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CASE REPORT

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Pneumoperitoneum caused by tracheal rupture due to tracheotomy: a case report

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Abstract

Background Tracheal rupture is a rare but serious complication associated with tracheal intubation, often presenting with clinical manifestations such as subcutaneous emphysema, mediastinal emphysema, and pneumothorax. Pneumoperitoneum after tracheal intubation is an unusual occurrence. Treatment strategies typically include surgical intervention and conservative management. This article presents a case of tracheal rupture following tracheal intubation that led to massive pneumoperitoneum and was successfully managed with conservative treatment.

Case presentation A patient underwent tracheal intubation, during which a tracheal rupture occurred, leading to significant pneumoperitoneum. The condition was initially diagnosed based on clinical symptoms, including abdominal distension and imaging results. Conservative treatment, including respiratory support and monitoring, was implemented. Follow-up abdominal CT on day 6 demonstrated resolution of the pneumoperitoneum, and repeat bronchoscopy on day 10 confirmed healing of the tracheal laceration.

Conclusion Tracheal rupture and subsequent pneumoperitoneum should be considered as differential diagnoses in patients presenting with abdominal distension following tracheal intubation. Prompt diagnosis and appropriate conservative management can lead to favorable outcomes.

Keywords Tracheal rupture, Pneumoperitoneum, Tracheal intubation, Conservative treatment, Case report

Background

Tracheal rupture following endotracheal intubation is a serious but extremely rare complication that can be life-threatening. It commonly presents with subcutaneous emphysema, mediastinal emphysema, and pneumothorax. The occurrence of pneumoperitoneum secondary to tracheal rupture is exceedingly uncommon, with only a few cases reported in the literature. As such, clinical awareness of this condition remains limited. The etiology is often associated with high-risk procedures, such as mechanical ventilation or cardiopulmonary resuscitation, or with anatomical abnormalities like tracheal diverticulum. Clinicians should maintain a high index of suspicion in appropriate settings. Management of pneumoperitoneum caused by tracheal rupture should

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be individualized based on the severity of the condition, underlying causes, and associated complications. Treatment strategies generally include surgical intervention or conservative management. Herein, we report a rare case of massive pneumoperitoneum caused by tracheal rupture following tracheostomy, which was successfully managed with conservative treatment, aiming to provide reference for similar cases.

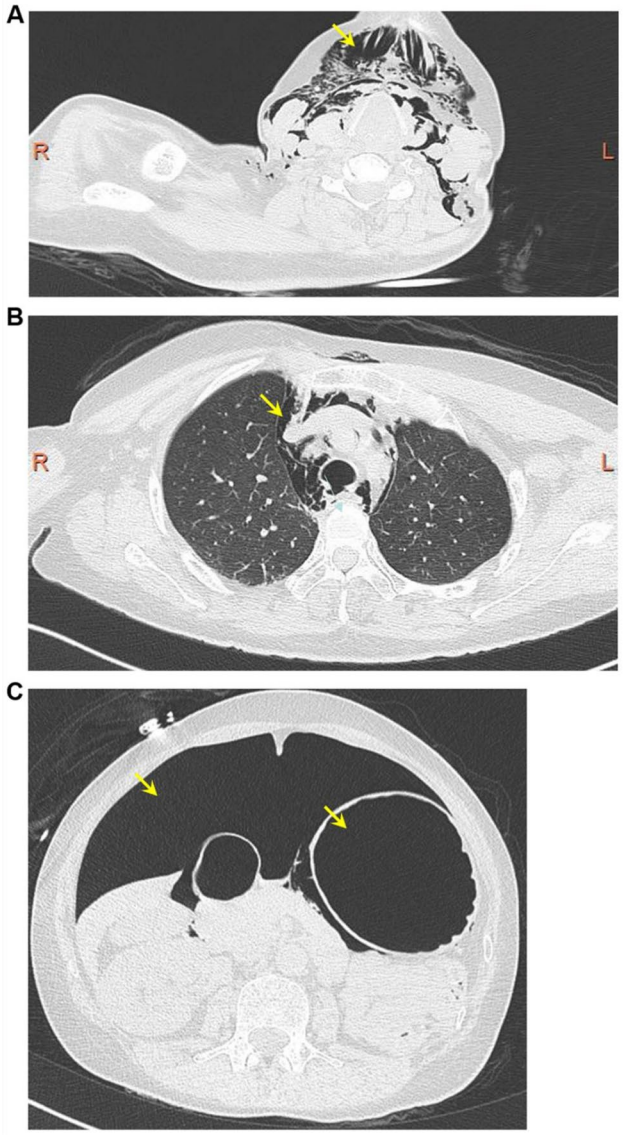

Fig. 1 Head and chest CT showing subcutaneous emphysema, mediastinal emphysema, pneumothorax, tracheal rupture, pneumoperitoneum, and gastric distention. (**A**) Subcutaneous emphysema in the neck. (**B**) Mediastinal emphysema, right-sided pneumothorax, subcutaneous emphysema in the chest wall, and tracheal rupture. (**C**) Free gas shadows and gastric pneumoperitoneum are visible in the abdominal cavity (arrows). CT imaging revealed extensive and punctate collections of free gas in both the intraperitoneal and retroperitoneal spaces

