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RESEARCH ARTICLE

"THE LEAKY AIRWAY": SPONTANEOUS TRACHEO BRONCHIAL TREE INJURY (TBI) - CASE SERIES

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ABSTRACT

Spontaneous tracheal rupture is one of the rare life threatening conditions. Tracheal lacerations are generally secondary to cervical or chest trauma or occurring as a complication of endotracheal intubation. Spontaneous trachea-bronchial tree injury (TBI) are potential killers if not diagnosed early and treated adequately. We hereby report 2 case reports of spontaneous TBI, one secondary to a lower respiratory tract infection (LRTI) and the other secondary to COPD with long term steroid use. There are only less than 5 case reports till date, published on spontaneous TBI.

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INTRODUCTION

Tracheo-bronchial tree injuries are rare, but potentially life-threatening, in both adults and children. They are well-known sequel of massive blunt or penetrating injuries of the neck or chest.(1,2) and also may occur as a rare complication of endotracheal intubation.(3–5) Spontaneous tracheal ruptures are extremely uncommon, and few cases have been reported in literature. We report 2 cases of trachea-bronchial tree injury secondary to lower respiratory tract infection and COPD with steroid use worsened secondary to intubation. Challenges of airway management and stabilisation are dealt with in these case reports.

CASE REPORT

CASE 1: A 23 year old female came with history of fever and cough with expectoration since 3 days and associated breathlessness since 1 day. She also complained of sudden onset swelling of chest and face, the same day. Fever was moderate grade with chills not subsiding with oral medication associated with expectorant cough and dyspnoea, which acutely worsened. No history of trauma.

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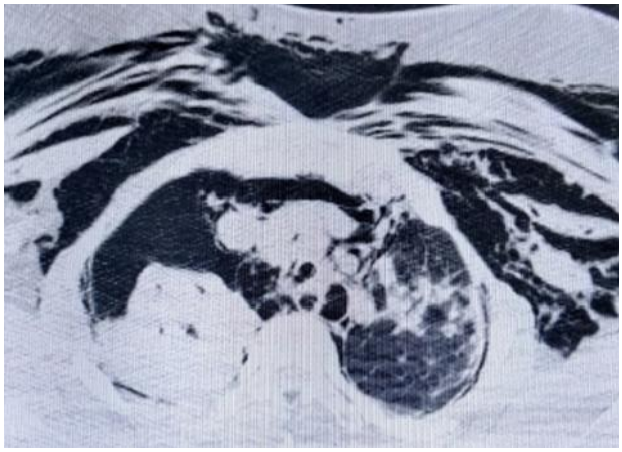
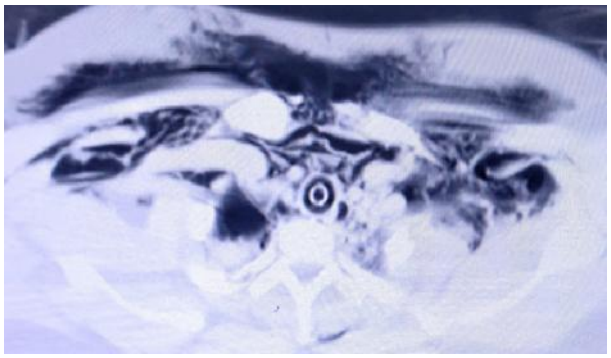
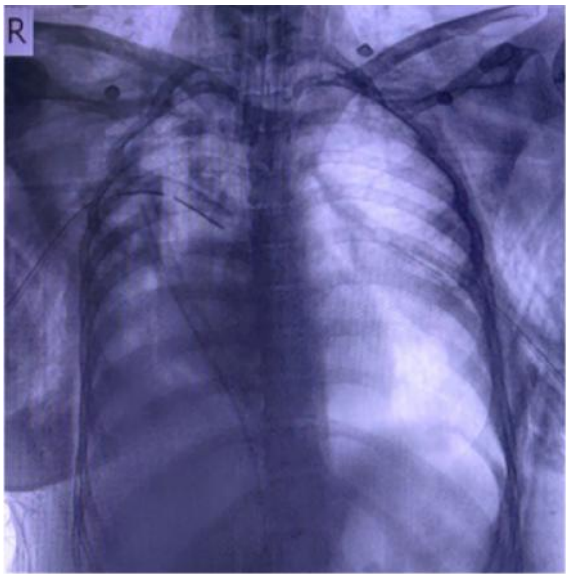
Patient had no comorbidities at the time of presentation The patient was referred from another hospital on oxygen mask with sudden onset swelling of her face and chest and both upper limbs since the last 3 hours. On arrival her pulse rate was 130/min, BP recording was 90/60 mm of Hg, RR was 44/min, room air saturation was 78% and on oxygen it was 84% and she was afebrile. General physical exam revealed extensive subcutaneous emphysema with crepitus over face, chest wall, extending to bilateral upper limbs. A possibility of trachea bronchial tree injury (TBI) was considered at this point. Anaphylaxis was another consideration but subcutaneous crepitation with sparing of lips pointed more in favour of TBI. The patient was intubated under direct vision with smooth and gentle passage of the tube. Measures were ensured for anticipated difficult airway and front of neck access (FONA) team was standing by. After intubation her vitals were HR of 110/min, BP: 80/60mm Hg, Spo2 of 88% on a ventilator. Bilateral inter-costal drainage (ICD) was placed in view of persistent hypoxia, shock and presence of extensive subcutaneous emphysema and the BP picked up to 90/60mm Hg with a saturation of 94%. A CT neck with thorax was done, as shown below in Figure 1 and 2.

