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CLINICAL REPORT

# Post-intubation long-segment tracheal stenosis of the posterior wall: a case report and review of the literature

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**Abstract** Tracheal stenosis, a well-known complication of endotracheal intubation and artificial ventilation, is most likely to occur in critically ill patients requiring prolonged mechanical ventilation. Although a rare complication, and despite technological improvements and better patient care in intensive care units, tracheal stenosis still constitutes a serious clinical problem which can also develop after a short period of mechanical ventilation. In this article, we present a very rare case report of a patient who developed a long-segment tracheal stenosis localized at the posterior wall after a relatively short period of endotracheal intubation with a high-volume, low-pressure cuffed endotracheal tube, and a review of the literature.

**Keywords** Endotracheal intubation · Tracheal stenosis · Complication · Nasogastric tube

## Introduction

Although endotracheal intubation and mechanical ventilation are therapeutic approaches for management of critically ill patients, they are associated with serious complications. One possible complication that can occur after endotracheal intubation is tracheal stenosis. Post-intubation tracheal stenosis most commonly occurs after prolonged intubation. The area of the trachea that is affected is usually 2 cm long and involves the anterior and lateral walls. The posterior wall is relatively protected from stenosis, because of its ability to distend into the esophagus [1, 2]. In this article we report a female patient who developed critical long-segment tracheal stenosis localized at the posterior wall after a relatively short period of intubation.

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## Case report

A 32-year-old female with body mass index 44 kg/m$^2$ and a history of lifelong obesity was admitted to our surgical department for elective Roux-en-Y gastric bypass surgery because of her morbid obesity. Her background history revealed she was a smoker and had failed several diet treatments for losing weight. Her obesity was complicated by mild bronchial asthma which she had had since childhood and which, in spring and autumn only, required treatment with inhaled bronchodilatator therapy. The initial gastric bypass surgery and anaesthesia were uneventful. However the postoperative course was complicated by pancreatitis, anastomotic leak, and sepsis, and two re-operations were necessary. Subsequently, she developed respiratory failure requiring mechanical ventilation. Her lung examination revealed diffuse rhonchi and wheezes throughout both lung fields. Her chest X-ray revealed a patchy infiltrate in the left lower lobe. *Acinetobacter baumannii* was isolated from the tracheal aspirate and she was treated with meropenem (3 g/day). She was mechanically

ventilated for 10 days using a 7.0 mm ID high-volume, low-pressure cuffed endotracheal tube until her condition improved and then the trachea was extubated. During this 10-day period one attempt of extubation was unsuccessful and she required re-intubation 10 h later because of acute respiratory failure following bronchospasm. Also she once required emergency re-intubation because of endotracheal tube obstruction. Each intubation procedure was done without difficulty using a 7.0 mm ID high-volume, low-pressure cuffed endotracheal tube without stylet. The cuff was inflated only to the point of eliminating leakage during ventilation Adequacy is generally checked by palpation of the pilot balloon and is sometimes re-inflated by injection of air in 0.5-ml increments, large enough to just stop an audible leak. The depth of insertion was 22 cm at the level of the corner of her mouth.

Two weeks after the third operation and 26 days after gastric bypass surgery she developed adhesive ileus and underwent a fourth laparotomy during which adhesiolysis was performed. During the laparotomy, a nasogastric tube was removed and replaced with a nasojejunal feeding tube (Freka® Trelumina CH 16/9, 150 cm, Fresenius Kabi) which was placed beyond the distal anastomosis. After the operation she was re-admitted to the ICU and extubated after 3 h. During her stay in the ICU, specimens for microbiological analysis were sampled as clinically indicated. Initial broad-spectrum antibiotic therapy was reassessed pending culture and sensitivity results. She required complex management with the main role going to surgeons and anesthetists/intensivists who worked closely with other specialists and health-care professionals. On the fifth day after her fourth and last operation the course was further complicated by an enterocutaneous fistula that manifested as intestinal contents draining from the wound. It was a high-output fistula draining between 900 and 1500 ml/day. The fistula was managed conservatively by administration of total parenteral nutrition and enteral feeding through the tube. Initially the elemental diet was started at a rate of 10 ml/h. We were able to achieve a feeding rate of 70 ml/h in 3 weeks and then total parenteral nutrition was stopped. The fistula output gradually decreased and stopped completely after 5 weeks. Oral feeding was also started with a low-residue diet, and the feeding tube was removed. The total duration of tube placement was 69 days (nasogastric tube 27 days, nasojejunal feeding tube 42 days).