Case presentation

A 48-year-old female patient was admitted to our hospital on August 20th, 2024, with a chief complaint of intermittent wheezing and dyspnea for over three years, which had worsened in the last 8 h. The patient had a history of hysterectomy in 2015 for uterine fibroids, left kidney donation to a family member in 2016, and a diagnosis of hyperthyroidism in 2019, for which she received oral methimazole therapy. Two months prior, she experienced an acute asthma attack that required intubation and mechanical ventilation. Despite successful extubation, the patient continued to experience intermittent episodes of wheezing and throat discomfort, leading to the diagnosis of allergic laryngitis.

On admission, the patient presented to our emergency department with acute exacerbation of bronchial asthma. At approximately 04:30, she developed respiratory distress following nebulization treatment. Upon examination, breathing sounds were clear in both lungs; however, her SpO_2 decreased to 85%. Oxygen flow was increased to 10 L/min, and pharmacological interventions were administered; however, SpO_2 levels showed no significant improvement, and the symptoms of dyspnea and respiratory distress persisted. The patient displayed retraction and SpO_2 further declined to 78%. Arterial blood gas analysis revealed a pH of 7.165, partial pressure of carbon dioxide of 63.4 mmHg, partial pressure of oxygen (PaO_2) of 179 mmHg, and lactate level of 3.4 mmol/L. Tracheal intubation was attempted, and the glottis was swollen under a visual laryngoscope. Considering the difficulty of the airway, tracheal intubation was unsuccessful. A laryngeal mask was placed to maintain oxygenation, but SpO_2 continued to decrease progressively. Maintenance ventilation was provided by using a simple ventilator. Emergency tracheotomy was performed at 05:53, during which SpO_2 and heart rate declined, necessitating supportive measures to maintain vital signs. Tracheotomy was completed at 06:10, and mechanical ventilation was initiated [volume control mode, positive end-expiratory pressure (PEEP) 5 cm H_2O, tidal volume 450 mL, FiO_2 50%]. At this point, the patient's heart rate was 90 bpm, SpO_2 was 100%, and blood pressure was 133/94 mmHg. At 07:37, head and chest CT revealed subcutaneous emphysema in the right temporal region and retropharyngeal space, mediastinal emphysema, a small right-sided pneumothorax, and subcutaneous emphysema in the chest wall, raising the suspicion of tracheal membrane rupture (Fig. 1A-B). CT revealed gastric distention and pneumoperitoneum (Fig. 1C).

At 07:56, the patient was transferred to the Respiratory Intensive Care Unit and was in a sedated state but responsive to stimuli. Mechanical ventilation was continued through a tracheostomy. Upon examination, subcutaneous emphysema was palpable in the submandibular,

neck, and chest regions. Auscultation revealed coarse breath sounds with dry and wet rales in both lungs. The abdomen was distended, and percussion revealed tympany. Gastrointestinal decompression was performed, gas was expelled, and the mechanical ventilation settings were adjusted to the pressure control mode (PC, 13 cmH_2O; PEEP, 5 cmH_2O, FiO_2 40%). At 14:45, a tracheoscopy via a tracheostomy tube revealed a patent trachea with poorly defined tracheal rings. The tracheal mucosa at the distal end of the tracheostomy tube appeared disrupted, with a rupture measuring approximately 5 mm (Fig. 2A). The bronchoscope was passed through the rupture, revealing a false lumen in the posterior wall and a tear in the distal segment (Fig. 2B). After regaining consciousness, the patient was weaned off the invasive ventilator and was switched to high-flow oxygen therapy via a tracheostomy tube. Multidisciplinary discussion identified a posterior membranous wall injury. Multidisciplinary consultations were conducted to guide further management. Radiology and gastroenterology both considered tracheal rupture, with gastroenterology confirming the integrity of the esophagus and ruling

