Case Report

Negative pressure pulmonary edema following tracheostomy tube corking unmasking suprastomal granuloma: A case report

Jayabharathi[1]*, .Ramanathan kannan subbaiah[2]

[1]MEM PG, Department of Emergency Medicine, Kauvery Hospital, Tennur, Trichy, Tamil Nadu
[2]Consultant Haemato Oncologist, Kauvery Hospital, Cantonment, Trichy, Tamil Nadu
*Correspondence

Abstract

Background: Negative pressure pulmonary edema (NPPE) is a form of non-cardiogenic pulmonary edema caused by acute upper airway obstruction. A 79-year-old male with diabetes, hypertension, COPD, and chronic kidney disease on tracheostomy developed sudden dyspnea, tachypnea, and desaturation during a corking trial for decannulation. Examination revealed bilateral crepitations, and point-of-care ultrasound showed diffuse B-lines with preserved cardiac function, suggestive of NPPE. Immediate removal of the cork and supportive management led to rapid improvement. Bronchoscopy subsequently revealed a suprastomal granulomatous lesion causing significant airway narrowing, which became clinically relevant when airflow was redirected through the upper airway during corking. This case highlights that NPPE can occur during tracheostomy corking and may unmask previously undiagnosed suprastomal airway pathology.

Keywords : Negative pressure pulmonary edema; Tracheostomy corking; Suprastomal granuloma; Airway obstruction; Non-cardiogenic pulmonary edema

Citation: Jayabharathi, .Ramanathan kannan Subbaiah. Negative pressure pulmonary edema following tracheostomy tube corking unmasking suprastomal granuloma: A case report. Kauverian Med J. 2026;3(8):63—68.

Academic Editor: Dr. Venkita S. Suresh

ISSN: 2584-1572 (Online)

1. Introduction

Negative pressure pulmonary edema (NPPE) is a form of non-cardiogenic pulmonary edema that results from generations of markedly negative intrathoracic pressure during forceful inspiratory efforts against an obstructed airway. This leads to increased venous return, elevated pulmonary capillary hydrostatic pressure, and rapid transudation of fluid into alveolar spaces.

NPPE is most commonly reported in the perioperative setting, particularly following post-extubation laryngospasm. However, its occurrence in nonanaesthetic settings such as tracheostomy corking or decannulation trials is less frequently recognized.

Suprastomal or upper tracheal lesions may remain clinically silent because airflow bypasses them through the tracheostomy tube. These lesions can become clinically significant only when airflow is redirected through the upper airway during corking, poten-

tially resulting in acute obstruction. We present a case of NPPE precipitated by tracheostomy tube corking, which led to the identification of a suprastomal granuloma as the underlying cause of airway obstruction.

2. Case presentation

A 79-year-old male with known history of type 2 diabetes mellitus, hypertension, chronic obstructive pulmonary disease, and chronic kidney disease was on tracheostomy and admitted for planned decannulation.

Clinical course

During a supervised corking trial, the patient developed acute respiratory distress within hours . He experienced sudden dyspnea, tachypnea with respiratory rate approximately 30–36 breaths per minute, tachycardia, and oxygen desaturation with SpO_2 dropping to 83–93%.

Examination

On examination, the patient was conscious but anxious. Bilateral diffuse crepitations were noted on chest auscultation. Blood pressure remained stable around 130/70 mmHg. There were no signs of fluid overload such as peripheral edema or raised jugular venous pressure. Point-of-care ultrasound (POCUS) showed preserved left ventricular systolic function, no right atrial or right ventricular dilatation, a small and collapsing inferior vena cava, and bilateral diffuse B-lines across multiple lung zones. Lower limb venous Doppler was negative for deep vein thrombosis.

3. Differential diagnosis

Cardiogenic edema**:** No evidence of pedal edema, no e/o elevated JVP,pocus showed preserved lv function ,and a collapsing IVC.

ARDS : Unlikely due to abrupt onset following airway obstruction.

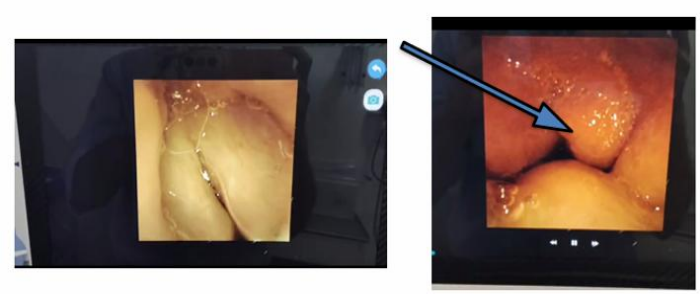
AeCOPD: Pocus showed diffuse b-lines.

