# Buried in Trapped Air: Tension Pneumothorax, Massive Subcutaneous Emphysema and the Battle for Airway Control

Parul Issar\*

Yatharth Superspeciality Hospital, Greater Noida, India.

#### \*Corresponding Author:

Parul Issar, MD. Yatharth Superspeciality Hospital, Sehdeo Hospital, Block A, Ansal Golf Links 1, Greater Noida, Uttar Pradesh 201315, India. Email: parulissar@gmail.com.

#### **ABSTRACT**

Primary Spontaneous Tension pneumothorax (STP) is a life-threatening medical emergency. The presence of extensive subcutaneous emphysema (SCE) can further aggravate the respiratory distress and make the airway management even more challenging in the ER. The situation can become even worse when such a patient needs cardiopulmonary resuscitation on arrival.

We report an unusual case of a 55-year-old man who presented in the ER with a history of sudden onset of respiratory distress while taking a bath. The patient was brought to the ER in a state of gasping, hypoxia, with swelling over the face and chest, and shock. We made a clinical diagnosis of tension pneumothorax (TP), and bilateral needle decompression of the pneumothoraces was done. But it was not sufficient, and he landed in cardiac arrest. The CPR and the airway management were extremely difficult due to airway edema. He was quickly tracheostomised in the ER, and a surgical airway saved his life.

Primary spontaneous pneumothorax is an uncommon condition, but it can rarely end up in tension pneumothorax, and accompanying subcutaneous emphysema can make the management further challenging. Airway skills of the ER team are important in saving such patients' lives.

Keywords: Tension pneumothorax, massive subcutaneous emphysema, CPR, difficult intubation.

### INTRODUCTION

Primary Spontaneous Tension pneumothorax (STP) is a life-threatening TP presenting without any known lung disease. It can be managed with emergency needle decompression and other supportive treatment. Few patients with TP may also have massive accumulation of air in deep tissues of the neck (SCE), compromising airway and venous return. Airway management in such patients can be very challenging.

Our patient in discussion here had a similar life-threatening combination of STP and severe extensive SCE. He was a 55-year-old man who developed cardiac arrest due to tension pneumothorax (TP) and extensive subcutaneous emphysema (SCE). During the CPR, the patient

was in a "cannot intubate, cannot ventilate" (CICO) situation. An emergency percutaneous tracheostomy was done, and ROSC was finally achieved. It is a rare combination of life-threatening primary SPT and severe SCE successfully managed with surgical Front of the Neck Airway (FONA).

#### **CASE ILLUSTRATION**

A 55-year-old male patient with no past medical and surgical history presented in the ER with an acute onset of breathing difficulty of around 2 hours duration, followed by swelling over the neck, chest, and face. Dyspnoea soon progressed to distress with difficulty in speaking and swallowing. He was a heavy smoker.

Quick clinical examination showed the patient was afebrile, drowsy, restless, dyspnoeic, and gasping for air. His heart rate was 156 beats per minute, NIBP was 65/43 mmHg, and oxygen saturation was 67% on room air at the time of arrival. He had subcutaneous emphysema all over his face, neck, anterior chest, and whole abdomen (Figures 1 and 2). On auscultation, there was crepitus all over the chest and abdomen. A clinical diagnosis of primary SPT with massive SCE was made. Oxygen support, fluid resuscitation, and inotropes were initiated with simultaneous emergency needle decompression of bilateral hemithoraces in the 5th intercostal space in the anterior axillary line. SpO2 levels improved to 81% but his respiratory distress continued. Soon, the patient became cyanotic, unresponsive, and pulseless. Cardiopulmonary resuscitation (CPR) was immediately started. Bag-mask ventilation during the CPR was not effective due to the severe upper airway emphysema. Oral and nasopharyngeal airways also failed to improve the ventilation. An attempt at orotracheal intubation was made, but due to extreme emphysema of the upper airway, laryngoscopy failed. Rescue airway with SAD was the next step. Proseal LMA was inserted, and finally, ROSC was achieved. But due to extensive chest wall emphysema and perhaps air infiltration around the glottis, ventilation was still inadequate, and target EtCO2 and SpO2 levels could not be attained. Hoping for a better decompression of pneumothoraces, bilateral intercostal drainage tubes were inserted and connected to the underwater seal. But this also did not improve the ventilation. Video laryngoscopy was tried, which showed excessive infiltration of air in the glottic structures, leaving an extremely narrowed glottic chink. Further attempts for intubation were therefore deferred, and a plan for an elective tracheostomy was made. To continue ventilation, Proseal LMA was reinserted, but unfortunately, this time it didn't fit well, and air started leaking, perhaps due to progressively increasing airway emphysema and high airway pressures. Consequently, SpO2 levels started dropping rapidly. It was a CICO situation with impending cardiac arrest; an emergency percutaneous tracheostomy was done