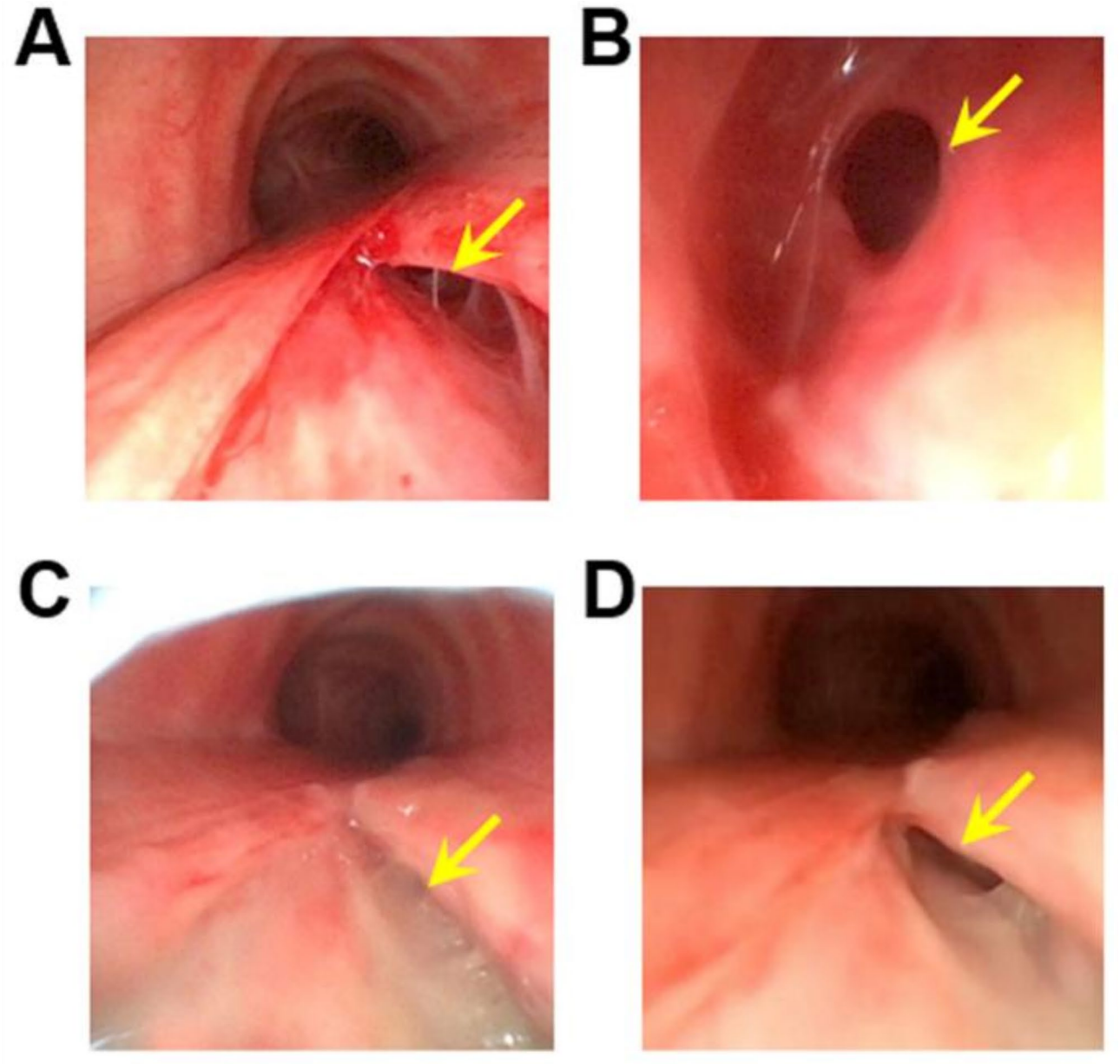

Fig. 2 Tracheobronchoscopic view showing rupture of the membranous part of the trachea. (**A**) Proximal segment. (**B**) Distal segment. (**C**) Disruption and rupture of the tracheal membranous mucosa, approximately 3.5 cm from the carina, extending upward to the distal end of the intubation site. (**D**) False lumen and fistula observed in the right lateral wall during inhalation, obscured distal view with purulent slough covering the mucosal surface

out esophageal injury. Gastric distention was noted, and continuation of gastrointestinal decompression was recommended. The presence of pneumoperitoneum raised concern for potential progression to abdominal compartment syndrome, which could be life-threatening; symptomatic decompression was advised. Otolaryngology suspected air leakage into the mediastinum through a lateral membranous tracheal defect and recommended avoiding positive pressure ventilation to prevent worsening mediastinal emphysema and potential cardiac arrest. Placement of a covered tracheal stent via a trans-laryngeal approach was suggested. Thoracic surgeons recommended either surgical repair or covered stent placement for tracheal rupture. The bronchoscopy team identified a membranous tracheal tear without significant airway stenosis but cautioned against stent placement due to risks of migration, airway obstruction, and difficulties with removal, favoring surgical intervention. General surgery advised further evaluation with gastroscopy to exclude esophageal injury, continuation of gastrointestinal decompression, complete abdominal CT imaging, and closed peritoneal drainage if necessary. Taking all opinions into consideration, and in light of the patient's history of asthma and acute exacerbation requiring emergency mechanical ventilation via tracheostomy, a diagnosis of tracheal rupture with associated mediastinal emphysema and pneumoperitoneum was made. Given the risks associated with endoscopic interventions, surgical management was deemed appropriate. Conservative treatment for the pneumoperitoneum was continued, including gastrointestinal decompression, fasting, and further imaging, with plans for percutaneous drainage if indicated. The timeline of clinical progression and treatments administered is summarized in Table 1.

