Doi: 10.5152/TJAR.2020.53323

Case Report
Resuscitation

Turkish Journal of Anaesthesiology & Reanimation

An 84-Year-Old Woman with Shortness of Breath and Low Oxygen Saturation: "Think Outside the Box"

Stefania Buttera[1], Cristian Deana[2], Fabio Beltrame[1], Flavio Bassi[1], Luigi Vetrugno[3,4], Tiziana Bove[3,4]

[1]Anaesthesia and Intensive Care Unit 2, Department of Anaesthesia and Intensive Care, Academic Hospital of Udine, Udine, Italy
[2]Anaesthesia and Intensive Care Unit 1, Department of Anaesthesia and Intensive Care, Academic Hospital of Udine, Udine, Italy
[3]Anaesthesia and Intensive Care Clinic, Department of Anaesthesia and Intensive Care, Academic Hospital of Udine, Udine, Italy
[4]Anaesthesia and Intensive Care Clinic, Department of Medicine, University of Udine, Udine, Italy

Cite this article as: Buttera S, Deana C, Beltrame F, Bassi F, Vetrugno L, Bove T. An 84-Year-Old Woman with Shortness of Breath and Low Oxygen Saturation: "Think Outside the Box". Turk J Anaesthesiol Reanim 2020; 48(6): 505-8.

Abstract

An 84-year-old woman, who had been admitted to the emergency department (ED) several times because of dyspnoea, was treated for acute exacerbation of chronic respiratory failure without satisfactory clinical improvement. According to her medical history, 8 years earlier, she underwent a complicated cardiosurgical procedure that required tracheostomy and mechanical ventilation in the post-operative period for 45 days. Traditional X-Ray did not show any abnormal findings; however, high resolution thorax computed tomography (HRCT) scan revealed a severe tracheal stenosis, which was confirmed with bronchoscopy, and required immediate tracheostomy. Tracheal stenosis is a rare but severe complication that should be suspected when a patient with previous tracheostomy presents to the ED with dyspnoea even if tracheostomy had been closed many years before, because adaptive mechanism results in asymptomatic life for a long period.

Keywords: Dyspnoea, tracheostomy, tracheal stenosis

Introduction

Tracheal stenosis is a well-known complication of tracheostomy. Its symptoms become highlighted when the tracheal lumen is reduced by 50–75% (1). Many patients with tracheostomy are severely affected by the other pathologies, which may be misleading and side-tracking in the early diagnosis of tracheal stenosis.

We present a case of tracheal stenosis that was diagnosed eight years after tracheostomy was closed; the patient was repeatedly misdiagnosed and treated for acute on chronic respiratory failure of medical origin.

Case Presentation

An 84-year-old woman with dyspnoea and low oxygen saturation (SpO_2, 76%), with an FiO_2 0.4 oxygen mask was admitted to the emergency department (ED). She was treated for acute on chronic respiratory failure with intravenous methylprednisolone and nebulized salbutamol, resulting in a slight clinical improvement. The patient had been admitted on multiple occasions for the same problem in the last 3 years and then discharged home with a final diagnosis of acute exacerbation of chronic respiratory failure.

Eight years earlier, she underwent coronary artery bypass grafting plus aortic valve replacement and mitral valve repair; it was complicated by prolonged post-operative respiratory failure, requiring tracheostomy and mechanical ventilation. After 45 days, the mechanical ventilation was withdrawn, and tracheostomy was closed.

Corresponding Author: Cristian Deana E-mail: deana.cristian@gmail.com

Received: 31.08.2019 Accepted: 19.09.2019
Available Online Date:18.05.2020

In the ED, the patient appeared tachypnoeic (30 breaths min^{-1}) using accessory respiratory muscles. The arterial blood gas revealed severe hypoxemia with respiratory acidosis: pH, 7.28; partial pressure of arterial oxygen (PaO_2), 60 mm Hg; partial pressure of arterial CO_2 ($PaCO_2$), 65 mm Hg; [HCO^{-3}], 18 mmol L^{-1}; and base excess (BE), −4 mmol L^{-1}; with a PaO_2 / fraction of inspired oxygen (FiO_2) ratio of 150 mm Hg; lactates, 1.5 mmol L^{-1}; body temperature, 36.5°C; heart rate (HR), 120 beats min^{-1}; and ambulatory blood pressure (ABP), 110/50 mm Hg. The 12-lead electrocardiogram (EKG) showed sinus tachycardia with unspecific ST and T-wave changes. Troponin was 0.6 ng mL^{-1} (normal value <0.045 ng mL^{-1}).