Findings: Extensive subcutaneous emphysema, pneumo-mediastinum, sub-segmental collapse of the left upper lobe, right sided pneumothorax and a wall irregularity in the trachea 1 cm above carina-suggestive of tracheal rupture.





On reassessing the patient, post CT scan there was a BP drop to 80/60 mm Hg. As the patient did not improve significantly an anterior mediastinal ICD was placed by Cardio-thoracic vascular surgeon (CTVS) to decompress the heart. She improved vitally with a HR of 100/min, BP of 120/90 mm Hg and Spo2 improving to 97%. Chest x-ray with bilateral ICD and mediastinal tube as shown below in Figure 3*:* Owing to the short history she was started on oseltamivir but Bronchoscopy and lavage grew MRSA and septate fungal elements and was started on atibiotics and anti-fungal injections. Bronchoscopy revealed showing membranous lesion over the anterior aspect of trachea 1-2 cm above carina. Right main bronchus had blood clots and the left main bronchus showed nodular infiltration. Patient was managed with conservative therapy for the tracheal rent with antibiotics, anti-fungal, mechanical ventilation and inotropic support for 7 days after which she succumbed to severe sepsis.

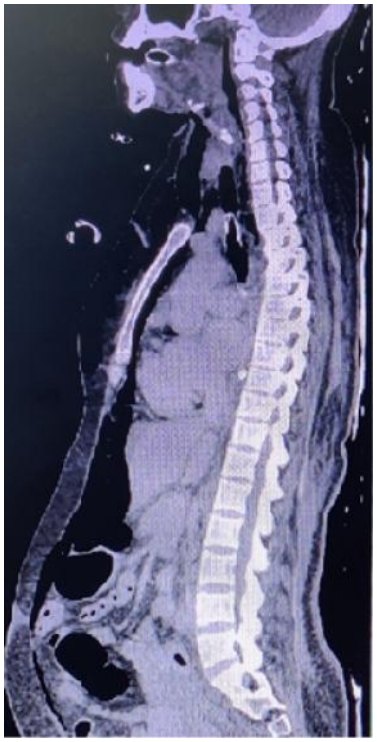


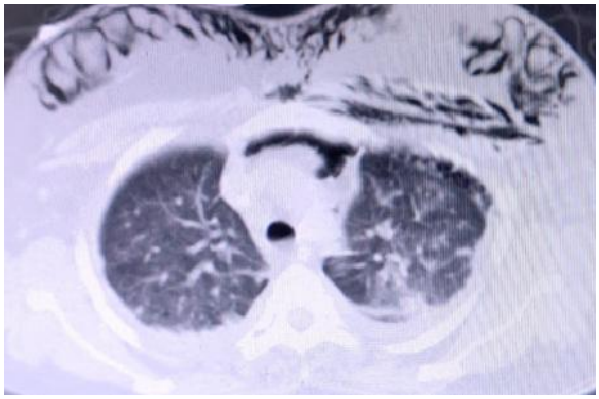
Our final diagnosis was:

Bilateral pneumothorax and pneumomediastinum with obstructive shock s/p spontaneous TBI secondary to LRTI

CASE 2: A 42 year old obese female, known case of Bronchial asthma on long term steroids presented with breathing difficulty since 1 week, worsened acutely since 3-4 hours with altered sensorium since 1 day. She was previously on inhaled steroids for a period of 10 years. On arrival airway was threatened with poor respiratory efforts on BMV with a HR of 120/min, BP of 70 mm systolic and saturation of 70% RA. Patient was started on 100% oxygen via BMV and the saturation improved to 80%. Bedside ABG showed severe respiratory and lactic acidosis with a pH of 7.021, pCO2 of 99.7 and pO2 of 70.7 and HCO3 of 22.4 with lactates of 16.