Table 1 Summary of treatment interventions and clinical course

Date & Time	Intervention and Clinical Course
August 20, 2024	Acute exacerbation of bronchial asthma
August 20, 2024-05:53	Tracheostomy performed; mechanical ventilation initiated
August 20, 2024-07:37	Imaging suggested tracheal membranous rupture, gastric distension, and pneumoperitoneum
August 20, 2024-07:56	Transferred to RICU; mechanical ventilation continued; gastrointestinal decompression initiated
August 20, 2024-14:45	Bronchoscopy confirmed tracheal rupture
August 20, 2024-21:16	Percutaneous abdominal decompression performed
August 21, 2024	Bronchoscopy showed fibrinous exudate at the rupture site
August 26, 2024	Abdominal drainage tube removed
August 26, 2024-20:00	Tracheostomy cannula removed
August 30, 2024	Bronchoscopy revealed complete healing of the tracheal rupture
September 2, 2024	Clinical condition stabilized; patient discharged

At 18:41, complete abdominal CT revealed a substantial amount of intra-abdominal gas. To prevent hemodynamic instability and potential gas embolism, closed abdominal drainage was performed, which resulted in the removal of 1000 mL of gas and a reduction in abdominal distention. On August 21st, repeat bronchoscopy revealed a patent trachea with poorly defined tracheal rings, and a rupture was identified in the membranous part of the trachea, approximately 3.5 cm from the carina. The right lateral wall showed a false lumen and fistula during inspiration, with an unclear view of the distal segment, and the mucosal surface was covered with purulent slough (Figs. 2C-D). Laboratory tests showed elevated white blood cells (11.34×10^9/L) with a neutrophil ratio of 84%, IL-6 at 37.6 pg/mL, and PCT at 0.54 ng/mL, indicating an increase in white blood cells and infection markers. Antibiotic therapy was administered to prevent infection and the possible development of mediastinal abscesses or mediastinitis. No further gas was expelled from the nasogastric or abdominal cavities. Enteral nutritional support was initiated on August 22nd, 2024.

A multidisciplinary consultation within the hospital considered various treatment options for tracheal rupture, including surgical repair, airway stent placement, or conservative management with regular bronchoscopic monitoring of the healing progress. The following key factors were analyzed for this patient: rupture involving the membranous part of the airway without esophageal involvement, ruling out the indication for covered airway stent placement. The rupture was relatively small, measuring approximately 5 mm. A significant amount of gas leaked into the abdominal cavity; however, closed abdominal drainage effectively relieved abdominal distention without causing hemodynamic instability or significant mediastinal compression. A small pneumothorax was observed; however, no evidence of significant pulmonary compression was observed. The patient's vital signs were stable, with no worsening of the pneumoperitoneum or mediastinal emphysema, and no episodes of respiratory distress or hypoxemia. Based on these factors, conservative management was chosen.

Following conservative treatment, repeat chest and abdominal CT on August 26th showed resolution of the pneumothorax and pneumoperitoneum (Fig. 3) with improvement in mediastinal emphysema, although the continuity of the membranous tracheal wall remained disrupted. Bronchoscopy revealed granulation tissue growth on the upper side of the tracheostomy tube and around the distal end, and the mucosa was still disrupted approximately 3.5 cm from the carina. The right lateral wall displayed a false lumen and fistula on inspiration,

