 2.0% in previous studies.[12] Functional recovery is very poor in such patients and there is significant morbidity. There is a lot of variation in the sensitivity of various brain structures to hypoxia, hence cerebral hypoxic injury is unevenly distributed.

Cardiac complications

Up to 50% patients with near hanging present to the hospital with CA as reported by de Charentenay *et al.*[12] in their retrospective study. It can occur immediately due to compression of the vagus nerve, hypoxia, or neurogenic myocardial stunning. Takotsubo cardiomyopathy, which is a transient apical ballooning, has also been reported in the literature in a few case reports.[13,14]

Predicting outcomes in near-hanging patients

Poor GCS at admission to the hospital and intensive care unit (ICU) has been consistently reported to be associated with poor outcomes across multiple studies conducted on a diverse study population of both adults and children.[15-18] High Acute Physiology and Chronic Health Evaluation (APACHE II) score at admission, low arterial pH at admission, hanging-induced CA, hyperglycemia, and hyperlactatemia have also been reported to be poor prognostic markers that are associated with mortality rate as high as 45% in various studies.[12,15,19] More recently, a retrospective study by Ramadoss *et al.*[20] observed GCS <8, need for ICU stay, CA due to hanging, aspiration pneumonia, hypotension, and cerebral edema to be independent predictors of mortality [Table 1].

Management

Relief of suspension and removal of the ligature should be done on immediate basis at the scene of hanging. As no specific guidelines regarding management of near-hanging exist, clinicians should adhere to the Advanced Trauma Life Support (ATLS) guidelines.[21] Clinicians should focus on airway, breathing, and circulation as per ATLS guidelines. Victims with shorter suspension time and lesser degree of hypoxia may rapidly resume adequate spontaneous respiration and regain consciousness. If the patient does not regain consciousness, endotracheal intubation followed by mechanical ventilation is warranted to prevent aspiration and hypoxia which can further worsen the clinical condition. Rapid sequence induction of anesthesia with in-line immobilization of the cervical spine should be performed during intubation (generally orotracheal) to avoid excessive motion of the head and neck.

Despite the low incidence of cervical spine injury, among patients who have not sustained a significant drop, it may be prudent to treat all near-hanging victims by assuming that they have an unstable cervical spine until proven otherwise. Nonavailability of information about the risk factors and preexisting spine disorders also lends weight to this approach. A noncontrast computed tomography scanning of the neck can rule out tracheal and C-spine injuries.[22] Until then, a hard collar can be used to stabilize the spine, along with using log roll technique for patient mobilization.

Delayed respiratory impairment results from soft tissue swelling causing airway compromise. Although the risk is small, all spontaneous breathing should be monitored for at least 24 h.

After primary screening and initial hemodynamic resuscitation, patients with need of mechanical ventilation, invasive hemodynamic monitoring/support, and post-CA care are generally managed in ICU [Table 2]. Tharmarajah *et al.*,[23] in their prospective cohort study, reported that with early intubation, targeted temperature management (TTM), and strict blood pressure (BP) control, decreased mortality and increased survival with good neurologic outcome can be expected. In the absence of definitive guidelines catering specifically to near-hanging patients, we have collated pertinent information regarding guideline-directed management of the various clinical features these patients might present with and have outlined them under the following headings:

Table 1: Prognostic markers for unfavorable outcome in partial hanging

1.	Time from hanging discovery to unhanging more than 5 min
2.	Lactate >3.5 mmol/l at ICU admission
3.	Hyperglycemia (RBS -Random blood sugar >140 mg/dl) at admission
4.	Hanging-induced cardiac arrest
5.	Status epilepticus
6.	Total number of organ failures >2 at day 1
7.	Fever at the time of admission
8.	Hypotension on admission
9.	Presence of cerebral edema and aspiration pneumonia
10.	Need for intensive care stay
11.	Poor GCS at admission[a]

[a]GCS at admission poorly correlates with neurologic recovery in near hanging. GCS=Glasgow Coma Scale, ICU=Intensive care unit

Table 2: Summary of management of near hanging

Emergency management	ICU management
1. Initial resuscitation and screening	1. Maintain oxygenation and ventilation (suspect aspiration pneumonia, pulmonary edema in case of poor Partial pressure of oxygen/fraction of inspired oxygen p/f ratio)
2. Maintain airway patency and cervical spine stabilization	2. Post-cardiac arrest care: Consider TTM in case of poor GCS after ROSC
3. Airway protection-endotracheal intubation if GCS <8, status epilepticus	3. No role of prophylactic antiepileptics
4. Maintain cerebral perfusion by maintaining MAP >60 mmHg	4. No role of empirical antibiotics
5. Start cerebral edema measures if CT reveals evidence of cerebral edema	5. Anti-cerebral edema measures • Head-end elevation • Sedation • Maintain normocapnia ($PaCO_2$ 30–35 mmHg) • Osmotic diuretics (3% NaCl, mannitol)
	6. General supportive care including deep vein thrombosis prevention, enteral feeding, maintenance of euglycemia, stress ulcer prophylaxis if indicated, and physiotherapy, both respiratory and limb

CT=Computed tomography, GCS=Glasgow Coma Scale, ICU=Intensive care unit, MAP=Mean arterial pressure, ROSC=Restoration of spontaneous circulation, TTM=Targeted temperature management

