PSUMJ
PSU MEDICAL JOURNAL

Short Report

Near Hanging Induced Negative Pressure Pulmonary Edema

Pornchanan Wuttisakdanon, M.D.[1], Tattharit Sakdaauscoskul, M.D.[1], Anon Saelim, M.D.[1], Jittima Phativitayaporn, M.D.[1], Osaree Akaraborworn, M.D., Ph.D.[2]

[1]Department of Surgery, Faculty of Medicine, Prince of Songkla University, Hat Yai, Songkhla 90110, Thailand.
[2]Division of Trauma and Critical Care, Department of Surgery, Faculty of Medicine, Prince of Songkla University, Hat Yai, Songkhla 90110, Thailand.

ABSTRACT

Negative pressure pulmonary edema (NPPE) is a rare condition with a high mortality rate. This condition causes acute respiratory failure occurring when sudden, forceful inspiration is against an obstructed airway. Near-hanging is referred to patients having survived a hanging injury. During hanging, the entire neck structure is compressed. This causes sudden airway obstruction, and may develop into acute respiratory failure from suddenly increase intrathoracic pressure. To our knowledge there have not often been reported incidences of negative pressure pulmonary edema after a near hanging condition. Herein, we report a case of negative pressure pulmonary edema after hanging condition.

Keywords: hanging; near hanging; NPPE; post obstructive pulmonary edema

INTRODUCTION

Negative pressure pulmonary edema (NPPE)[1], also known as post obstructive pulmonary edema, is uncommon; however it is a life-threatening condition. The estimated incidence rate of NPPE is between 0.05-0.1%[2]. The main cause of NPPE is post extubating laryngospasm or acute upper airway obstruction[3]. The Most common clinical presentations include: breathing difficulties, tachypnea, hypoxemia and pink-frothy sputum. A Patient with a condition of acute airway obstruction causing negative pressure will result in increased venous return to the right side of the heart, which subsequently increases pulmonary pressure[4]. Intravascular fluid shifts lead to interstitial and pulmonary edema, resulting in the patients entering acute respiratory distress syndrome (ARDS).

The impact of near hanging compresses the entire neck structure; including the esophagus, trachea, internal jugular vein and carotid artery. When the trachea

Corresponding author: Osaree Akaraborworn, M.D., Ph.D.
Division of Trauma and Critical Care, Department of Surgery, Faculty of Medicine, Prince of Songkla University, Hat Yai, Songkhla 90110, Thailand.
E-mail: aosaree@gmail.com
doi: 10.31584/psumj.2025273421
https://he01.tci-thaijo.org/index.php/PSUMJ/

PSU Med J 2025;5(2):75-79
Received 28 August 2024
Revised 9 March 2025
Accepted 10 March 2025
Published 22 August 2025

is compressed and the upper airway is blocked, forceful inspiratory efforts against the obstruction create negative intrathoracic pressure. This elevated pressure in the pulmonary veins causes fluid leakage from the capillaries into the lungs, leading to negative pressure pulmonary edema (NPPE). However, NPPE resulting from hanging is uncommon.

The following case describes the clinical presentation, point of concern and management strategies.

SHORT REPORT

A 34-year-old female was brought to the emergency department by her relative. The relative informed that the patient had attempted suicide by hanging, 30 minutes prior to arriving at the hospital.

During the Initial assessment at the emergency department, the patient could not talk due to being unconsciousness with a Glasgow Coma Scale of 4 (E1V1M2). Upper airway obstruction was not suspected, as the patient was spontaneously breathing, had 100% Oxygen saturation during on room air and showed no stridor. Breath sounds were equal in both lungs, without adventitious sound nor subcutaneous emphysema. No external wounds were detected: other vital signs were stable. Due to an altered level of consciousness, the patient was intubated: initial chest radiography is shown in Figure 1 as unremarkable. Brain and neck computed tomography, with angiography, showed patent cerebrovascular structures without signs of hypoxic encephalopathy. The patient was then admitted for neurological and respiratory observation.

