

CASE REPORT

Necrotising Tracheitis Resulting in Complete Laryngotracheal Stenosis: A Life-Threatening Complication of Tracheal Intubation

MARYAM JAMILAH MOHAMAD SHAHRIMAN, ZARA NASSERI*

Department of Otorhinolaryngology – Head and Neck Surgery, Hospital Canselor Tuanku Muhriz, Universiti Kebangsaan Malaysia, 56000 Cheras, Kuala Lumpur, Malaysia

**Correspondence: zaranasserivork@gmail.com; Tel: +6012 4841322*

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ABSTRAK

Dalam situasi kecemasan, intubasi endotrakeal merupakan langkah penting untuk melindungi saluran pernafasan. Walau bagaimanapun, salah satu komplikasi yang jarang berlaku tetapi sangat membimbangkan berkaitan dengan intubasi endotrakea ialah trakeitis nekrotik. Keadaan yang mengancam nyawa ini boleh menyebabkan komplikasi yang serius, termasuk saluran pernafasan tersumbat, perforasi trakea dan akhirnya kematian akibat gangguan ventilasi. Pertimbangan terhadap komorbiditi pesakit, seperti diabetes mellitus, adalah amat penting. Keadaan imuniti yang terjejas meningkatkan risiko komplikasi yang teruk secara signifikan, di mana kumpulan ini cenderung mengalami kesan klinikal yang lebih buruk berbanding individu yang lebih sihat.

Kata kunci: *Intubasi; laringotrakeal; nekrotik; stenosis; trakeitis*

ABSTRACT

In emergency situations, endotracheal intubation serves as a crucial measure for airway protection. However, one of the rare but highly concerning complications associated with endotracheal intubation is necrotising tracheitis. This life-threatening condition can result in severe outcomes, including airway obstruction, tracheal perforation and ultimately, death due to compromised ventilation. Considering a patient's comorbidities, such as diabetes mellitus, is of paramount importance. An immunocompromised state significantly heightens the risk of severe complications, with this group experiencing worse outcomes when compared to healthier individuals.

Keywords: Intubation; laryngotracheal; necrotising; stenosis; tracheitis

INTRODUCTION

Endotracheal intubation is performed for airway protection in emergency cases and electively during anesthesia. One of the rare complications of endotracheal intubation is necrotising tracheitis. It is a life-threatening condition where it can lead to airway obstruction, tracheal perforation and death due to poor ventilation (Al-Qadi et al. 2013). In this report, we presented a case that we

encountered, highlighting necrotising tracheitis as a life-threatening complication following tracheal intubation.

CASE REPORT

A 43-years-old man had a history of motor vehicle accident and sustained severe traumatic brain injury secondary to diffuse axonal injury.

He was intubated for airway protection in view of his Glasgow Coma Scale (GCS) was scored 4 with the eye-opening component scored 1, verbal response component scored 2 and motor response component scored 1 (E1V2M1). He was intubated with a cuffed endotracheal tube size 7.5 under direct vision using a video laryngoscope in a single attempt and the tube was anchored at 22 cm. He was extubated 5 days later. During this admission, he had hospital acquired infection with *Streptococcus mitis* bacteremia. He completed culture directed intravenous benzylpenicillin for 14 days and was discharged after completing the antibiotic. He also had poorly controlled diabetes mellitus (DM).

One month later, he presented to the Emergency Department with upper respiratory tract infection. It was associated with worsening exertional noisy breathing and shortness of breath for 1 week. He was unable to expectorate his sputum. Otherwise, he has no other significant symptoms. On examination, he was sitting in a tripod position and there was audible expiratory stridor. His vitals were within normal range. His oxygen saturation was 97% under room air. The respiratory examination was unremarkable. Bedside flexiblescopy

(FNPLS) revealed thick greenish mucopus at the subglottis and slough within necrotic tracheal mucosa. Other supraglottic structures were normal (Figure 1a). Bilateral vocal cords were mobile and appeared normal. Then, we proceeded with direct laryngoscopy, examination under anesthesia and removal of slough and necrotic tissue. Intraoperatively, there was necrotic cartilage in the trachea, superiorly 2 cm from the free edge of the vocal cord (Figure 1b). The necrotic segment was 3 cm and the distance from inferior edge of necrotic segment to carina was 5.5 cm. It was multisegmented. It was likely that the first and second tracheal rings were involved. In the same setting after the removal of the necrotic tissue, we performed tracheostomy and bronchoscopy. Bronchoscopy done was unremarkable up to level of inferior lobar bronchi bilaterally.