Chest auscultation revealed diffuse inspiratory and expiratory wheezes. Chest X-ray was normal. Extended ultrasound examination excluded acute heart failure or lung parenchyma disease. An urgent HRCT scan (Figure 1) revealed a concentric narrowing of the tracheal lumen under the subglottic cone with an obstruction of the cross-section higher than 85%, confirmed by fibreoptic bronchoscopy (Figures 2a, b). The patient immediately underwent tracheostomy after endotracheal intubation via the insertion of an 11 Fr airway exchange catheter (AEC, Airway Exchange Catheter, Cook Medical LLC, Bloomington, IN, USA) through which an endotracheal tube of 4 mm internal diameter was positioned.

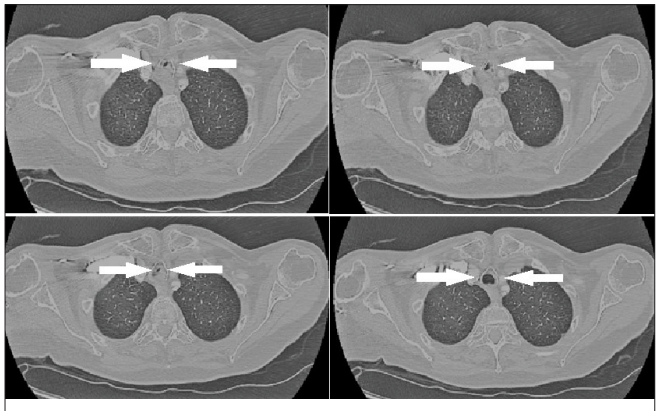

Figure 1. Thoracic CT scan revealing concentric narrowing of the tracheal lumen under subglottic cone

Main Points:

- The case of an 84-year-old female patient, with tracheostomy closed eight years prior, who presented to the ED with dyspnoea several times is reported.
- HRCT scan revealed, and bronchoscopy confirmed, a severe tracheal stenosis, requiring immediate tracheostomy.
- Adaptive mechanism results in a long asymptomatic period; hence, tracheal stenosis should be suspected when a patient with previous tracheostomy presents with dyspnoea, and can be confirmed by bronchoscopy.

Discussion

Most patients undergoing endotracheal intubation/tracheostomy develop some degree of tracheal stenosis. However, the symptoms become apparent only when the tracheal lumen is reduced by 50–75% (1, 2). Some patients nevertheless remain asymptomatic even under 70% of stenosis of the original lumen. In addition, many of these patients are severely affected by the other pathologies that may pose a challenge to early clinical diagnosis.