1. Management of neurologic complications

a. Cerebral edema: Cerebral edema is an accumulation of excessive fluid within either brain cells or extracellular spaces.[24] The identification and treatment of cerebral edema is central to the management of critical intracranial pathologies. Measurement of cerebral edema relies on surrogate markers like imaging studies with evidence of tissue shifts or structural changes or is performed via devices which monitor the intracranial pressure (ICP). General recommendations for the management of cerebral edema include nursing care in a 30°–45° head-up position, with prevention of seizures and agitation and maintenance of normal BP and $PaO_2/PaCO_2$ (arterial partial pressure of oxygen and carbon dioxide). Patient usually needs short-acting sedative infusion like dexmedetomidine, fentanyl, or propofol. Usually, hypertonic saline or mannitol is also prescribed to reduce brain swelling, but these agents might worsen pulmonary edema temporarily.

b. Seizures: There is no evidence to suggest that use of prophylactic antiepileptics may improve outcomes in patients with neurologic insult post-near hanging. But aggressive control of seizure, once it occurs, is recommended to prevent secondary insult.

c. HIE: The development of HIE leads to severe morbidities in the patient and is a leading cause of mortality. It is usually diagnosed on the basis of imaging modalities like magnetic resonance imaging (MRI) brain, with features like altered signal intensities in the basal ganglia, thalamus, hippocampus, and cerebellar hemispheres (metabolically most active) in diffusion-weighted imaging.[25] No specific therapy has been found to be beneficial, and supportive care is the cornerstone. HIE carries a poor prognosis with patients usually not regaining full consciousness and leading to a poor quality of bedridden life dependent on caretakers.[26]

2. Pulmonary complications

a. Pulmonary edema: In near-hanging patients, the cause of pulmonary edema is usually postobstructive due to generation of excessive negative pressures. It is non-neurogenic and noncardiogenic. Usually, such patients are obtunded with poor GCS, hence invasive mechanical ventilation with positive end expiratory pressure (PEEP) is warranted. Lung protective ventilation strategies, which include low tidal volume ventilation, adequate PEEP, and adjustment of minute ventilation to keep the pH above 7.25, should be routinely practiced.[27] Conservative fluid management, minimal sedation to prevent agitation and bucking on the endotracheal tube, and judicious use of diuretics if BP is within normal limits are recommended.[28]

b. Aspiration pneumonitis: Due to the usual obtunded status of these patients, delayed securing of the airway may result in aspiration pneumonitis. This condition usually results in hypoxemia, fever, tachycardia, and chest radiographic anomalies. Persistence of radiographic infiltrates and fever beyond 48 h warrants a course of antibiotics.[29]

3. Supportive care: Early enteral feeding, thromboprophylaxis (both mechanical and pharmacologic), strict glycemic control (140–180 mg/dl), and use of judicious sedation with daily assessment for readiness for extubation and removal of vascular devices should be undertaken as usual.[30] Prophylactic antimicrobials are not of any help, but high index of suspicion against development of hospital-acquired infections and aspiration pneumonia is to be kept. Daily SOFA (Sequential Organ Failure Assessment) score and temperature monitoring is also important.[30]

4. Psychiatric evaluation: As suicide attempts account for most cases of near hanging, once the patient becomes conscious, a thorough psychiatric evaluation is a must.[31] Assessment of mental health status through patient and family interviews should begin as soon as the patient becomes communicable. Multidisciplinary rehabilitation and discharge plan needs to be created to not only treat existing mental health issues, but also prevent and treat post-intensive care syndrome. Psychiatric evaluation and support to the patients and their relatives is the key to preventing such attempts in future.
5. Post-CA care: Post-CA syndrome is a constellation of myocardial dysfunction, anoxic brain injury, and ischemia–reperfusion injury and the underlying cause of CA.[32] Patients who continue to be comatose after achieving restoration of spontaneous circulation (ROSC) should be treated with TTM at 32°C–36°C for at least 24 h.[33] Contrary to the current American College of Cardiology/American Heart Association (ACC/AHA) recommendations, Hsu *et al*.[34] reported in their multicenter retrospective trial that TTM CA patients had worse unadjusted survival and neurologic outcome than non-TTM patients. These findings may be explained by the higher severity of illness in the TTM group, variable TTM implementation, and differences in post-CA management. More recently, a multicenter randomized controlled trial (RCT) involving out-of-hospital CA patients failed to demonstrate any benefit of hypothermia over normothermia in terms of mortality and neurologic outcome. Moreover, in the hypothermia group, hemodynamic instability and arrhythmias were more common. In light of the findings of this trial, it might be more prudent to maintain normothermia with active prevention of fever, rather than targeting hypothermia.[35]

Along with TTM, strict maintenance of mean arterial pressure (MAP) >65 mmHg, SpO_2 (Oxygen saturation) between 92% and 98%, and end-tidal CO_2 between 35 and 45 mmHg is a must as per the latest ACC/AHA guidelines.[33] Neuro-prognostication should be multimodal and must not be attempted before 72 hours of normothermia. If following resuscitation, TTM, rewarming, and after stopping all sedative drugs, the patient remains comatose at 72 h of normothermia, presence of at least two of the following indicates high likelihood of poor outcome: (a) absent pupillary or corneal reflexes, (b) bilaterally absent N20 somatosensory evoked potential wave, (c) highly malignant electroencephalogram, (d) myoclonus, (e) neuron-specific enolase >60 μg/l at 48 and 72 h, and (f) evidence of diffuse and extensive anoxic injury on brain computed tomography/MRI.[32]

Conclusion

Near-hanging victims suffer from a wide array of clinical features almost always requiring care in ICU. Early recognition of organ failure and aggressive resuscitation can lead to successful management of these cases in terms of reduced mortality and good neurologic outcome. Early psychiatric evaluation and therapy to both patients and their families may help reduce further suicidal attempts.

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Conflicts of interest
There are no conflicts of interest.

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