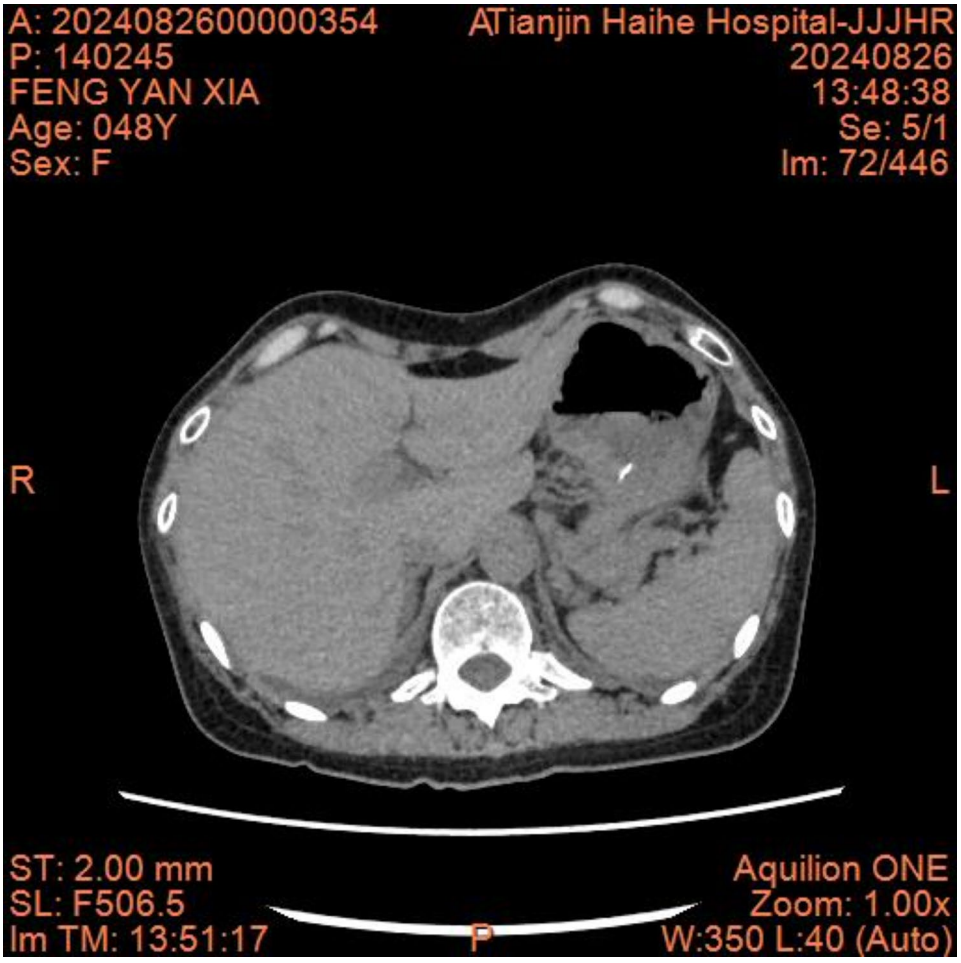

Fig. 3 Repeat abdominal CT revealed no residual free air within the peritoneal or retroperitoneal cavities

with purulent slough covering the mucosal surface. Yellow purulent secretions were observed inside the tracheostomy tube, and the distal end of the tube appeared twisted with significant granulation tissue growth (Fig. 4). The tracheostomy tube was removed at 20:00 on the same day, and the patient remained stable. The patient was able to ambulate freely without signs of wheezing or acute respiratory distress. On August 30th at 14:28, a follow-up bronchoscopy showed no significant laceration of the tracheal membrane (Fig. 5). The patient was discharged in a stable condition after a 10-day hospital stay. Following discharge, the patient's condition remained stable. Sutures were removed during an outpatient follow-up on September 9. However, intermittent wheezing episodes were noted subsequently. One month later, the patient was readmitted owing to another episode of wheezing. Follow-up chest CT revealed notable resorption of the emphysema in all regions compared to previous scans, but the continuity of the membranous tracheal wall remained disrupted without evidence of fistula formation (Fig. 6).

Discussion

Tracheal rupture is one of the most serious complications of endotracheal intubation. Primary clinical manifestations include subcutaneous emphysema, mediastinal emphysema, and pneumothorax [1]. The risk factors for tracheal rupture include anatomical abnormalities of the trachea, advanced age, female sex, prolonged use of nonsteroidal anti-inflammatory drugs, forceful intubation, procedural errors, protruding tracheal guidewires, excessive cuff inflation, improper selection of intubation size, and variations in head and neck positioning during intubation [2].

Pneumoperitoneum after endotracheal intubation is relatively rare. Following tracheal rupture, elevated airway pressure during mechanical ventilation or spontaneous respiration may cause air to leak through the tracheal defect into the mediastinum, resulting in mediastinal emphysema. The air can then spread along anatomical pathways such as the esophageal hiatus, aortic hiatus, or retroperitoneal spaces, ultimately entering the peritoneal cavity and leading to pneumoperitoneum [3, 4]. This mechanism is particularly common in patients

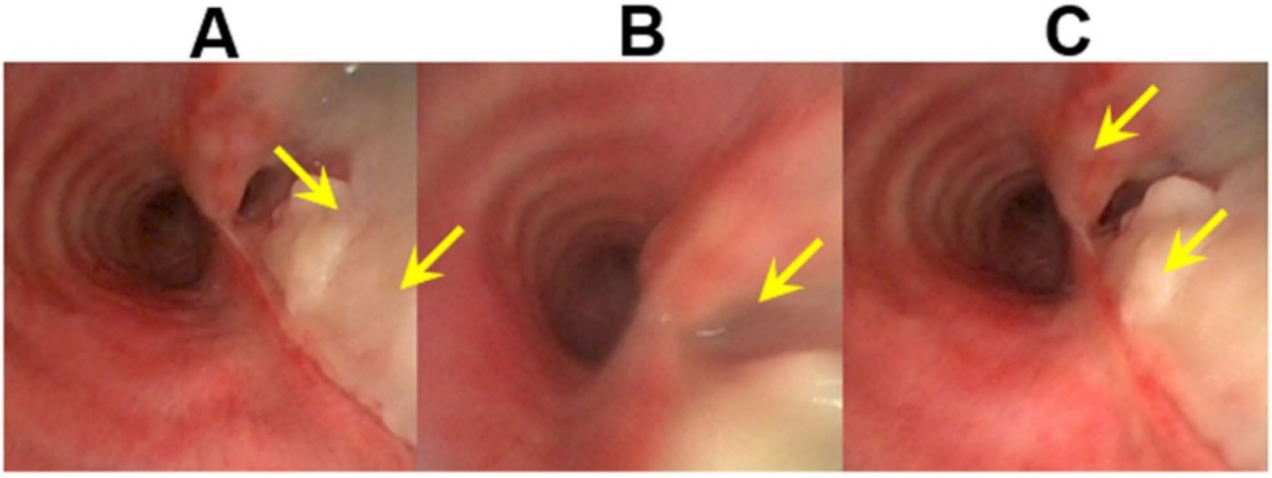

Fig. 4 Tracheobronchoscopic view showing rupture of the tracheal membrane and granulation tissue proliferation. (**A**) The mucosa exhibited discontinuity, beginning approximately 3.5 cm from the carina and extending to the membrane at the end of the intubation tube. (**B**) A false lumen was observed on the right wall during inhalation, with limited visibility of the distal end. (**C**) Increased presence of purulent slough covering the mucosal surface compared to previous observations

