American Journal of Case Reports

e-ISSN 1941-5923
© Am J Case Rep, 2026; 27: e952567
DOI: 10.12659/AJCR.952567

Received: 2025.12.24
Accepted: 2026.05.06
Available online: 2026.05.28
Published: 2026.XX.XX

Secondary Pneumomediastinum Following Bronchoscopic Interventions in Patients With Prolonged Tracheostomy Intubation: A Case Series

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

BE Lu Pengcheng*
D Fan Tao*
BC Zeng Qing*
B Zheng Peng
B Chen Na
EF Tong Fangming
AG Xie Qiuyou

Department of Rehabilitation Medicine, Zhujiang Hospital, Southern Medical University, Guangzhou, Guangdong, PR China

* Lu Pengcheng, Fan Tao, and Zeng Qing contributed equally to this work

Corresponding Authors: Xie Qiuyou, e-mail: xqy7180@163.com; Tong Fangming, e-mail: tfm373@163.com

Financial support: The study was supported by the grants from Guangzhou Municipal Science and Technology Bureau (2023A04J0444), Southern Medical University Youth Incubation Program (PY2018N057), Natural Science Foundation of China (82171174, 82371184), and the Key Technologies R&D Program of Guangdong Province (2023B0303020003)

Conflict of interest: None declared

Case series

Patients: Mele, 38-year-old • Mele, 69-year-old • Mele, 19-year-old

Final Diagnosis: Prolonged tracheostomy intubation

Symptoms: A severe cough • shortness of breath • subcutaneous emphysema

Clinical Procedure: Anti-infection • closed chest drainage • cough suppression • subcutaneous incision

Specialty: Critical Care Medicine • Rehabilitation

Objective: Rare coexistence of disease or pathology

Background: Tracheostomy is a cornerstone of long-term intensive care, utilized in approximately 15% of intensive care unit patients. While beneficial, prolonged tracheostomy intubation initiates structural changes within the tracheal architecture, predisposing patients to severe late-stage complications, including secondary pneumomediastinum, which can rapidly progress to tension physiology and obstructive shock. Specific triggers, pathophysiological pathways (eg Macklin effect vs direct disruption), and multi-compartment air tracking in tracheostomized populations remains incompletely understood. This report seeks to guide early prevention and emergency management by describing chronological clinical presentations, diagnostic decision-making, and therapeutic outcomes of complications in 3 prolonged-tracheostomy patients.

Case Reports: Three male patients underwent prolonged tracheostomy intubation (>1 month) and developed pneumomediastinum following airway manipulation. A 38-year-old patient developed severe subcutaneous emphysema (SE) 48 hours post-decannulation following tracheal polypectomy. Computed tomography (CT) ruled out tension pneumothorax, and stable hemodynamics justified successful conservative management. A 69-year-old patient experienced sudden dyspnea and SE during bedside bronchoscopy. CT revealed extensive soft-tissue gas and left-sided pneumothorax, requiring an emergency bedside subcutaneous incision and closed chest drainage due to rapid respiratory deterioration. A 19-year-old patient developed rapidly spreading SE and progressed to cardiac arrest during a bronchoscopic polypectomy. Malignant tension pneumomediastinum ensued following massive gas entry, severely obstructing venous return, which proved fatal despite advanced life support.

Conclusions: Secondary pneumomediastinum during airway manipulation demands a high index of clinical suspicion. While stable cases resolve conservatively, rapid gas accumulation causes obstructive shock requiring immediate thoracic decompression. Strict preoperative cough suppression and continuous cuff pressure monitoring are critical preventative strategies.

Keywords: Intubation • Pneumomediastinum • Pulmonology • Tracheostomy • Bronchoscopy • Subcutaneous Emphysema • Case Reports

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/952567

2064 — 4 12

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

Introduction

The utilization of tracheostomy in a clinical environment has undergone a notable transformation, evolving from an emergency measure for acute upper airway obstruction to a cornerstone of long-term intensive care management. Approximately 15% of patients admitted to intensive care units (ICUs) eventually require the procedure to facilitate prolonged mechanical ventilation or provide a secure airway for patients with chronic neurological deficits [1]. However, prolonged tracheostomy intubation (exceeding 1 month) inevitably initiates a series of structural and physiological changes within the tracheal architecture. These changes predispose patients to late-stage complications such as tracheal stenosis (occurring in 6% to 21% of cases), tracheomalacia, and the formation of obstructive polyps [2,3].