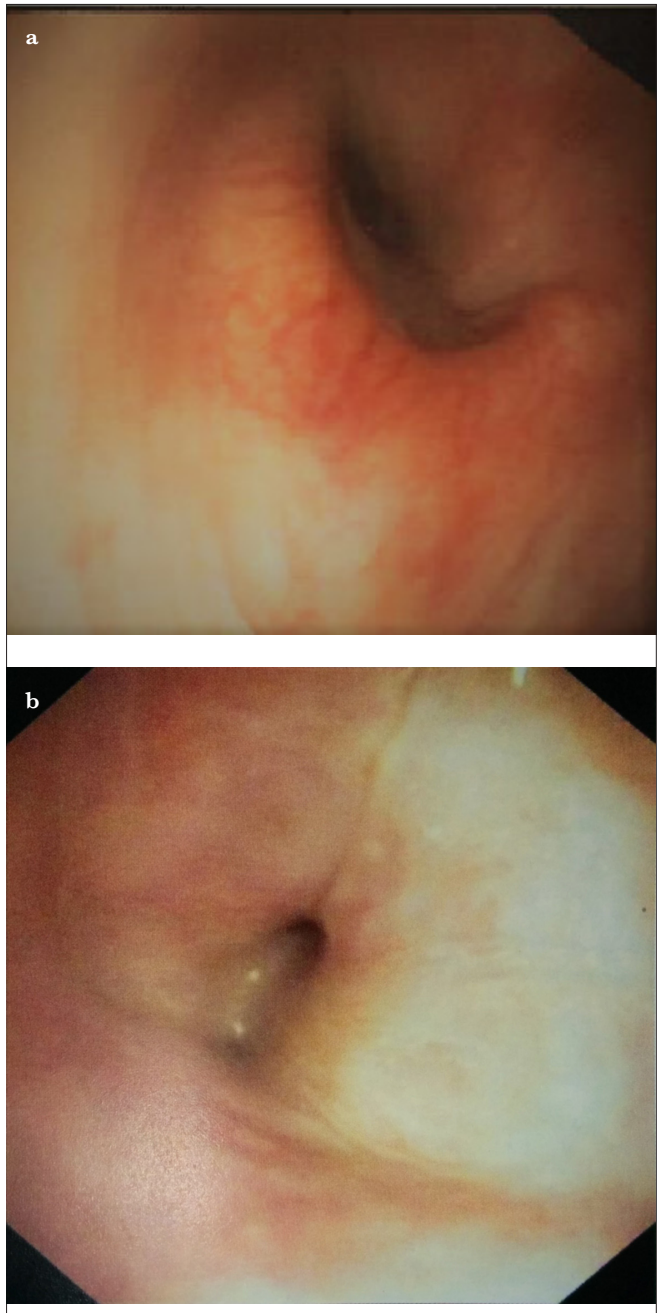

Figure 2. a, b. Bronchoscopic visualization of severe subglottic tracheal stenosis following surgical tracheostomy

Tracheal stenosis often occurs at the level or above the stoma, but below the vocal cords. It may also occur at the site of the tracheostomy tube cuff or at the tube's distal tip (2).

The aetiology of post-intubation/post-tracheostomy tracheal stenosis is multifactorial. One of the most notable causes is the cuff pressure exceeding the mucosal capillary perfusion pressure (30 mm Hg). This ischemia leads to ulceration, chondritis, and fibrosis within 3–6 weeks (3).

Onset of stenosis usually ranges from 2–24 weeks following extubation, and the incidence increases with the duration of intubation (1, 4, 5).

The pathophysiological explanation for the slow progression of tracheal stenosis symptoms is provided by the adaptive inspiratory time over the total respiratory cycle time (*Ti/Ttot*) (i.e. duty cycle).

For a given inspiratory load, the tidal volume is maintained because of the increase in the neuromuscular output and inspiratory duration. This compensatory mechanism requires an appropriate balance between the energy supply and demand.

The energy demand increases proportionally with the increase in pressure developed by the respiratory muscles, expressed as the ratio of the maximum pressure developed (mean inspiratory pressure/maximal inspiratory pressure, *Pi/Pimax*) and minute ventilation (*Ve*). *Ve* can also be analysed in terms of the mean inspiratory flow rate (i.e., tidal volume/inspiratory time, *Vt/Ti*) and the ratio *Ti/Ttot.* This analysis allows us to consider *Vt/Ti* as an index of inspiratory driving and *Ti/Ttot* as an index of respiratory timing. The latter is an important determinant of the load imposed on the respiratory muscles recruited during inspiratory time (6).