Ten hours after the accident, the patient developed dyspnea, agitation and exhibited marked, pink, frothy sputum production; requiring frequent clearance every 15-30 minutes. The ventilator settings included pressure-controlled ventilation, with a rate of 16 breaths per minute, an inspiratory pressure of 16 cmH_2O, PEEP of 5 cmH_2O, and FiO_2 of 0.4.

Suddenly, Oxygen saturation decreased to 85-88%, and arterial blood gas (ABG) analysis showed a pH of 7.35, pCO_2 of 26.8 mmHg, pO_2 of 61.4 mmHg, HCO_3 of 16 mEq/L, and lactate of 2.6 mmol/L. Fentanyl was administered as a continuous drip for pain and agitation control. After sedation, the patient developed hypotension, prompting fluid resuscitation with an acetate Ringer's solution. During this time, the patient also presented with a high-grade fever (body temperature 40 °C) and tachycardia (heart rate 130-140 bpm). Consequently, intravenous antibiotics were initiated as empirical treatment for aspiration pneumonia. Despite receiving 2,000 mL of fluid, the patient rexperienced hypotensive, necessitating vasopressor infusion.

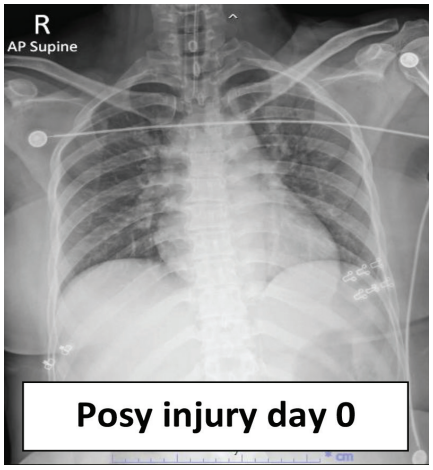

Figure 1 Chest X-ray with unremarkable findings

Sixteen hours after the accident, the patient had progressive hypoxia, with increased production of copious and loose sputum. Chest auscultation revealed bilateral crepitations. A repeat chest radiograph demonstrated bilateral, generalized, heterogeneous hyperdensities involving both lungs (Figure 2). The fluid balance review indicated a positive fluid status of 3 liters. Arterial blood gas (ABG) analysis on 100% FiO_2 revealed severe hypoxemia, with a PF ratio of 65 (pH 7.34, pCO_2 24 mmHg, pO_2 65 mmHg). Ventilator settings were adjusted to pressure-controlled ventilation, inspiratory pressure of 24 cmH_2O, PEEP of 12 cmH_2O, and FiO_2 of 1.0.

Bedside echocardiography demonstrated preserved left ventricular ejection fraction (LVEF) by the eyeball. From diffuse bilateral infiltrates on imaging and a PF ratio of 65, a diagnosis of severe acute respiratory distress syndrome (ARDS) was established. Sputum culture identified *Haemophilus haemolyticus*, though it was deemed an unlikely causative pathogen for severe pneumonia due to sudden onset of dyspnea. Central venous pressure (CVP) monitoring was not performed as we did not insert a central venous catheter.The patient was placed under deep sedation and neuromuscular relaxation using morphine, midazolam, and cisatracurium. A lung-protective ventilation strategy was applied.

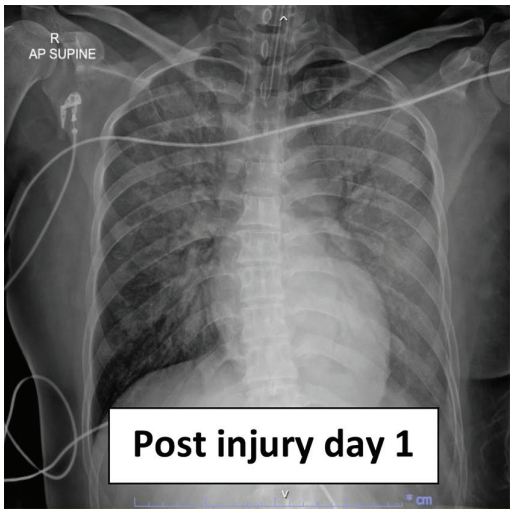

Figure 2 Chest X-ray with perihilar infiltration

48 hours after the injury and 28 hours after the lung protective ventilator strategy was applied oxygenation was improved. The ventilator setting was decreased to an inspiratory pressure of 16 cmH_2O, PEEP 10 cmH_2O, FiO_2 0.5, with ABG showing a PF ratio of 290 (pH 7.4, pCO_2 42 mmHg, pO_2 145 mmHg).

