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Case report

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Acute pulmonary edema after hanging attempt.

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Abstract

Hanging is commonly used to commit suicide, but in case the person is rescued before death happens, multiple complications may occur, such as negative pressure pulmonary edema (NPPE). We report the case of a 15 years old child who was admitted in our intensive care unit after attempting suicide by hanging. She was put on mechanical ventilation with a positive outcome and discharged seven days later. This case highlights a rare cause of NPPE which can evolve favorably if known and precariously treated.

Keywords: pulmonary edema, hanging, cardiac complication.

Introduction

Post hanging pulmonary edema is a Post Obstructive Pulmonary Edema (POPE), which corresponds to a rare form of non-cardiogenic pulmonary edema; it is also known as negative pressure pulmonary edema (NPPE). It is due to an alveolar fluid exudation, that happens after a brutal intra-thoracic depression due to a relief of upper airways obstruction [1]. It is a life-threatening complication that can be secondary to postoperative recurrent paralysis, laryngospasm, obstructing goiter, sleeping obstructive apnea or following suicidal or accidental near hanging or strangulation [2]. We report a case of a 15 years old girl who presented a post hanging pulmonary edema, and we discuss its physiopathology and therapeutic implications.

Case report

A 15 years old girl, without any medical history, was admitted at the hospital after trying to commit suicide by hanging (posterior knot, undetermined duration). Upon admission, she was unconscious with a Glasgow Coma Scale at 4/15, pupils equal and reactive to light, her blood pressure was 120/80 mmHg, heart rate was 100 beats/min, respiratory rate of 30 cycles/min and oxygen saturation of 55% on room air. The clinical exam revealed a neck abrasion of the rope of the hanging (figure 1A) with a thin ligature mark on the posterior neck and bilateral crepitations on the pulmonary auscultation. She was intubated based on neurological and respiratory criteria (tidal volume at 400 ml, FiO2 at 100%, PEEP at 8 and Frequency at 12). A neck brace was put on before intubation to respect the straightness of the cervical spine.

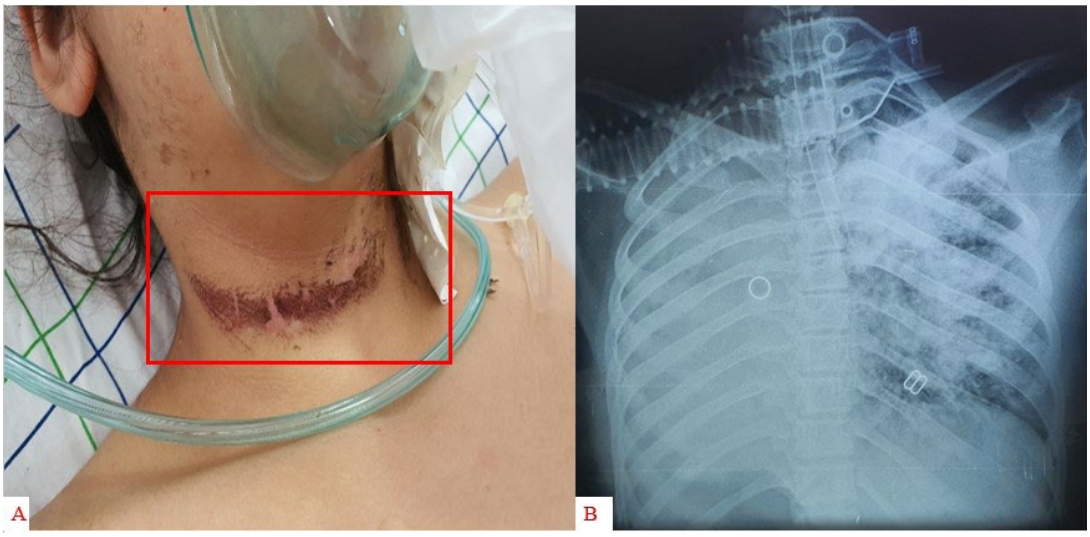

Figure 1 : clinical constat and results of Chest X-ray
• A : neck abrasion of the rope of the hanging
• B: bilateral interstitial alveolar opacities with right hemithorax

A chest X-ray showed bilateral interstitial alveolar opacities with right hemithorax white-out (figure 1B), which was confirmed by the thoracic CT scan that showed a diffuse pulmonary edema without pneumothorax nor pneumomediastinum (figure 2A and 2B). A cerebral CT scan was normal and cervical CT showed reversal of cervical lordosis (figure 2C). The laboratory results revealed an Hemoglobin at 12.9 g/dl, leukocytosis at 15200/mm3, her platelet count was 280000, Hemoglobin 12.9 g/dl, Urea 0,15 g/l and blood glucose was 2.21g/l.

