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ACUTE POST-EXTUBATION NEGATIVE PRESSURE PULMONARY EDEMA

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ABSTRACT

Negative pressure pulmonary edema (NPPE) is a rare but serious complication that can occur after extubation due to upper airway obstruction. We report the case of a healthy 20-year-old male who developed acute respiratory distress shortly after emergency orthopedic surgery. Clinical signs and imaging were consistent with NPPE. Supportive treatment, including corticosteroids, diuretics and non-invasive ventilation, led to complete recovery within 24 hours. This case highlights the importance of early recognition and prompt management of NPPE, even in low-risk patients.

INTRODUCTION

NPPE is a rare but potentially life-threatening complication that can occur following acute upper airway obstruction, often in the immediate postoperative period. It was first described by Capitanio and Kirkpatrick. NPPE results from the generation of high negative intrathoracic pressure during forceful inspiratory efforts against an obstructed airway, leading to fluid transudation into the alveolar spaces.[1] Though uncommon, NPPE should be considered in the differential diagnosis of sudden postoperative respiratory distress, especially in otherwise healthy patients. This case report describes an instance of NPPE in the immediate postoperative period after extubation, highlighting the clinical presentation, timely diagnosis and effective management that led to a full recovery.

CASE REPORT

A 20-year-old male with no comorbidities presented for elective open reduction and internal fixation of a displaced left olecranon fracture. His airway assessment and pre-operative investigations were normal.

Cardiac monitor was connected, showing normal baseline heart rate and blood pressure with normal sinus rhythm.

Under all aseptic precautions, ultrasound (USG) guided left supraclavicular block was administered under local anesthesia with 12 ml of 0.5% Bupivacaine and 12 ml of 2% Lignocaine with Adrenaline and adequacy of block was confirmed. General anesthesia was induced with IV Fentanyl (100 µg), Propofol (200 mg), and Cisatracurium (12 mg). The patient was intubated with an 8.0 mm cuffed endotracheal tube. He was ventilated with a mixture of oxygen and nitrous oxide (1:1) and Sevoflurane to maintain a MAC of 1.2–1.4. Surgery lasted for one and half hour and his vitals remained stable. He was infused with 1500 ml of crystalloid solution.

At the conclusion of surgery, he was reversed with IV Neostigmine (2.5 mg) and Glycopyrrolate (0.4 mg). He was extubated after oropharyngeal suction. He was awake and following oral commands but shortly after, he had difficulty in breathing. He was not maintaining oxygen saturation (SpO2). Immediately he was placed in thirty degrees propped up position. There were coarse crackles with bronchospasm and reduced bilateral air entry on auscultation. Immediate assisted mask ventilation started. IV Hydrocortisone(100mg), IV Dexamethasone (8mg) and IV Frusemide (20mg) given. This was followed by Salbutamol and Budesonide nebulization. Arterial blood gas was normal except pO2 of 48 mmHg and SO_2 of 83.6%.

After stabilization, he was shifted to Intensive care unit and started on overnight non-invasive ventilation. Chest X-ray showed diffuse bilateral opacities but no pneumothorax. Fig.1 Pulmonology consultation ruled out

other causes like Pulmonary embolism or cardiac involvement. (Trop T and D-dimer were normal). Chest X-ray on the following morning was normal, and the patient recovered uneventfully.

DISCUSSION

Pulmonary edema occurring after extubation in a young, otherwise healthy patient may be attributed to various causes such as NPPE, aspiration, or bronchospasm.[2–4] Although rare, NPPE has been well-documented, especially in cases involving upper airway obstruction or laryngospasm.[5]

NPPE is usually caused by intense inspiratory effort against a closed glottis, creating negative intrathoracic pressure that draws fluid into alveolar spaces.[6] Although no frank obstruction was observed in this case, transient upper airway resistance or laryngeal spasm cannot be ruled out.[7]