Chest radiography showed rapid improvement. The chest radiography is shown in Figure 3. The muscle relaxant was discontinued at 72 hr. and weaning started at 96 hr.

After 5 days of intensive care, the chest radiography returned to normal without infiltration. The patient was successfully liberated from the mechanical ventilation. Regained full consciousness and was discharged home with a Glasgow Outcome Scale (GOS) of 5/5.

DISCUSSION

Negative pressure pulmonary edema was first described in 1973[5], and is classified into two types. Type 1 usually occurs in acute upper airway obstruction, commonly due to biting of the endotracheal tube or post-extubated laryngospasm. Type II occurs after the relief of partial chronic airway obstruction; such as a resection of laryngeal tumor, adenoids, tonsils, or intrathoracic goiters[6]. Type 1 of NPPE can be found in strangulation or near hanging, typically developing immediately after rescue from upper airway obstruction. In our case, the patient presented with a delayed onset of type 1 NPPE. Previous literature reports associated morbidity and mortality rates as high as 40%[7-10]. Within 24 hours of a near-fatal hanging, our patient developed severe agitation without intracranial injury, paradoxical ventilation, and pink frothy sputum. Serial chest radiographs showed pulmonary edema, consistent with NPPE[11].

NPPE is caused by multifactorial components. It mainly occurs due to sudden, forceful inspiration against an obstructed airway, resulting in the generation of negative transpulmonary pressure. This leads to a marked increased in blood flow to the pulmonary system. Subsequently, the elevated hydrostatic pressure causes bilateral and diffused pulmonary edema. Additionally, neurogenically mediated sympathetic surge and systemic vasoconstriction shunt blood flow to the lower resistance pulmonary circulation, resulting in accumulated hydrostatic pressure. In the hypoxic state, pulmonary permeability also increases, aggravating the pulmonary edema. This pathophysiology explains the hypotensive state in this patient, due to marked depletion of systemic volume, shunting to the pulmonary circulation, and the edematous state of the pulmonary system[12].

These conditions can be managed by adjusting ventilatory support, based on the severity of acute respiratory distress syndrome (ARDS), providing hemodynamic support through intravenous fluid resuscitation during the fluid shifting phase, and administering vasopressors guided by fluid responsiveness strategies. Following stabilization, intensive monitoring is required during the self-recovery phase (24–48 hours post-injury) to minimize secondary brain insult due to hypoxia, a common occurrence in patients with near-hanging injuries following suicide attempts.

CONCLUSION

Negative pressure pulmonary edema is a rare condition developing in patients after airway obstruction, which results in high morbidity and mortality if not promptly recognized and immediately properly managed.

Human ethics

Consent was obtained or waived by the patient in this study.

CONFLICT OF INTEREST

The authors have declared that no competing interests exist.