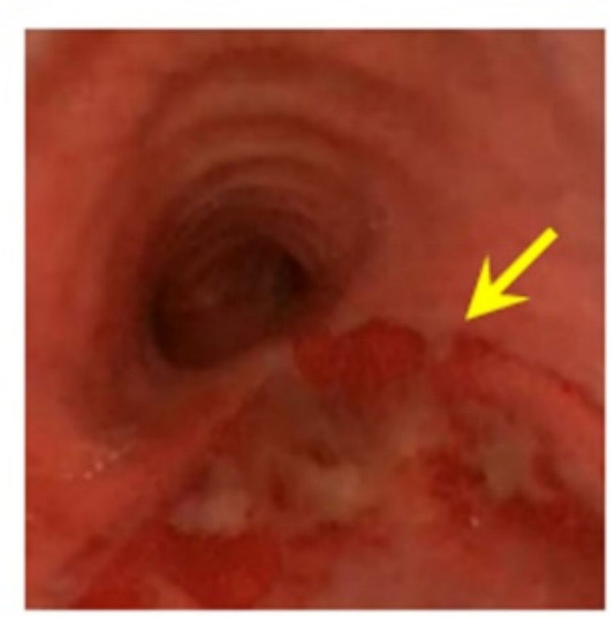

Fig. 5 Tracheobronchoscopic view showing the healed tracheal membrane

receiving mechanical ventilation, especially those with concurrent alveolar leaks or barotrauma [3]. During tracheal intubation, procedural errors may result in perforation of the esophagus or stomach, an event more likely in cases of difficult intubation or multiple attempts, which may also lead to pneumoperitoneum [5, 6]. For instance, unrecognized esophageal intubation can cause excessive gastric insufflation or direct damage to the gastrointestinal wall, resulting in air leakage into the peritoneal cavity [6]. Moreover, mechanical ventilation can exacerbate gastric insufflation, particularly when mask ventilation is used or when a nasogastric tube is not promptly inserted. Increased intragastric pressure may lead to gastric wall ischemia or rupture, allowing air to escape into the peritoneal cavity [5]. Animal studies have shown that when the intratracheal pressure exceeds 40 cmH_2O, interstitial pulmonary emphysema typically occurs. Pneumothorax develops when the pressure exceeds 50 cmH_2O, and subcutaneous emphysema and pneumoperitoneum may occur at pressures above 60 cmH_2O [7–9]. Existing clinical reports have categorized pneumoperitoneum following intubation into several potential pathways: (1) Ruptured alveoli allow air to enter the mediastinum, from where it can migrate along large vessels, the esophagus, or retropleural spaces into the abdominal cavity. In some cases, mediastinal emphysema precedes and induces pneumothorax [10]. (2) Excessive inspiratory pressure causes air to enter the retroperitoneal space and subsequently rupture into the free abdominal cavity, with pneumoperitoneum pre2ceding secondary pneumothorax [10, 11]. (3) Secondary pneumoperitoneum can still occur in cases of esophageal rupture without evident defects in the diaphragm. Case reports have described the development of pneumoperitoneum during nasal cannula oxygen therapy for pneumonia, suggesting the presence of a Macklin effect [12]. This mechanism involves air accumulation around the hilum, traveling along the bronchovascular sheath into the mediastinum, and potentially migrating into the abdominal cavity under certain conditions [13].

In this case, CT imaging from the neck to the abdomen revealed varying degrees of emphysema in the subcutaneous tissues of the neck, entire mediastinum, right thoracic cavity, and greater sac of the peritoneal cavity. Based on the anatomical structures and known mechanisms,