in the ER with the Seldinger technique. A size 7.5 mm ID tracheostomy tube was inserted, and bag ventilation was established. SpO2 levels quickly improved to >90%. The time taken to secure the airway was not more than 90 seconds.

The patient was then shifted to the Intensive Care Unit for further management. CT scan of his thorax showed bilateral pneumothoraxes with emphysematous lungs and multiple bullae with extensive subcutaneous emphysema extending from the neck above to the thighs below (**Figures 3,4,5**). Upper airway structures



**Figure 1.** Extensive subcutaneous emphysema over the face and the eyelids at the time of admission



Figure 2. Percutaneous tracheostomy tube in situ



**Figure 3.** CT thorax showing extensive air collection between the layers of the chest wall. Bilateral pneumothoraces with right mediastinal shift

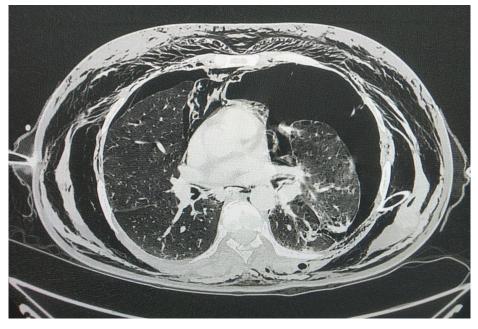


Figure 4. Bilateral Intercostal drainage tubes in place



Figure 5. Bullous emphysematous changes are visible in both lungs



Figure 6. Completely resolved subcutaneous emphysema. Post tracheal decannulation

were also emphysematous. The patient was discharged home in a stable condition without any neurological deficit after bilateral pleurodesis (**Figure 6**).

## **DISCUSSION**

We aim to draw attention to a rare primary SPT that presented with a deadly combination

of tension pneumothorax (TP) and severe subcutaneous emphysema (SCE). Simple needle thoracostomy initially followed by chest tube insertion was not sufficient due to the concomitant airway obstruction due by severe SCE. Worsening hypoxia and progressive CO2 retention resulted in cardiac arrest. Dealing with a CICO situation, a challenging but quick

percutaneous tracheostomy was done, and ventilation was re-established.

Pneumothorax, in the absence of a known underlying etiology, is called primary spontaneous pneumothorax (PSP).<sup>4</sup> PSP is less common but has been associated with lung bullae or blebs in imaging studies.<sup>5</sup> Subcutaneous emphysema is a well-recognized complication of pneumothorax. Rarely, the combination may become lethal, as happened in our patient.<sup>6</sup> Compression of the superior vena cava and lung compression aggravate the shock and hypoxia. This can lead to cardiovascular collapse.<sup>7</sup>

Subcutaneous emphysema (SCE) is the collection of air or gas beneath the skin, recognized as crepitus, which feels like 'walking in the snow'. The incidence ranges from 0.43% to 2.34%. In a recent study done on 405 trauma patients, all 24 patients presenting with subcutaneous emphysema were found to have an underlying pneumothorax on CXR.