Among the rarest but most clinically devastating of these sequelae is secondary pneumomediastinum, defined as the abnormal presence of ectopic air within the mediastinal space following an iatrogenic trigger. Unlike spontaneous pneumomediastinum (Hamman's Syndrome), which typically follows a benign and self-limiting course in healthy individuals, secondary cases in critically ill patients carry significantly higher morbidity and mortality [4]. The clinical significance of secondary pneumomediastinum lies in its potential for rapid progression into tension physiology, a state where gas accumulation occurs at a rate exceeding the body's ability to dissipate it. This progression compromises venous return, leading to obstructive shock and cardiac arrest [5].

Despite its life-threatening potential, there remains a significant lack of standardized analysis regarding the specific triggers of pneumomediastinum in the tracheostomized population, particularly during routine interventions such as bronchoscopy or decannulation [6]. We hypothesize that mechanical airway manipulation, combined with paroxysmal coughing, exacerbates underlying ischemia-induced structural weaknesses, leading to varied degrees of full-thickness airway disruption. This case series aims to address the current literature gap by delineating the chronological clinical presentations of 3 distinct cases, evaluating the rationale behind specific diagnostic and therapeutic interventions, and proposing practical clinical protocols for early prevention and emergency management.

Case Reports

The information on 3 cases was collected through the referral process, with subsequent follow-up details obtained via communication with the primary intensive care team. Authorization to use anonymized patient data and associated images was obtained from the patients' next of kin.

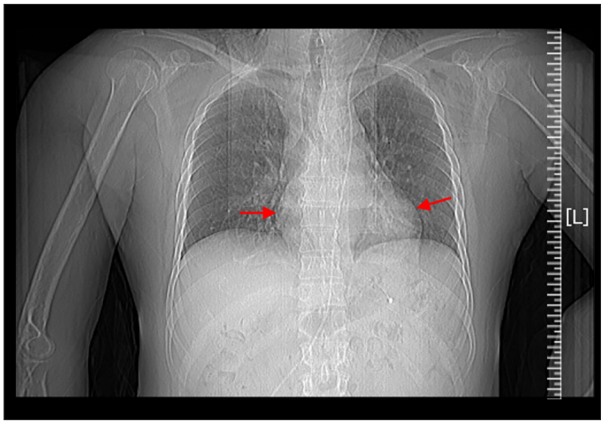

Figure 1. Chest CT scan of the patient in Case 1 revealed a small amount of mediastinal emphysema. CT, computed tomography.

Case 1

A 38-year-old man presented with postoperative closed craniocerebral trauma and a ventriculoperitoneal shunt. He had been tracheostomy intubated for 4 months, which led to the development of a tracheal polyp obstructing 90% of the tracheal lumen. To facilitate decannulation, a bronchoscopic tracheal polypectomy was performed. After a thorough evaluation and a successful 24-hour period of tube capping, the patient demonstrated good tolerance with stable vital signs (SpO_2 >95%, respiratory rate 16-20 breaths/min). The tube was subsequently removed under close monitoring.

However, 48 hours post-decannulation, the patient developed a severe, paroxysmal cough and mild shortness of breath. Palpation revealed crepitus in the neck, indicating subcutaneous emphysema (SE). To explicitly differentiate pneumomediastinum from tension pneumothorax or acute pulmonary embolism, an urgent chest CT scan was selected as the optimal imaging modality. The scan revealed SE of the neck and anterior thoracic region, along with a small amount of mediastinal emphysema, effectively ruling out a pneumothorax (**Figure 1**). Because the patient's hemodynamics and oxygen saturation remained stable, indicating a slow and localized air leak, a conservative management rationale was adopted to avoid unnecessary surgical trauma. Treatment included broad-spectrum anti-infection therapy, strict cough suppression using antitussives to prevent further air leakage through the healing stoma, and bed rest. The patient's condition gradually improved over the next week, and a follow-up chest CT scan confirmed the resorption of ectopic air.