This implies that when an increase in *Ve* is required, the patient could adopt rapid shallow breathing pattern by increasing the mean inspiratory flow or decreasing the inspiratory time or both. Moreover, the product of *Pi/Pimax* and *Ti/Ttot* is a useful index, the so-called "tension time index (*TTI*)", which defines the respiratory muscle failure, and is directly related to the endurance time. If *TTI* exceeds the so-called "fatigue zone" of 0.15–0.18, the inspiratory load cannot be sustained indefinitely. When resistance to flow increases to the level such that the contraction of the respiratory muscles cannot be sustained without fatigue, *Vt/Ti* cannot be further increased as it approaches the peak inspiratory flow (*PIF*). In these circumstances, ventilation can only be maintained by prolonging *Ti/Ttot*. Unfortunately, the inspiratory load and increase in *Ti/Ttot* are energetically counterproductive, leading to fatigue. For any given *Ti/Ttot*, the respiratory rate increases with an accompanying decrease in *Vt* that worsens hypoxemia and leads to hypercapnia, which in turn aggravates the muscle fatigue (7).

For many years, our patient developed increased usage of the inspiratory accessory muscles, which can explain the physiological adaptive mechanisms.

However, any infection or cardiac failure can lead to an increase in oxygen consumption and respiratory decompensation. At any hospital, contact dyspnoea is misdiagnosed and treated as acute on chronic respiratory failure of medical origin until bronchoscopy reveals the real nature of the respiratory failure.

Conclusion

This case report highlights the importance of early recognition and management of an uncommon and potentially lethal complication of long intubation and tracheostomy, which is often interpreted as acute respiratory failure of medical origin. We found some explanations for the delayed diagnosis of this patient's tracheal stenosis: the rarity of severe stenosis diagnosis at a long distance from the tracheostomy, co-existing severe illnesses that have hidden the suspicion of tracheal stenosis, and lack of bronchoscopy, which is not routinely performed in emergency situations in patients with acute onset dyspnoea. The suspicion of tracheal stenosis must arise even after a long time has passed since the patient underwent tracheostomy, and the diagnosis can only be confirmed by bronchoscopy.

Informed Consent: Written informed consent was obtained from patient who participated in this study.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – S.B., F.Beltrame.; Design – S.B., C.D., F.Beltrame., L.V.; Supervision – F.Beltrame., L.V.; Data Collection and/or Processing – S.B., C.D.; Analysis and/or Interpretation – S.B., F.Beltrame., C.D., F.Bassi., L.V., T.B.; Literature Search – S.B., F.Beltrame., C.D.; Writing Manuscript – S.B., C.D., F.Beltrame.; Critical Review – S.B., F.Beltrame., C.D., F.Bassi., L.V., T.B.

Conflict of Interest: The authors have no conflicts of interest to declare.

Financial Disclosure: The authors declared that this study has received no financial support.

References

1. Epstein SK. Late complications of tracheostomy. Respir Care 2005; 50: 542-9.

2. Sue RD, Susanto I. Long-term complications of artificial airways. Clin Chest Med 2003; 24: 457-71. [CrossRef]
3. Zias N, Chroneou A, Tabba MK, Gonzalez AV, Gray AW, Lamb CR, et al. Post tracheostomy and post intubation tracheal stenosis: report of 31 cases and review of the literature. BMC Pulm Med 2008; 8: 18. [CrossRef]
4. Nesek-Adam V, Mrsić V, Oberhofer D, Grizelj-Stojcić E, Kosuta D, Rasić Z. Post-intubation long-segment tracheal stenosis of the posterior wall: a case report and review of the literature. J Anesth 2010; 24: 621-5. [CrossRef]
5. Beltrame F, Zussino M, Martinez B, Dibartolomeo S, Saltarini M, Vetrugno L, et al. Percutaneous versus surgical bedside tracheostomy in the intensive care unit: a cohort study, Minerva Anestesiol 2008; 74: 529-35.
6. Milic-Emili J, Grunstein MM. Drive and timing components of ventilation. Chest 1976; 70: 131-3. [CrossRef]
7. Bellemare F, Grassino A. Effect of pressure and timing of contraction on human diaphragm fatigue. J Appl Physiol Respir Environ Exerc Physiol 1982; 53: 1190-5. [CrossRef]