Isolated SCE is mostly a self-limiting condition, though there are case reports of deaths due to SCE alone because of precipitant ventilatory failure. 10,11 In their series of patients with pneumothorax, Jones et al found an increase in mortality with SCE (16% versus 5% in cases where SCE was not present). 12 SCE is thought to arise through the 'Macklin Effect'13, which causes air leak into the loose connective tissue around the pulmonary vessels. This air follows the fascial planes and leaks into the soft tissues of the upper torso. Though the involvement of the hypopharynx is sufficient enough to cause airway obstruction is extremely rare, but when present, it can be rapidly fatal.<sup>14</sup> This could be the main reason behind the near-complete respiratory obstruction and CICO in our patient, also.

Tension pneumothorax is a red flag in the ER. Air escaping after large-bore needle insertion in the 4<sup>th</sup> or 5th intercostal space (ICS) in the anterior axillary line confirms the diagnosis of TP. The initial assessment involves a chest radiograph (CXR) to confirm the diagnosis. A chest computed tomography (CT) can be done if the diagnosis is unclear on CXR, though it is not recommended for routine use.

Ultrasound is 94% sensitive and 100% specific with a skilled operator. POCUS may

be useful in unstable patients and shows the absence of "lung sliding" and the presence of a "lung point". 15,16

Management depends on the patient's hemodynamic stability. Needle thoracostomy followed by the CTT (Chest Tube thoracostomy) is the standard treatment. Extensive SCE needs infraclavicular incisions, placement of 14G fenestrated subcutaneous catheters, <sup>16</sup> and insertion of subcutaneous drains, tracheostomy, etc. When TP and SCE are present together, immediate intervention to secure the airway with simultaneous release of the pneumothorax is required.

The present case describes a CICO situation during CPR due to extensive SCE and primary SPT in an otherwise asymptomatic patient, arriving in the ER with severe airway obstruction and shock. The severity of SCE caused direct constriction of the proximal airway with severe airflow obstruction. He also had progressive hemodynamic failure associated with the underlying tension pneumothorax. Immediate placement of a thoracostomy tube bilaterally should likely have mitigated the shock and the respiratory distress. But unfortunately, airway obstruction rapidly progressed, causing hypoxia severe enough to result in cardiac arrest. The priority shifted to relieving the airway obstruction caused by the massive subcutaneous air. Conventional airway management was unsuccessful because of the anatomical distortion due to subcutaneous swelling narrowing down the air passages, the progressive respiratory obstruction, and clinical (cardiovascular) instability. Tracheostomy was risky as the anterior neck was emphysematous, and it was difficult to determine landmarks, in addition to the possibility of fatal bleeding and the difficulty of dissection. But this patient was safely and successfully managed by an emergency percutaneous tracheostomy. To prevent the recurrence and in view of the severe first presentation, VATS and pleurodesis were done. Patients were discharged home after tracheal decannulation in stable condition.

The Difficult Airway Algorithm is based on the guidelines provided by DAS (Difficult Airway Society), UK, as a strategy to manage unanticipated difficulty with tracheal intubation in a critical situation.<sup>17</sup> It says that supra-glottic airway devices (SAD) should be used if the tracheal intubation fails.<sup>18</sup> When both tracheal intubation and supra-glottic airway devices have failed, waking the patient is the default option, which was not possible in our patient. If at this stage, face-mask oxygenation is impossible in the presence of muscle relaxation (CICO), cricothyroidotomy should follow immediately.

We managed our patient as per these guidelines, and the percutaneous tracheostomy proved lifesaving for him. Short procedure time helped in preventing any neurological insult to the patient.

#### CONCLUSION

Practitioners should be very vigilant while treating TP when accompanied by extensive SCE, which might quickly progress even after needle decompression. Another noteworthy point is that in a patient with massive subcutaneous emphysema, a previously patent airway can, rarely, later become obstructed. In such a critically difficult airway situation, DAS guidelines provide a strategy to manage difficult tracheal intubation. Point to remember- "Do not wait for life-threatening hypoxemia before transitioning to FONA". FONA (front of neck airway) can be lifesaving in such situations.