Case 2

A 69-year-old man, primarily diagnosed with intracerebral hemorrhage with intraventricular extension, had been tracheostomy

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

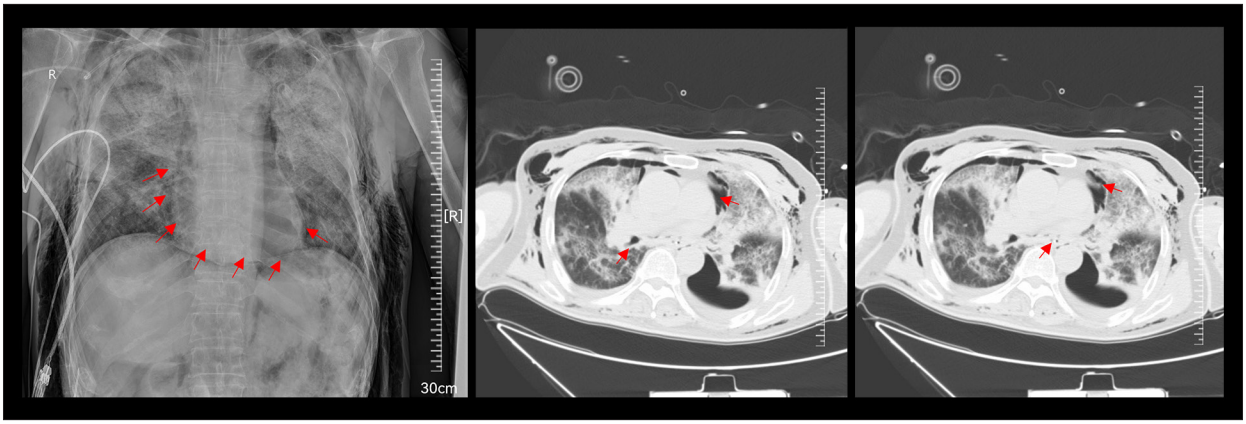

Figure 2. A CT chest scan of the patient in Case 2 demonstrated gas in the soft tissue involving the neck and mediastinum, predominantly surrounding the trachea and esophagus. CT, computed tomography.

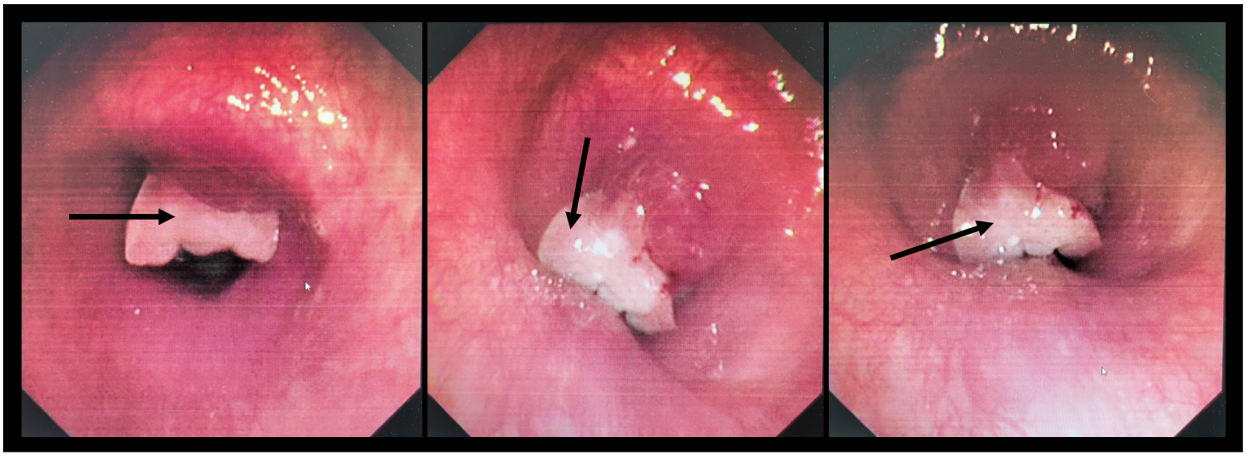

Figure 3. Case 3 involved prolonged tracheostomy intubation complicated by a large tracheal polyp.

intubated for more than 1 month. Due to recurrent pulmonary infections and mucus obstruction, a bedside bronchoscopy was performed to clear the airway and collect a bronchoalveolar lavage sample. During the procedure, the patient experienced severe coughing fits, immediately followed by sudden dyspnea, a drop in oxygen saturation (SpO_2 <85%), and the rapid onset of extensive SE spreading across the chest and neck.