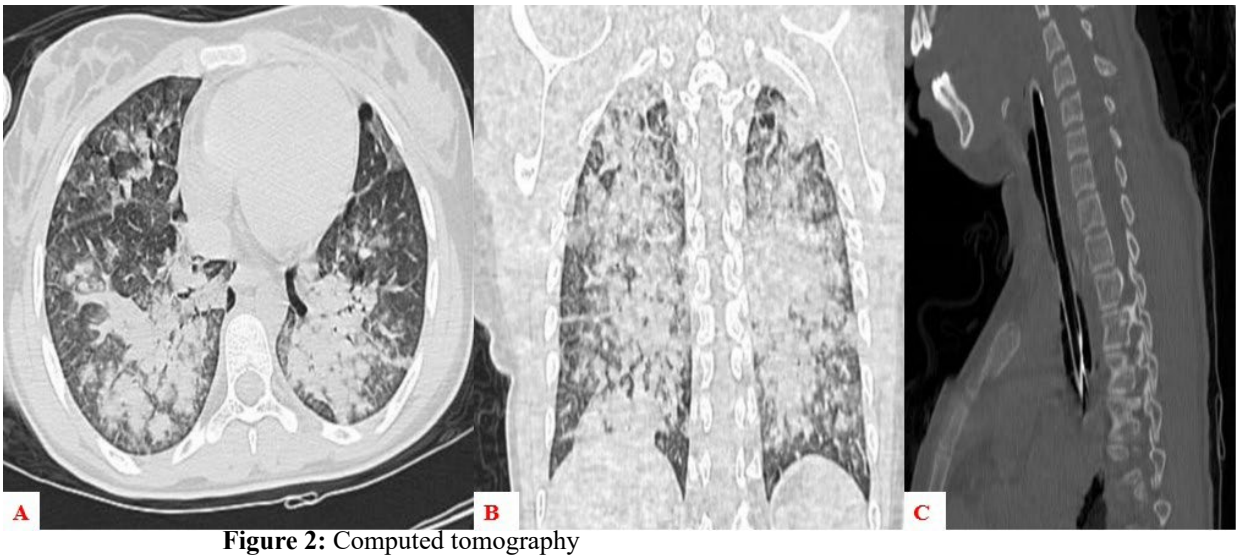

Figure 2: Computed tomography
- A, B: chest CT showing a diffuse pulomnary edema
- C : cervical CT showing reversal cervical lordosis

The child was sedated with midazolam and fentanyl, and was put on antibiotics with ceftriaxone, gentamicin, metronidazole and therapeutic anticoagulation was started using enoxaparin 1 mg/kg once daily.

During the next 3 days, the respiratory status improved progressively with the reduction of FiO2. An X-ray chest showed a regression of the opacities. The biological parameters were also improving hemoglobin at 11.6 g/dl, WBC at 12600/mm3, platelet count at 184000/mm3.

On day four, the patient was extubated and oxygen saturation of 92% on 15 l/min of oxygen via a non rebreather mask. Her respiratory rate was 20 bearths/min. The patient was discharged to the medical department 3 days later.

Discussion

The NPPE incidence is estimated at 1 in 1000 general anesthesia cases [3]. However, the post hanging NPPE is not well known by physicians. In fact, there are few published cases of post hanging NPPE, probably due to the high mortality rate of hangings [1].

Two physio-pathological types are described. In type 1, the inspiration effort with occluded upper airways can create an intrathoracic depression with an important pressure decrease, which increases the venous return and the right cavities' pressure. A

pressure gradient is then created and transfers fluids from the capillaries to the interstitium. The NPPE is more frequent in healthy young people, due to their higher thoracic extension capacities that can generate more important negative pressure (down to -140 mmHg). In type 2, the effort of expiration on an occluded airway increases the intrathoracic pressure and decreases the venous return. When the obstacle is lifted, the sudden drop in intrathoracic pressure is responsible for the pulmonary edema with a type 1-like mechanism [3,4]. Ackland et al. suggest that pulmonary edema can be secondary to the alveolar-capillary membrane damage, which can imitate the clinical and radiographic expression of an ARDS, thus misleading the diagnosis and delaying the proper treatment [5].

The type 1 is more common due to its more frequent etiologies (laryngospasm, residual paralysis, bite of the intubation tube or the laryngeal mask, laryngeal foreign body, recurrent laryngeal nerve paralysis…) [2-6].

The hypoxia and hyperadrenergic state contribute to the edema. In fact, the airways obstruction is responsible for hypoxia which increases the pulmonary vascular resistance and therefore the pulmonary capillary pressure. The hyperadrenergic response to the stress increases the blood flow in the pulmonary system and then increases the pulmonary vascular resistance. A neurogenic contribution secondary to cerebral hypoxia can also be incriminated. All these factors contribute to the edema constitution [3-7].

Death can occur either by cerebral hypoxia (secondary to the occlusion of airways or cervical vessels) or by cardiac arrest (due to vagal inhibition secondary to carotid compression) [8].
The NPPE may present as acute respiratory failure with dyspnea or polypnea, paradoxical breathing and desaturation. Cracking rales can be found at auscultation and pink foamy secretions at aspirations [2, 7-9].

The management of this form of pulmonary edema is based on the optimization of oxygenation by maintaining adequate supplementation of oxygen and addition of PEEP or the use of non-invasive ventilation in non-intubated patients. PEEP level must be adapted to saturation and arterial blood gas parameters [8-10].

In our case, upon the arrival of the patient, she was intubated considering her neurological and respiratory state. The pulmonary edema was diagnosed on chest-computed tomography and treated by titration of PEEP level with favorable outcome. The exact duration of hanging was unknown, but there was no cardiac arrest.

Conclusion

Cardiac complications of hanging may be various, pulmonary edema is one of them; it can be caused by various mechanisms. The management of this complication is based on mechanical ventilation and prognosis depends on the immediate consequences of hanging [11, 12].

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