Although during the 63 days in the intensive care unit she suffered numerous respiratory problems presenting mostly as bronchospasm, bacterial pneumonia, and pleural effusion, by use of antibiotics, bronchodilatators, and drugs with anti-inflammatory activity, her clinical condition progressively improved and she was transferred to the surgical ward. However, 2 weeks later her respiratory function worsened and she developed dyspnoea and shortness of breath, especially on exertion. Rhonci and wheezing were present on auscultation and she was treated with systemic corticosteroids and inhaled bronchodilatators. She did not improve and was noticed to have stridor. Tracheal stenosis was suspected and therefore a chest X-ray, fiberoptic bronchoscopy, multislice computed tomography (MSCT), and magnetic resonance imaging (MRI) were performed. Bronchoscopy revealed a stenotic trachea. Multislice computed tomography and magnetic resonance imaging confirmed that diagnosis and revealed marked thickening of the posterior wall of the trachea 4.0 cm long and narrow lumen. The minimum diameter of the trachea at the point of stenosis was 4.9 mm (Figs. 1, 2).

A pulmonary function test revealed a drop in the forced expiratory volume in 1-s (FEV1), a decline in forced expiratory flow (FEF 25–70%), and a significant reduction in peak expiratory flow (PEF) to 1.3 l/s (predicted 7.6 l/s) affecting both inspiratory and expiratory flow volume curve.

The FEV1 was only 34% predicted. The chest X-ray was normal. After the diagnostic procedures were performed, she was transferred to the Department for Thoracic Surgery, "Jordanovac" University Hospital for Lung Diseases. She was initially treated conservatively with repeated dilatation and laser resection, considering her health condition. Four days later she was returned to our hospital with slight clinical and functional improvement, but still complaining of exertional dyspnoea. Although spirometry

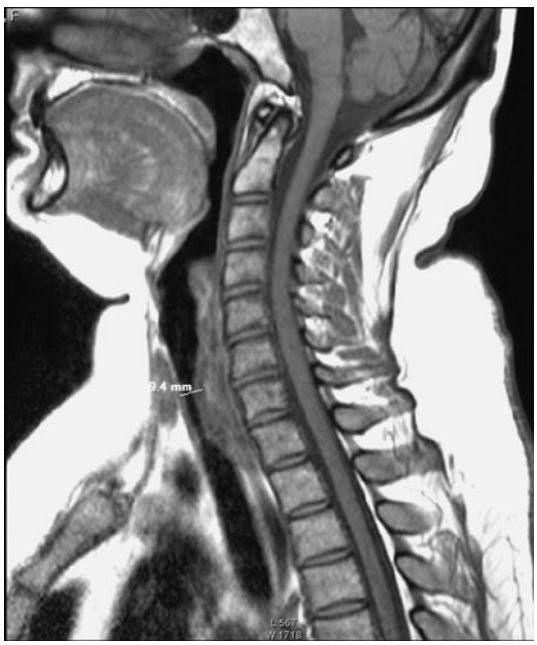


**Fig. 1** Severe long-segmental tracheal stenosis noted by multislice CT scan. Marked thickening of the posterior wall of the trachea of 4.0 cm long and narrow lumen. The minimum diameter of the trachea at the point of stenosis was approximately 4.9 mm