Given the acute respiratory distress, a differential diagnosis of alveolar rupture (the Macklin effect) or major iatrogenic airway laceration was considered. A follow-up chest CT, performed under respiratory support, confirmed extensive gas in the soft tissue, extending into the neck and mediastinum, alongside a left-sided lower lobe pneumothorax and bilateral lung infection foci (**Figure 2**). The rapid accumulation of air combined with the presence of a pneumothorax under positive-pressure support indicated an impending tension physiology. The clinical rationale shifted, as conservative treatment alone would be insufficient to prevent hemodynamic collapse. An urgent consultation with a thoracic surgeon resulted in an emergency bedside subcutaneous incision and the placement of closed chest drainage to explicitly restore ventilation. Following the mechanical release of trapped air, conservative treatments (anti-infection and oxygen therapy) were continued in the ICU. A subsequent chest CT scan conducted 5 days later revealed a significant reduction in gas in the soft tissue.

Case 3

A 19-year-old man with a history of subarachnoid hemorrhage had been tracheostomy intubated for more than 4 months. Prolonged high cuff pressures resulted in severe irritation and ischemic injury to the tracheal mucosa, forming a large tracheal polyp that obstructed 70% of the lumen and caused respiratory distress (**Figure 3**). A bronchoscopic tracheal polypectomy was initiated. During the procedure, the patient experienced severe coughing and suddenly became impossible to ventilate, rapidly developing extensive SE over the entire body.

The chronology of his hemodynamic decline was catastrophic: within minutes, his respiratory rate increased to 46 breaths/min, SpO_2 dropped to 54% on balloon mask-assisted breathing,

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

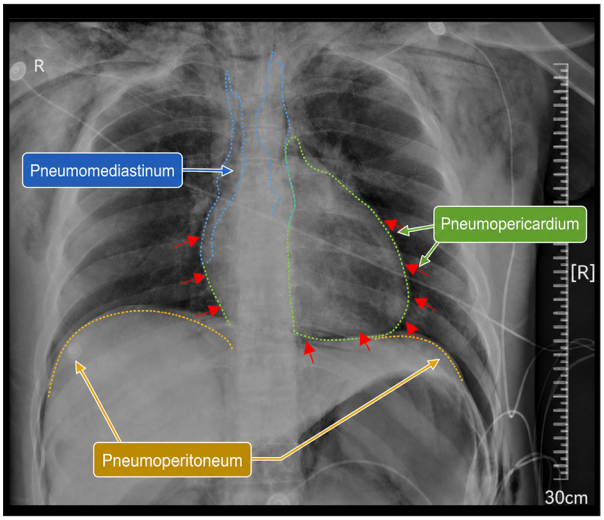

Figure 4. An emergency bedside chest X-ray of the patient in Case 3 showed linear streaks of air outlining the mediastinal structures. The annotated labels indicate pneumomediastinum (blue), pneumopericardium (green), and pneumoperitoneum (yellow).

and his heart rate plummeted to 20 beats/min, rapidly progressing to cardiac arrest. Because the patient was too unstable for transport to the CT suite, a bedside chest X-ray was utilized. Imaging showed linear streaks of air outlining the mediastinal structures, a pneumopericardium, and a massive pneumoperitoneum visible around the liver, along with extensive SE (**Figure 4**). Despite persistent cardiopulmonary resuscitation (CPR) and advanced life support in the ICU, the patient did not regain spontaneous circulation, and clinical death was declared. The massive and rapid gas entry had created a malignant tension pneumomediastinum, severely obstructing venous return to the heart and rendering standard chest compressions fundamentally ineffective.

Discussion

This case series illustrates the severe spectrum of secondary pneumomediastinum triggered by airway manipulation in patients with prolonged tracheostomy. Although local complications of tracheostomy are frequently discussed, our findings highlight a critical gap regarding the life-threatening potential of rapid ectopic air accumulation. The distinct clinical outcomes observed in our cases underscore the necessity of deeply understanding the underlying pathological pathways.