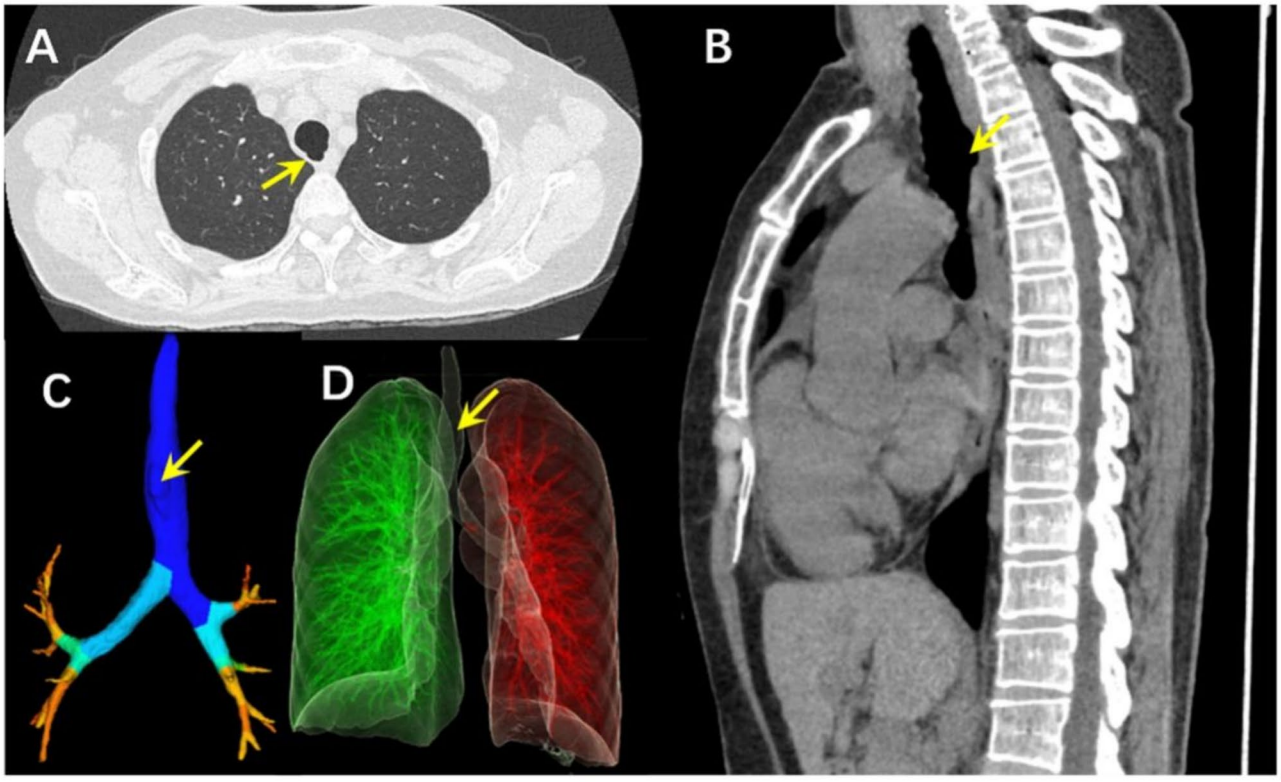

Fig. 6 Chest imaging indicating discontinuity of the tracheal membranous wall. (**A**) Localized posterior bulging of the tracheal membrane. (**B**) Sagittal view showing sac-like posterior bulging of the tracheal membrane at the T3 vertebral level. (**C**-**D**) Reconstructed images showing healed tracheal rupture with posterior bulging of the membranous part

we hypothesized two potential pathways for free gas to migrate from the chest to the abdominal cavities: Free air in the mediastinum could have entered the abdominal cavity through a diaphragmatic rupture, as evidenced by the presence of uniformly distributed gas throughout the mediastinum and visualized in the mediastinal and vertebromediastinal recesses on chest CT. Notably, free gas was observed only in the greater sac of the abdomen, whereas the lesser sac remained unaffected. The diaphragm is composed of denser, central fibrous tissue surrounded by looser muscle tissue [14], and previous reports have hypothesized that excessive intrathoracic pressure could stretch muscle fibers, allowing gas leakage. However, the absence of free gas in the lesser sac indirectly indicates that the gas did not migrate into the abdominal cavity through the loose muscle tissues at the periphery of the diaphragm. Furthermore, no subcutaneous emphysema was observed in the abdominal wall, ruling out this pathway. The most plausible pathway for gas entry into the abdominal cavity involves the passage of high-pressure ventilated air through the tracheal rupture into the mediastinum. The gas then migrates along the esophagus and associated vascular bundles through the esophageal hiatus, where it is encapsulated by the visceral peritoneum. Significant gastric distention suggests a weakened visceral peritoneum in the region around the esophageal hiatus and gastric lesser curvature, ultimately allowing gas to rupture into the greater sac of the peritoneal cavity. Alternatively, a rupture in the diaphragm, which is not clearly discernible on imaging reconstruction, could explain these findings. The patient's CT scans demonstrated non-contiguous interlobular septa in the lower lobes, with occasional alveolar tears and fusions, raising the possibility of ventilator-induced pulmonary barotrauma. According to the Macklin effect, air leaking from the ruptured alveoli into the hilum could have subsequently migrated to the abdominal cavity. Although the specific pathways for this effect remain unknown, this mechanism cannot be excluded as a possible source of abdominal gas accumulation. The patient presented with tracheal rupture following endotracheal intubation, which led to pneumothorax, mediastinal emphysema, and pneumoperitoneum. The patient was a female with a history of severe asthma exacerbation and a high risk of tracheal rupture. During intubation, vocal cord edema and airway narrowing were observed, indicating that the patient's airway anatomy posed an elevated risk of tracheal rupture. Although the presence of pneumoperitoneum secondary to pneumothorax and mediastinal emphysema is clear, the disproportionately large volume

of gas in the abdominal cavity compared to that in the pleural cavity and mediastinum suggests that another pathway, such as a small diaphragmatic or peritoneal rupture, may be involved. The absence of imaging evidence of diaphragmatic defects complicates the complete attribution of extensive pneumoperitoneum to loose connective tissue or the Macklin effect. It is also important to consider that air embolisms and lethal events are more likely to occur in various compartments where gas may leak, such as the mediastinum, subcutaneous neck tissues, and epidural space. Reports have documented cases of air embolism leading to fatal outcomes due to retrograde airflow along the infusion lines in the extremities [15].