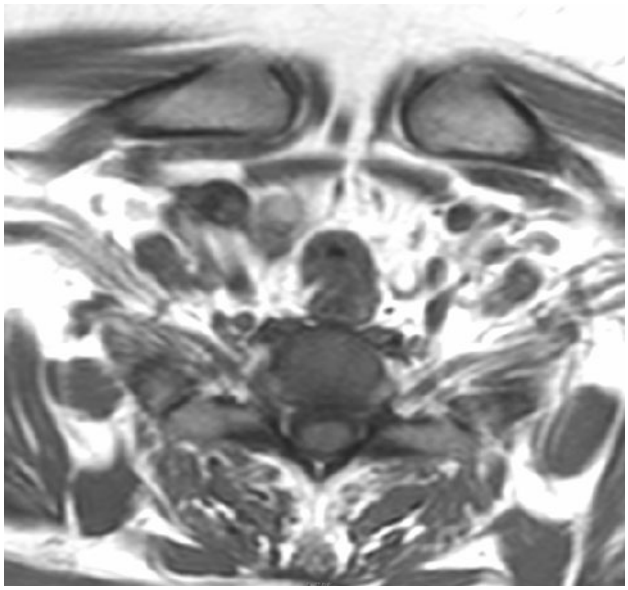

**Fig. 2** Post-intubation tracheal stenosis at the critical point of the stenotic area. MSCT scan shows narrowing of the tracheal lumen by increased thickness of the posterior tracheal membrane

revealed partial improvement of ventilatory function, the MSCT characteristic remained unchanged.

Her hospitalization lasted 14 weeks.

One month after discharge, she was readmitted to the Department for Thoracic Surgery "Jordanovac" University Hospital for Lung Diseases and underwent tracheal reconstruction. The stenotic part was resected and the trachea was reconstructed by end to end anastomosis. The histological findings of the resected trachea showed considerable thickening of the mucosa and submucosa with inflammatory mononuclear cell infiltration, granulation tissue, and fibrosis. The luminal surface was devoid of epithelium.

Her postoperative course was uncomplicated. She recovered very well and was discharged from the hospital after fiberoptic confirmation of a fluent trachea.

## Discussion

Post-intubation tracheal stenosis was first described in 1969 [3], although as an entity it was first recognized by Colles [4] in 1886. He described four cases of tracheal stenosis as the cause of tracheal obstruction in 57 individuals who had undergone tracheostomy for treatment of diphtheria [4]. Since then post-intubation tracheal stenosis has been a relatively rare but serious complication occurring mostly in critically ill patients requiring prolonged intubation.

Despite technological improvements and more skillful patient care, the incidence of post-intubation tracheal stenosis in intensive care units still remains between 6 and 21% [5] although only 1–2% [6] of patients are symptomatic or have severe stenosis. More recent studies using endotracheal tubes with a large area of contact (high volume, low pressure cuff), however, have shown that clinically significant stenosis was less common (1‰–1%) [7].

Patients usually remain asymptomatic until the tracheal stenosis exceeds a critical point of >30% of its original diameter, and it may take as long as 3 months before the diagnosis [8, 9]. Stenosis can occur anywhere from the level of the endotracheal tube tip up to the glottic and subglottic area, but the most common sites are where the endotracheal tube cuff has been in contact with the tracheal wall and has impaired microcirculation in the tracheal mucosa. It is usually localized at the anterior and lateral walls of the trachea. The posterior wall is relatively protected because of the possibility of its distending into the oesophagus [1, 2].

However, in patients with simultaneously indwelling firm nasogastric tube for an extended period of time the posterior wall can also be involved. Prolonged intubation and the presence of a firm nasogastric tube cause pressure on the sandwiched mucosa between the cuff of the tracheal tube and the nasogastric tube and contribute to the development of tracheal stenosis [1, 10, 11]. An appropriate diagnosis of tracheal stenosis, especially in patients with lower degrees of stenosis, is sometimes difficult, and the literature indicates that 44% of patients are often incorrectly treated for asthma or chronic bronchitis [12]. This is because these patients usually remain asymptomatic for a variable period. Patients who develop symptoms often do so between 10 and 42 days, although symptoms can develop within 2 days. Exertional dyspnoea is the most common presenting symptom of tracheal stenosis and is usually evident when the tracheal lumen becomes 10 mm in diameter, whereas stridor appears only when the tracheal lumen is narrowed to 5 mm or less. Duration of intubation and artificial ventilation are the most important factors in the development of stenosis. Whited et al. [13] reported 12% incidence of laryngeal stenosis in patients with tracheal intubation for 11 days or longer, 5% incidence for intubation between 6 and 10 days, and a 2% incidence for less than 6 days intubation. During prolonged intubation, the tracheal mucosa becomes ischemic which increases the likelihood of tracheal wall damage and subsequent stenosis [14]. When the cuff pressure exceeds the critical point in the mucosal capillary perfusion pressure (the mean capillary perfusion pressure in the tracheal wall is approximately 35 mmHg), mucosa that lies between the cuff and the cartilage develops ischemia. Ischemia then leads to ulceration and chondritis followed by fibrotic healing, leading to progressive tracheal stenosis of the tracheal cartilages. Ischemic injury may occur as early as 15 min