4. Diagnosis

Based on the sudden onset following airway obstruction, ultrasound findings, and absence of cardiac dysfunction, a diagnosis of Negative pressure pulmonary edema was made.

5. Management

Immediate removal of the tracheostomy cork restored airway patency. The patient was managed with supplemental oxygen therapy, nebulization, tracheal suctioning, fluid restriction, and diuretics. Flexible bronchoscopy performed the following day revealed a granulomatous lesion located above the tracheostomy stoma (suprastomal region), causing significant narrowing of the airway lumen. The lesion did not significantly impair respiration while the tracheostomy tube was patent but became critical when airflow was redirected through the upper airway during corking, leading to acute obstruction.



Bronchoscopic view of the suprastomal airway demonstrating a smooth, pale, lobulated granulation tissue arising from the tracheal wall ,causing significant luminal narrowing

6. Outcome

The patient demonstrated rapid clinical improvement with normalization of oxygen saturation and within resolution respiratory distress within a short duration.

7. Discussion

Pulmonary edema

It is the accumulation of fluid in the lung intersititium and alveoli, leading to impaired gas exchange and hypoxia.

Classification of pulmonary edema

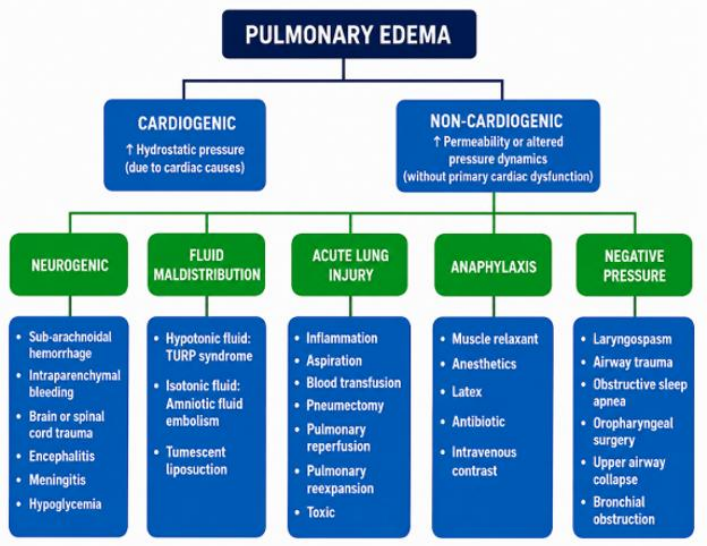


Image Source: *Ali Hossein Samadi Takaldani, ScienceDirect. Vol 1; Issue 7: 2024*

Pathophysiology

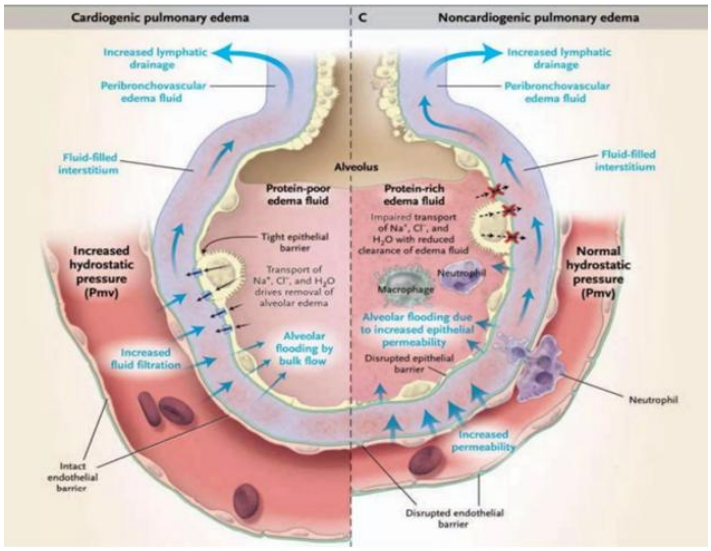
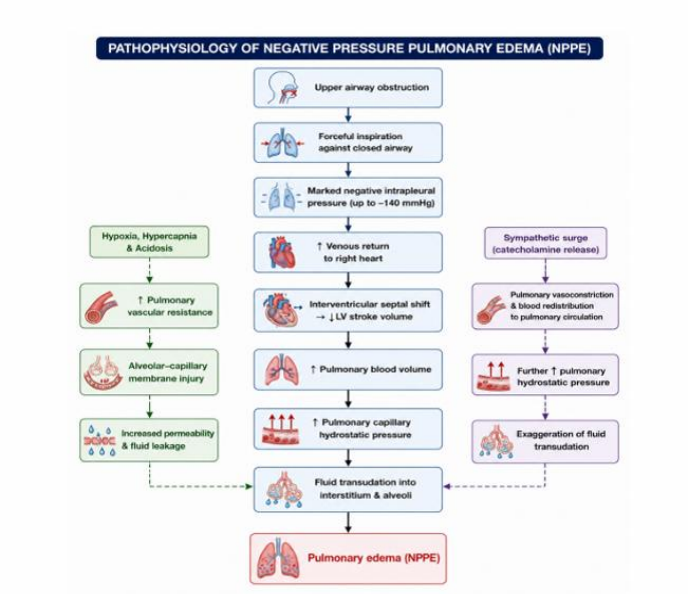