#### **CONFLICT OF INTEREST**

The authors declare there is no conflict of interest.

#### REFERENCES

- Selby CD, Sudlow MF. Deficiencies in the management of spontaneous pneumothoraces. Scot Med J 1994;39:75-6.
- Kainat A, Ain N, Mukhtar N, Fitzpatrick M. Tension pneumothorax and subcutaneous emphysema - A rare cause of dysphagia in a patient with Marfan syndrome. Am J Respir Crit Care Med. 2023;207:A1540.
- 3. Sahoo NK, Singh S, Bhandari A. Early postoperative malignant subcutaneous emphysema: Report and review. J Maxillofac Oral Surg. 2017;16(1):85–9.
- Jalota Sahota R, Sayad E. StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022. Tension pneumothorax. [Updated 2022 Nov 28]

- Lien WC, Yuan A, Tsai KC, et al. Primary spontaneous pneumothorax with clinical manifestation mimicking acute cholecystitis. J Emerg Med. 2004;26(3):354–6. doi: 10.1016/j.jemermed.2003.12.017.
- 6. Noppen M, De Keukeleire T. Pneumothorax. Respiration. 2008;76:121–7.
- Nelson D, Porta C, Satterly S, Blair K, Johnson E, Inaba K, Martin M. Physiology and cardiovascular effect of severe tension pneumothorax in a porcine model. J Surg Res. 2013;184(1):450-7.
- 8. Aghajanzadeh M, Dehnadi A, Ebrahimi H, Karkan MF, Jahromi SK, Maafi AA, Gilda Aghajanzadeh. Classification and management of subcutaneous emphysema: a 10 year experience. Indian J Surg. 2015;77(2):673–7.
- Ball CG, Ranson K, Dente CJ, et al. Clinical predictors of occult pneumothoraces in severely injured blunt polytrauma patients: a prospective observational study. Injury. 2008;40(1):44–7. doi: 10.1016/j. injury.2008.07.015.
- 10. Conetta R, Barman AA, Iakovou C, Masakayan RJ. Acute ventilatory failure from massive subcutaneous emphysema. Chest. 1993;10:978–80.
- 11. Murayama S, Gibo S. Spontaneous pneumomediastinum and Macklin effect: Overview and appearance on computed tomography. World J W J R Radiol. 2014;6(11):850–4.
- 12. Dubin I, Gelber M, Schattner A. Massive spontaneous subcutaneous emphysema. Am J Med. 2016;129(12):e337–8.
- Gibney RT, Finnegan B, FitzGerald MX, V Lynch. Upper airway obstruction caused by massive subcutaneous emphysema. Intensive Care Medicine 1984; 10: 43-4.
- 14. Light RW. Pleural diseases. Dis Mon. 1992;38(5):266-
- 15. Beck PL, Heitman SJ, Mody CH. Simple construction of a subcutaneous catheter for treatment of severe subcutaneous emphysema. Chest. 2002;121:647–9.
- 16. Higgs A, McGrath BA, Goddard C, Rangasami J, Suntharalingam G, Gale R, Cook TM. Difficult airway society; Intensive care society; Faculty of Intensive Care Medicine; Royal College of Anaesthetists Guidelines for the management of tracheal intubation in critically ill adults. British Journal of Anaesthesia. 2018;120(2):323-52.
- 17. Henry M, Arnold T, Harvey J. Pleural diseases group, standards of care committee. British Thoracic Society. BTS guidelines for the management of spontaneous pneumothorax. Thorax. 2003; 58 Suppl 2(Suppl 2): 39-52.
- 18. Donahue DM, Wright CD, Viale G, Mathisen DJ. Resection of pulmonary blebs and pleurodesis for spontaneous pneumothorax. Chest. 1993;104:1767–9. [IIb].