after insufflations of the cuff, and healing of the damaged region can result in fibrosis within 3–6 weeks.

It is also important to emphasize that numerous other factors predispose to the development of tracheal stenosis; these include the size of the tube relative to the tracheal lumen, frequent changing of the tube, traumatic intubation, ongoing infection, blood pressure in the period of intubation, female and estrogen effect, steroid administration, obesity, smoking history, gastric acid reflux, and an individual idiosyncratic reaction [15].

In patients with systemic infection and subsequent hypotension, cuff pressures may exceed the perfusion pressure of the trachea and further impair blood flow, resulting in significant tracheal damage. Local infection also worsens the mucosal damage, resulting in tracheal damage. It has also been assumed that estrogen increases the level of transforming growth factor $\beta 1$ sufficiently to promote production and deposition of collagens and, finally, fibrosis [16]. This is a probable explanation of the higher incidence of tracheal stenosis in females in some studies [17].

In this paper we describe a very rare long-segment tracheal stenosis localized at the posterior wall in a young patient who underwent elective gastric bypass surgery for weight loss. The coexisting illnesses of bronchial asthma and obesity contributed, postoperatively, to the development of bronchopneumonia and pleural effusion. Four surgical procedures with tracheal intubation were undertaken and re-intubation was required twice more during the period of mechanical ventilation Tracheal stenosis that occurred in our patient is the result of 10 days continuous intubation associated with prolonged ICU stay followed by systemic and local infection of the tracheobronchial tree, repeated intubation, and simultaneous placement of a firm nasogastric tube. Despite the relatively short period of intubation with a high-volume, low-pressure cuffed endotracheal tube she developed severe long-segment tracheal stenosis, localized at the posterior wall, over 4 cm long. The mechanism of development of such long tracheal stenosis of the posterior wall cannot be determinate from this report, but it could be assumed that the combination of repeated endotracheal intubation, prolonged mechanical ventilation, and simultaneous placement of a nasogastric tube for an extended period undoubtedly contributed to the development of tracheal stenosis. Although the cuff was inflated only to the point of eliminating leakage during ventilation, the cuff pressure was not measured and we cannot exclude the possibility of ischemic injury of the mucosa caused by cuff pressure exceeding the mucosal capillary perfusion pressure. Another possible factor that contributes to stenosis may be a difference of cuff position or alteration in cuff pressure. Although the endotracheal tube was fixed in the same manner every time, it is possible that the cuff position and the cuff pressure changed during mechanical ventilation and repeated intubation. A recent study by de Godoy [18] reported that changing the patient's position during mechanical ventilation can lead to significant alterations in cuff pressure. In addition, airway pressure changes caused by airway contraction because of bronchial asthma could have changed the cuff pressure. The different cuff position and cuff pressure may be important contributing factors to the development of long-segmental stenosis.

Other associated factors in our patient were also a combination of local and systemic infection, bacterial pneumonia, frequent tracheal suction, hypotension, obesity, history of smoking, and repeated tracheal intubation.

In conclusion, the case study presented in this paper demonstrates that cuff palpation alone is insufficient to detect high cuff pressures. The objective of this paper is, therefore, to alert anesthesiologists and intensivists to the need for continuous monitoring of cuff pressure and the need to avoid the use of large and rigid nasogastric tubes.

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