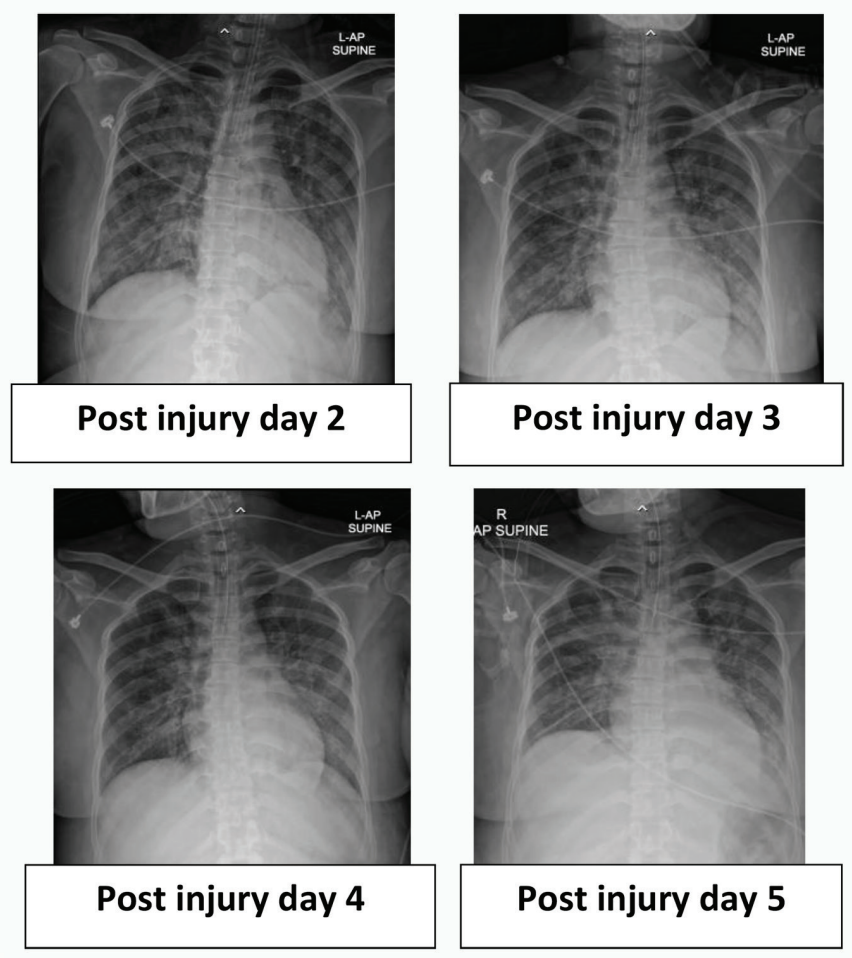

Figure 3 Perihilar infiltration, which resolved rapidly within a few days

REFERENCES

1. Bhattacharya M, Kallet RH, Ware LB, Matthay MA. Negative-pressure pulmonary edema. Chest 2016;150:927–33.
2. Liu R, Wang J, Zhao G, Su Z. Negative pressure pulmonary edema after general anesthesia: a case report and literature review. Medicine 2019;98:e1538. doi: 10.1097/MD.0000000000015389.
3. Lemyze M, Mallat J. Understanding negative pressure pulmonary edema. Intensive Care Med 2014;40:1140–3. doi: 10.1007/s00134-014-3307-7.
4. Ma J, Liu T, Wang Q, Xia X, Guo Z, Feng Q, et al. Negative pressure pulmonary edema (Review). Exp Ther Med 2023;26:1–7. doi: 10.3892/etm.2023.12154.
5. Capitanio MA, Kirkpatrick JA. Obstructions of the upper airway in children as reflected on the chest radiograph. Radiology 1973;107:159–61.
6. Udeshi A, Cantie SM, Pierre E. Postobstructive pulmonary edema. J Crit Care 2010;25:508.e1–5.
7. Scharf SM, Brown R, Tow DE, Parisi AF. Cardiac effects of increased lung volume and decreased pleural pressure in man. J Appl Physiol 1979;47:257–62.
8. Oswalt CE, Gates GA, Holmstrom MG. Pulmonary edema as a complication of acute airway obstruction. JAMA 1977;238:1833–5.
9. Tami TA, Chu F, Wildes TO, Kaplan M. Pulmonary edema and acute upper airway obstruction. The Laryngoscope 1986;96:506–9.
10. Lang SA, Duncan PG, Shephard DA, Ha HC. Pulmonary oedema associated with airway obstruction. Can J Anaesth J Can Anesth 1990;37:210–8.
11. Berdai AM, Labib S, Harandou M. Postobstructive pulmonary edema following accidental near-hanging. Am J Case Rep 2013;14:350–3. doi: 10.12659/AJCR.889415.
12. Mantha S, Rao SM. Noncardiogenic pulmonary oedema after attempted suicide by hanging. Anaesthesia 1990;45:993–4.