Intraoperative tissue cultures were mixed growth. Histopathological examination of the tracheal tissue reported that there were fragments of benign hyaline cartilage displaying areas of dystrophic calcification. There was mild superficial infiltration by neutrophils admixed with fibrin. Parts of the superficial cartilage appeared pale and unhealthy with loss of

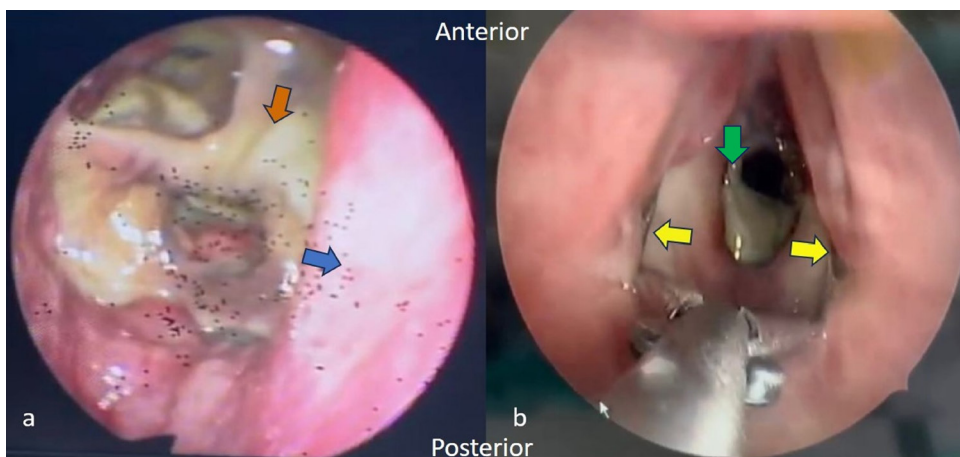


FIGURE 1: (a) Bedside FNPLS of subglottic view, revealed thick greenish mucopus at the subglottis and slough within necrotic tracheal mucosa (orange arrow), blue arrow showed right wall subglottic area; (b) Direct laryngoscopy with laryngeal spreader lateralising bilateral true cords (yellow arrow) showing that there was a necrotic segment of the trachea (green arrow) which then removed.

chondrocytes within the lacunae. However, there was no granuloma to suggest tuberculosis and no evidence of malignancy seen. Apart from that, his poorly controlled DM was kept within acceptable range during the admission. He subsequently completed 1 week of intravenous antibiotic and was discharged home with a double lumen, uncuffed tracheostomy tube.

About 3 weeks later, he was admitted electively for upper airway assessment. On further history, he was aphonic for 2 weeks prior to this admission. Otherwise, no other significant symptoms. His vitals were stable and his double lumen tracheostomy tube was patent. He underwent direct laryngoscopy and examination under anesthesia (Figure 2a & 2b). Then we proceeded with injection of triamcinolone acetonide and bougie dilatation up until size 40 Fr. Immediately post operatively, the stenosis improved to Cotton Myer grade II (Figure 2c). The tracheal tissue that was sent for culture intraoperatively, grew *Streptococcus mitis*. Subsequently patient was discharged with same size double lumen tracheostomy tube with follow up. However, restenosis occurred where patient subsequently underwent partial cricotracheal resection and anastomosis later on. In preparation for this case report, we had

secured the patient's consent to include images directly related to them.

DISCUSSION

Endotracheal intubation is a common procedure. It comes with multiple risks of complication. Even in a short period of intubation, it may cause various degrees of damage to the airway including laryngeal edema and ulceration of the airway mucosa (Al-Qadi et al. 2013; Benjamin & Holinger 2008).

Tracheal infection can be manifested as focal or necrotising tracheitis. Necrotising tracheitis is a rare and severe inflammation of the trachea, which forms a pseudo membrane in the airway and can be fatal (Louie et al. 2022). Post extubation necrotising tracheitis is rare and frequently underdiagnosed entity. It may be misdiagnosed as subglottic stenosis or other lung pathology or missed entirely (Ong et al. 2020). It may present with shortness of breath, change of voice and respiratory distress and it can occur from hours to days post extubation (Kaleem et al. 2023).