When the rupture is proximal, the endotracheal tube can be advanced beyond the site of the rupture, and surgical repair may be necessary under veno-venous extracorporeal membrane oxygenation conditions if needed [13]. Consensus is lacking on whether to opt for conservative management or surgical intervention, and a retrospective single-center study suggested that patients with spontaneous breathing capability, superficial tracheal ruptures, and low risk of mediastinitis and sepsis complications can be considered for conservative management [11]. Surgical intervention is recommended for patients with progressive mediastinal emphysema and respiratory distress or ruptures larger than 2 cm. The right thoracotomy approach is preferred for central and distal tracheal ruptures, a right thoracotomy approach is preferred [12]. Some studies have reported the placement of silicone-covered tracheobronchial stents, surgical repair [14], and conservative management, with healing durations ranging from 10 to 12 days [16, 17]. Reports have shown spontaneous healing of tracheal ruptures up to 4 cm in length with conservative management [8]. Pneumoperitoneum secondary to tracheal rupture is a rare but potentially life-threatening emergency, and its management should be tailored based on the etiology of the rupture, clinical severity, and presence of complications. In cases of tension pneumoperitoneum, immediate needle decompression of the abdomen is essential to relieve intra-abdominal pressure, thereby preventing hemodynamic collapse and abdominal compartment syndrome. This intervention is particularly indicated in patients who rapidly develop pneumoperitoneum following cardiopulmonary resuscitation (CPR) or mechanical ventilation [3, 18]. In the absence of visceral perforation or signs of intra-abdominal infection—such as when air enters the peritoneal cavity via mediastinal dissection following tracheal rupture—conservative treatment may be considered. This includes close monitoring of vital signs, limiting airway pressures through protective lung ventilation strategies, and avoiding high levels of positive end-expiratory pressure (PEEP) to reduce further air leakage [19, 20]. Additionally, aspiration or regurgitation of gastric contents during intubation may indirectly increase the risk of gastrointestinal perforation. In the present case, although the patient exhibited gastrointestinal distension, there were no signs of peritoneal irritation. Pneumoperitoneum was presumed to result from air dissection into the peritoneal cavity secondary to tracheal rupture. After percutaneous abdominal decompression, the intra-abdominal free air resolved and the patient's abdominal distension was alleviated.

Treatment decisions should be individualized according to the patient's condition. In this case, the rupture was approximately 5 mm in length, and bronchoscopy performed on day 10 of conservative treatment revealed healing of the rupture. A follow-up chest CT one month later showed significant resolution of emphysema in all regions, although the continuity of the tracheal membrane remained disrupted, and the newly formed tissue was structurally fragile. This indicates a higher risk of airway rupture during future intubation, and further monitoring of sequelae is necessary following conservative treatment. After tracheal rupture, potential long-term complications include tracheal stenosis and tracheoesophageal fistula, which may manifest as chronic cough, dyspnea, or recurrent respiratory infections. Long-term follow-up with pulmonary function testing and airway imaging is therefore essential. Pneumoperitoneum itself may irritate the peritoneum, potentially leading to adhesions and an increased risk of long-term intestinal obstruction; however, conservative management carries a lower risk of such complications compared to surgical intervention. In the present case, the patient was clinically stable and ambulatory at the time of discharge. She was advised to continue preventive measures at home, including avoiding upper respiratory infections and allergen exposure to prevent asthma exacerbations, monitoring for signs of aspiration during feeding, and attending regular follow-up visits.

Conclusion

In summary, tracheal rupture is a rare but serious complication that can lead to the entry of air into the thoracic and abdominal cavities, resulting in a variety of clinical manifestations. Tracheal rupture and subsequent pneumoperitoneum should be considered as differential diagnoses in patients presenting with abdominal distension following tracheal intubation. Prompt diagnosis and appropriate conservative management can lead to favorable outcomes. However, healthcare professionals must remain vigilant regarding the potential complications associated with tracheal intubation, particularly in patients with underlying respiratory conditions or structural abnormalities. Regular follow-up and close monitoring are essential to detect and address any potential

sequelae promptly, thereby ensuring optimal patient outcomes.

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Author contributions
H.W and X.L contributed to manuscript preparation. The corresponding author, W.Y, L.L and C.C conceptualized the presented idea and is responsible for this manuscript. L.S, C.G, H.Y, J.Z and S.Z were the attending physicians involved in data collection. Z.X and X.W supervised the manuscript. All authors read and approved the final version of the manuscript.

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Data availability
The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declarations

Competing interests
The authors declare no competing interests.
Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor of this journal.

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