Patient was intubated in view of hypercapnoea and hypoxia with repiratory and lactic acidosis. Intubation was easy and uneventful. Post intubation vitally HR was 108/min, BP was 90/60mm Hg and sPo2 was 88%. On attempting POCUS (point of care ultrasonography) and FOCUS (focussed cardiac ultrasonography) bedside, only hyperechogenecity was visible with inability to see structures below which raised the suspicion of subcutaneous emphysema. A CT thorax was done which confirmed subcutaneous emphysema (which was missed clinically due to the obesity), posterior tracheal wall irregularity at level of T3 with air column posteriorly suggestive of tracheal injury, and bilateral pneumothorax left more than right with pneumo-mediastinum and ET tube in place as shown in Figure 4 & 5 below: Therapeutically bilateral ICD was placed with improvement in the vitals. Patient was started on antibiotics, inotropic support and mechanical ventilation. However the patient succumbed to the overwhelming sepsis on day 4 of hospital stay. To conclude we had a diagnosis of Type 2 respiratory failure with bilateral pneumothorax and pneumomediastinum due to spontaneous TBI probably secondary to long term steroid use and bronchial asthma





DISCUSSION

Acute rupture of the trachea is a rare and life-threatening injury that may be fatal if not treated immediately. Localization of the rupture, its nature and extent, the mechanism, age and pre-existing risk factors have to be considered in order to define the optimal therapeutic option (4). The causes of tracheal rupture are trauma, intubation and spontaneous. Spontaneous rupture of the trachea is an unusual condition. It occurs after sudden increase of air pressure in a weakened upper airway (3). Mediastinal and subcutaneous emphysema of sudden onset after an effort of retching or coughing is suggestive for a spontaneous tear in the membranous part of the trachea. The patient usually presents with dyspnoea, cyanosis and severe respiratory distress (5). Trauma presents with history of crushing injuries to the chest with associated paradoxical respiratory movements and subcutaneous and neck emphysema (4).

Few of the causes of spontaneous TBI are mentioned below:

- Congenital tracheal diverticulum
- Tracheal bronchus
- Tracheal distortion: Airway or mediastinal neoplasms
- Age > 65 y
- Female sex
- Tracheal inflammation
- Use of inhaled corticosteroids

Other important risk factors are age, poor medical history, short stature, chronic obstructive pulmonary disease, tracheomalacia (5). Also, weakness of the tracheal wall due to continuous corticosteroid treatment was reported as a possible risk factor (2-4).

DIAGNOSIS: Diagnosis of tracheal rupture is mainly based on clinical signs (sudden onset of the pneumomediastinum and subcutaneous emphysema of the upper thorax diffusing to the neck). It can be confirmed on X-ray chest. Pneumothorax occurs if the tracheal air leak communicates with the pleural space due to a tear of the mediastinal pleura (11). Computerised tomography with the appropriated window settings allows direct visualization of the tracheal injury in mostof the cases (9). Bronchoscopy is the gold standard toolfor measuring the size and extent of trachea-bronchial tree injuries. However small tears can be missed on bronchoscopy too (9).

TREATMENT: After one-look laryngoscopy, a single attempt to gently pass the endotracheal tube is made under direct vision. Standby FONA (front of neck access) team and bronchoscopy are failsafe strategies if intubation is not feasible (8,9). Surgery is the preferred treatment for patients with acute tracheal rupture (8). However conservative management is an alternative for patients unsuitable for surgery and small tears, with a reasonable chance of spontaneous sealing and uneventful recovery (8, 12, 13, 14). According to the majority of authors, early surgical repair in healthy tissues remain the therapy of choice (1, 2, 7, 9, 13). According to Massard (10), when deciding for conservative treatment and in case of its failure, one should be aware that a later operation is not a valuable alternative: the chances of success of a delayed repair are jeopardized by the patient's poor general condition and by local infection or inflammation (17). For small lacerations less than a centimetre, conservative treatment is the most viable option (2, 8, 12). The proposed medical treatment consists of humidified air, broad-spectrum antibiotics and chest physiotherapy. Though different approaches are mentioned about treatment modalities its up to the clinician to choose the most feasible option with securing of airway taking preference over others.

Conclusion

We report two cases of trachea-bronchial tree injury, one secondary to an LRTI and the other secondary to bronchial asthma with prolonged steroid use. Both were managed conservatively as the rents were small, but both patients did not survive. Spontaneous TBI are life-threatening, under reported, rare and difficult to diagnose and has a high mortality rate.

Footnotes

There is no conflict of interests.

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