The exact pathology of developing necrotising tracheitis is not properly understood. Likely during intubation or ventilation, there was a degree of

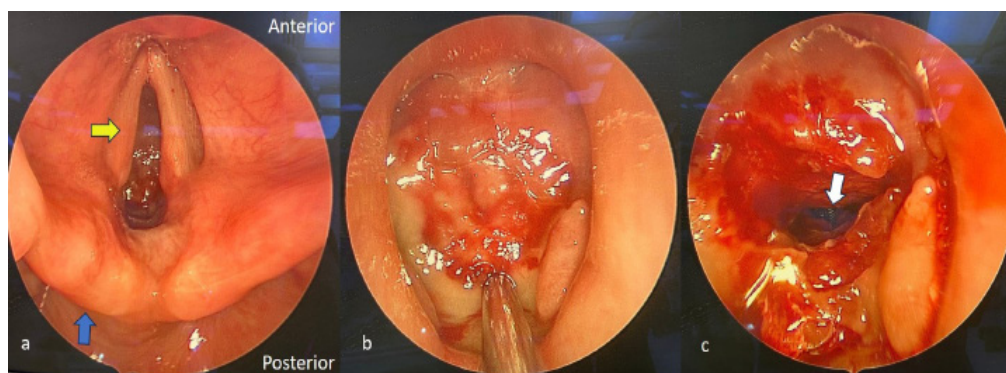


FIGURE 2: (a & b) in the second operation, intraoperatively, noted that there was a complete immature laryngotracheal stenosis Cotton Myer grade IV, consisting of granulation tissue with minimal slough and exposed cricoid cartilage seen at the posterior part which then proceeded with steroid injection and bougie dilatation (yellow arrow indicate left true cord, blue arrow indicate left arytenoid); (c) Post bougie dilatation, it became Cotton Myer grade II and we were able to visualise the tracheostomy tube (white arrow) below the stenotic segment

tracheal wall mucosal injury, subsequently the obstructive pseudo membrane formed and it was thought to be the initial steps prior to the developing of tracheal stenosis post intubation (Deslée et al. 2000). Cuff pressure measurements that were applied was not documented. High endotracheal cuff pressure on the tracheal wall can lead to necrosis of the tracheal mucosa due to ischemia. This leads to cartilage exposure and subsequently chondritis and perichondritis (Deslée et al. 2000). In addition, intubation outside a controlled operating theater setting, may be associated with increased risk of post intubation complications (Taboada et al. 2018).

Necrotising tracheitis is usually seen in the immunocompromised group as in our case. Our patient had recently been diagnosed with DM. His glycated hemoglobin (HbA1c) was 10.5% which indicates poor control. Diabetics are thought to be more susceptible to invasive infection as they are immunocompromised (Gupta et al. 2007). This immunocompromised state with poor glycemic control associated with more severe form of infection (Sharma et al. 2018).

Concurrent infection may act as another contributing factor. Our patient had hospital acquired pneumonia with positive blood culture with *Streptococcus mitis*. The bacteria may be hematogenously disseminated through the injured mucosa into bloodstream. Despite treatment with broad spectrum antibiotic, systemic bacteremia remains one of the major causes of morbidity and mortality (Franco-Paredes 2016; Vos et al. 2012). Postintubation injury initiates with mucosal inflammation, progresses to the loss of the epithelial layer, resulting in ulceration and ultimately extends to the cartilage leading to perichondritis.

CONCLUSION

Endotracheal intubation is a routine procedure. However, physicians have to keep in mind the complications that come with it. This includes the possibility of complications presenting after successful extubation, especially in the immunocompromised group. Regular assessment

and adjustment of cuff pressure can minimise tracheal injury and reduce the risk of subsequent infections, including necrotising tracheitis. Necrotising tracheitis can be fatal to patients as it can cause life-threatening upper airway obstruction and possibly death. Clinicians should maintain a high index of suspicion and follow vigilant monitoring protocols to ensure early detection and management of such potentially devastating complications.

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Ethics statement: Not applicable.

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