Pathophysiological Mechanisms: The Macklin Effect vs Direct Disruption

The migration of ectopic air into the mediastinum occurs via 2 primary pathways: the Macklin effect (alveolar rupture due to high intra-alveolar pressure gradients) or direct iatrogenic disruption of the central airway. The Macklin effect, initially described in 1944, offers a crucial explanation for pneumomediastinum in the absence of a visible central airway tear. Pathologically, it begins with a sudden, drastic increase in intra-alveolar pressure, often provoked by paroxysmal coughing during bronchoscopic interventions or high-pressure mechanical ventilation, which creates a severe pressure gradient relative to the lung interstitium. When this gradient exceeds tissue tolerance, the alveoli undergo rupture, specifically at their junction with the peribronchovascular sheaths [7]. Rather than entering the pleural space, the escaped air tracks proximally along the perivascular and peribronchial fascial sheaths toward the pulmonary hilum. From the hilum, the air is divided into the low-pressure fascial planes of the mediastinum. It is noteworthy that in critically ill patients with preexisting lung infections (as illustrated in Case 2), the inherent fragility of the lung parenchyma significantly reduces the pressure threshold required to trigger alveolar rupture during routine airway manipulation [7].

In stark contrast to the gradual interstitial tracking characteristic of the Macklin effect, direct iatrogenic disruption, as observed in our fatal Case 3, results in rapid and massive gas entry. The posterior membranous wall of the trachea is highly susceptible to mechanical injuries, with a capillary perfusion pressure of approximately 30 mmHg [8]. Chronic high cuff pressures induce insidious ischemia and full-thickness necrosis, severely weakening this tissue [9]. During bronchoscopic polypectomy, violent coughing drastically increases airway pressures, converting a structurally weakened posterior wall into a full-thickness tear. Unlike the Macklin effect, this direct breach, especially under positive-pressure ventilation, acts as a mechanical pump, forcing large volumes of air directly into the mediastinal space [6].

Multi-Compartment Air Leaks: The Genesis of Pneumopericardium and Pneumoperitoneum

A unique and highly lethal feature in Case 3 was the simultaneous presentation of pneumopericardium and massive pneumoperitoneum. Secondary pneumomediastinum rarely remains isolated due to the anatomical continuity of the fascial planes. Once massive amounts of air forcefully enter the mediastinum via a direct tracheal tear, the air rapidly tracks along these continuous low-pressure planes [10].

The pneumopericardium occurs when high-pressure ectopic air dissects along the venous sheaths at the root of the great vessels, penetrating the pericardial reflection. This intrapericardial air exacerbates the obstructive shock by causing an "extra-pericardial" cardiac tamponade, physically constricting the right ventricle and preventing diastolic filling. Simultaneously, the air tracks inferiorly through the natural diaphragmatic

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

hiatuses (such as the aortic or esophageal hiatus) or directly through anatomical foramina into the abdominal cavity, creating a tension pneumoperitoneum [10]. This massive intra-abdominal gas accumulation significantly elevates intra-abdominal pressure, further displacing the diaphragm upward, restricting ventilation, and severely impeding inferior vena cava blood flow. Clinicians must recognize that a sudden pneumoperitoneum following airway manipulation strongly indicates a massive descending mediastinal air leak rather than a primary gastrointestinal perforation [10].

Obstructive Shock and Rationale for Intervention

The fatal outcome in Case 3 underscores the profound physiology of obstructive shock driven by these multi-compartment leaks. When intramediastinal pressure surpasses 15 mmHg, the pressure gradient driving venous return is obliterated, precipitously dropping cardiac preload and leading to pulseless electrical activity.

The rationale for transitioning from conservative monitoring (Case 1) to surgical intervention (Case 2) relies entirely on identifying impending tension physiology. In fatal scenarios (Case 3), standard CPR alone is fundamentally futile because chest compressions cannot restore preload against massive intramediastinal and intrapericardial pressure. The immediate surgical release of trapped gas is the only definitive resuscitation measure. Recent advancements provide intensivists with new life-saving tools. For instance, a novel bedside mediastinal suction drain utilizing a modified peritoneal catheter (introduced in 2025) can rapidly reverse hemodynamic collapse via a subxiphoid approach [11]. Additionally, updated 2025/2026 guidelines for needle decompression emphasize utilizing the 4^{th}-5^{th} intercostal space along the anterior axillary line with a longer 5-8 cm needle to significantly reduce failure rates when concomitant tension pneumothorax is present [12].