Fig (1): Mechanisms of cardiogenic and non-cardiogenic pulmonary edema

Schematic representation showing the pathophysiological differences between cardiogenic and non-cardiogenic pulmonary edema. Cardiogenic pulmonary edema results from increased hydrostatic pressure leading to transudation of protein-poor fluid, whereas non-cardiogenic pulmonary edema is characterized by increased alveolar-capillary permeability resulting in protein-rich fluid accumulation.

8. Negative pressure pulmonary edema

NPPE is a non-cariogenic pulmonary edema which results from markedly negative intrathoracic pressure generated during forceful inspiration against an obstructed airway. This leads to increased venous returns, elevated pulmonary capillary hydrostatic pressure, and rapid accumulation of fluid within the alveolar spaces. It is most commonly reported in peri-anaesthetic settings, while occurrence during tracheotomy corking is rare.



Clinical features

- Acute respiratory distress (tachypnea, use of accessory muscle)
- Hypoxia, hypercapnia
- Cyanosis
- Pink frothy sputum
- Bilateral crackles on auscultation

Radiological findings

CHEST X-RAY -Bilateral, central(perihilar),alveolar and interstitial infiltrates,diffuesly increased intensity, widened vascular shadows; usually without apical diversion(unlike cariogenic edema).

Feature	**Type 1 NPPE**	**Type 2 NPPE**
Trigger	Acute obstruction	Relief of chronic obstruction
Mechanism	Strong inspiratory effort - negative pressure	Sudden loss of peep
Onset	Immediate	Minutes to hours
Typical setting	Post extubation laryngospasm	Post airway surgery
Patient profile	Young, healthy	Chronic obstruction history

Management

- Ensure airway patency
- Correct hypoxia
- Oxygen therapy (high flow if needed)
- Continuous monitoring (SpO_2, ABG, vitals)
- CPAP / BiPAP in mild to moderate cases
- Endotracheal intubation and mechanical ventilation in severe or failing cases
- Use lung-protective ventilation strategy
- Avoid fluid overload
- Maintain euvolemia
- Diuretics (e.g., furosemide) – not primary treatment, use selectively
- Relieve airway obstruction
- Manage laryngospasm or precipitating cause

- Consider venovenous ECMO in refractory cases
- Rapid recovery expected within 24–48 hours with timely treatment

9. Conclusion

Negative pressure pulmonary edema is a rare but important complication of tracheostomy corking. In this case, corking redirected airflow through the upper airway, where a suprastomal granuloma caused significant narrowing and acute obstruction. Forceful inspiratory efforts against this obstruction generated markedly negative intrathoracic pressure, leading to pulmonary edema. Early recognition and prompt relief of the obstruction result in rapid recovery and favorable outcomes.

References

[1] Bhattacharya M, Kallet RH, Ware LB, Matthay MA. Negative-pressure pulmonary edema. Chest. 2016;150(4):927–933.

[2] Deepika K, Kenaan CA, Barrocas AM, Fonseca JJ, Bikazi BG. Negative pressure pulmonary edema after acute upper airway obstruction. J Clin Anesth. 1997;9(5):403–408.

[3] Lemyze M, Mallat J. Understanding negative pressure pulmonary edema. Intensive Care Med. 2014;40(8):1140–1143.