Differential diagnoses such as aspiration pneumonitis or pulmonary embolism were ruled out based on clinical, laboratory and radiological evaluations.[8] Though USG reduces complications and improves regional block success, it does not eliminate the risk of pleural injury completely.[9] In our case, pneumothorax ruled out with radiological and clinical findings. The rapid resolution of symptoms and radiographic findings further supports the diagnosis of NPPE or a transient inflammatory pulmonary event.[10]

Post-anesthesia, pulmonary complications are more common in high-risk patients (elderly, obese, smokers), but can occasionally occur in healthy individuals, underscoring the importance of vigilance even in low-risk populations.[11] Use of volatile anesthetics, residual neuromuscular blockade and changes in positioning may all play contributory roles.[12,13]

Steroids, diuretics, and supportive oxygen therapy remain the cornerstone of management in suspected NPPE and post-anesthetic pulmonary edema.[14,15] Non-invasive ventilation has been shown to be effective in reversing hypoxia and preventing further deterioration.[16]

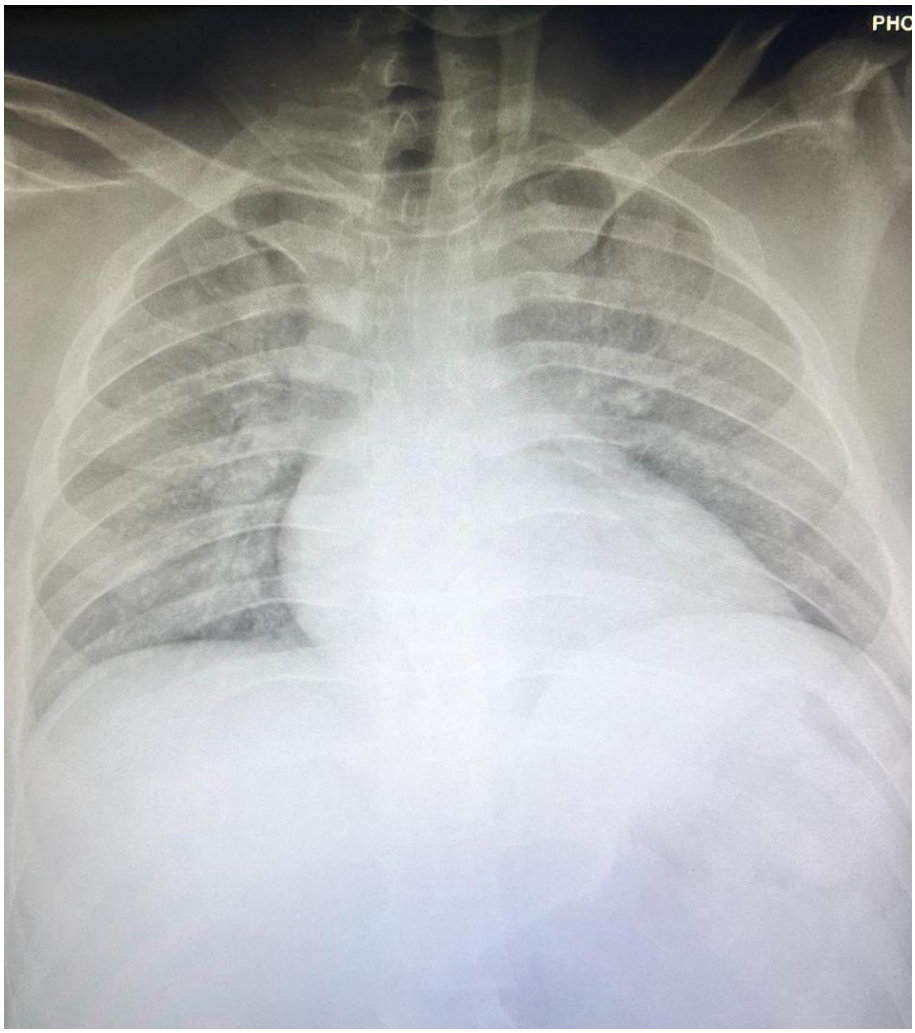

Fig. 1

CONCLUSION

This case highlights the successful recognition and prompt management of NPPE, underscoring the importance of early diagnosis, supportive care and airway protection in achieving favourable outcomes.

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