Re-evaluating Clinical Standards and Future Directions

A critical revelation from recent literature is the necessity of re-evaluating traditional standards for tracheostomy tube cuff pressures. Historically, 30 cmH_2O was accepted as safe. However, recent studies demonstrating significant microstructural damage and ciliary loss at this pressure indicate that it is too high for prolonged mechanical ventilation. Current models suggest the effective dose (ED_{50}) to prevent mucosal injury is 25.6 cmH_2O (95% CI: 19.3-29.4 cmH_2O) [5]. Clinicians must avoid the dangerous practice of inflating cuffs up to 40 mmHg to correct leaks, which simply accelerates underlying necrosis [9]. Future studies should prioritize the development of smart tracheostomy tubes with automated pressure-regulating cuffs. Additionally, exploring pharmacological mucosal protectors, such as alkalinized lidocaine, may help mitigate local inflammatory responses to ischemia.

Conclusions

Secondary pneumomediastinum triggered by bronchoscopic interventions in patients with prolonged tracheostomy is a severe complication driven by mechanisms ranging from the Macklin effect to direct tracheal necrosis. Massive ectopic air accumulation can rapidly track across fascial planes, causing pneumopericardium, pneumoperitoneum, and profound obstructive shock. Based on our clinical experience, we propose the following practical recommendations: (1) mandatory preoperative administration of antitussives to strictly suppress coughing during airway manipulation; (2) continuous titration of cuff pressures to a stricter target of 22-25 cmH_2O to preserve mucosal integrity; and (3) maintaining bedside emergency thoracic decompression equipment, anticipating the need for novel surgical drainage or optimized needle decompression whenever high-risk airway interventions are performed.

Department and Institution Where Work Was Done

Department of Rehabilitation Medicine, Zhujiang Hospital, Southern Medical University, Guangzhou, Guangdong, PR China.

Informed Consent

Written informed consent for publication of their clinical details and/or clinical images was obtained from the patients or their proxies/guardians.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

References:

1. Hernandez Martinez G, Rodriguez M-L, Vaquero M-C, et al. High-flow oxygen with capping or suctioning for tracheostomy decannulation. N Engl J Med. 2020;383(11):1009-17
2. Cooper JD. Tracheal injuries complicating prolonged intubation and tracheostomy. Thorac Surg Clin. 2018;28(2):139-44
3. Ansari N, Ozgur SS, Giannetti R, et al. An interesting presentation of pneumomediastinum secondary to hyperemesis gravidarum in the second trimester. Cureus. 2023;15(11):e48574
4. Knittel-Hliddal S, Dumond S, Crincoli JR, et al. Extensive pneumomediastinum and pneumoperitoneum following endotracheal intubation. Chest. 2023;164(4):A1906-7

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

5. Benes L, Chau H, Cordero HJ, et al. A feared complication of airway management: Post-intubation tracheal tear. Chest. 2023;164(4):A2016-17
6. Mu G, Wang F, Li Q, et al. Reevaluating 30 cmH_2O endotracheal tube cuff pressure: risks of airway mucosal damage during prolonged mechanical ventilation. Front Med (Lausanne). 2024;11:1468310
7. Wang Q, Yao G, Xu H, et al. Safety analysis of application of mediastinal CO_2 aeration in mediastinal esophagectomy. Ann Palliat Med. 2020;9(5):3107-14
8. Ali A, Shiraishi K, Capirig CJ, Velasco-Hughes A. Successful minimally invasive treatment of tracheal perforation with tension pneumomediastinum and pneumothoraces in an intubated patient. Am J Respir Crit Care Med. 2025;211:A1185
9. Talwar A, Rajeev A, Rachapudi S, et al. Spontaneous pneumomediastinum: A comprehensive review of diagnosis and management. Intractable Rare Dis Res. 2024;13(3):138-47
10. Mahmud S, Datla S, Hoang M, et al. Tension in the air-cardiac tamponade secondary to tension pneumomediastinum. Circulation. 2025;152(Suppl. 3):A4368674
11. Wang H, Li X, Shi L, et al. Pneumoperitoneum caused by tracheal rupture due to tracheotomy: A case report. BMC Pulm Med. 2025;25(1):237
12. Ratnayake A, Dassanayake B, Kumarihamy P, et al. Massive isolated pneumomediastinum in a ventilated patient with COVID-19 pneumonia managed with the insertion of a novel mediastinal drain. SAGE Open Med Case Rep. 2025;13:2050313X251329773

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]