Double respiratory sequelae of head injury: subglottic stenosis and bilateral pneumothoraces

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An 18-yr-old man with insulin-dependent diabetes developed severe subglottic stenosis after a very brief period of intubation. Emergency tracheostomy was complicated by the development of bilateral pneumothoraces. This case highlights the importance of making an early diagnosis to minimize the risk of complications and examines postintubation subglottic stenosis in the context of poorly controlled insulin-dependent diabetes mellitus.

Br J Anaesth 2003; 90: 94–6

Keywords: complications, diabetes; complications, pneumothorax; complications, stenosis

Accepted for publication: July 27, 2002

Subglottic stenosis is a late, uncommon and serious complication of intubation. Although usually associated with prolonged intubation, this is not the only factor in its development. We report a stenosis after brief intubation. Possible contributing factors include poorly controlled insulin-dependent diabetes mellitus (IDDM). A high index of suspicion for this rare but life-threatening complication is necessary.

Case report

An 18-yr-old man was admitted to hospital after a road traffic accident. He was a smoker with poorly controlled IDDM. Decreasing consciousness necessitated tracheal intubation after induction of anaesthesia with propofol and succinylcholine. A standard size 8.5 mm oral tracheal tube (ETT) was placed with ease, with the tip positioned 25 cm from the lips. The vocal cords and surrounding structures appeared normal. After this, sedation was provided by propofol infusion and paralysis was maintained with atracurium.

A computed tomography (CT) scan of the patient’s head showed multiple small haematomas at the interface between the grey and white matter, but was otherwise unremarkable.

He was admitted to the intensive care unit (ICU) and was extubated after an uneventful 36 h. He was discharged from the ICU and, some days later, home.

Two months later he re-presented complaining of progressive shortness of breath since discharge. There was no history of stridor, wheeze or other respiratory symptoms. Chest x-ray showed consolidation in the right middle lobe. Despite treatment with antibiotics, he remained dyspnoeic and developed respiratory failure. Intubation and ventilation were indicated.

Anaesthesia was induced with propofol and succinylcholine. A size 9.0 mm ETT could not be placed, and the largest tube that could be placed was size 6.0 mm. Subsequent fibroptic examination showed a swollen epiglottis obscuring the laryngeal inlet and vocal cords. No obstruction was seen beyond the tip of the ETT. An 8 mm ETT was placed easily. Respiratory failure was attributed to epiglottic swelling, although it was unclear whether the swelling was the primary problem or the result of trauma during attempts to intubate. He was nursed with a head-up tilt of 30° and received dexamethasone 4 mg four times daily.

He was weaned from ventilation on day 5. When he was examined by an ear, nose and throat (ENT) consultant using per-nasal fibroptic laryngoscopy, a subglottic stenosis was...
found. At this time the patient was clinically well, with audible stridor that was not unduly distressing. A magnetic resonance imaging (MRI) scan showed a subglottic stenosis 2 cm long with an internal aperture of 5 mm. Follow-up was arranged and the patient was discharged home.

Six days later he presented again with symptoms of progressive upper respiratory obstruction. He was in extremis with stridor, had a ventilatory frequency of 30 bpm, oxygen saturation of 99% on inspired oxygen 28%, an arterial blood pressure of 172/102 mm Hg and a heart rate of 137 beats min\(^{-1}\) in sinus rhythm. He was referred for emergency tracheostomy under general anaesthetic.

With two anaesthetists and one consultant ENT surgeon in attendance, he was preoxygenated and a difficult inhalation induction was started in the supine position with an oxygen–sevoflurane mixture. Spontaneous ventilation was maintained throughout and partial airway obstruction persisted. Attempts at assisted ventilation and laryngeal mask airway (LMA\(^3\)) insertion worsened obstruction. Of the two anaesthetists present, one required both hands to hold the airway and prevent complete obstruction. The surgeon performed an emergency surgical tracheostomy—a Bjork flap with no excision of cartilage. This was deliberately placed well below the cricoid cartilage to avoid the stenotic region of the trachea.

Approximately 10 min after tracheostomy, hypoxaemia developed, oxygen saturation decreasing to 90% despite inspired oxygen 100%. Chest x-ray confirmed the clinical signs of bilateral pneumothorax and chest drains were inserted. The patient’s subsequent course in the ICU was uneventful and he was later referred for laryngeal reconstructive surgery.

**Discussion**

Subglottic stenosis is a well-documented complication of intubation. Although rare, it is the most common reason for tracheal resection and reconstruction.\(^1\) However, it is usually associated with prolonged intubation. Presentation is usually from a few weeks to a few months after intubation,\(^2\) which in this case report correlates with the initial intubation of 36 h and the stenosis first being noted at 2.5 months. A number of papers illustrate the large range in duration of intubation leading to tracheal stenosis,\(^2\)\(^3\) suggesting that, although duration of intubation is an important factor, it is not the sole factor in stenosis formation.

Nordin and colleagues\(^4\) recommended a maximum safe cuff pressure of 20 mm Hg after studying capillary blood perfusion of rabbit tracheal mucosa using isotope labelling. The evidence provided by Nordin and others encouraged the use of high-volume, low-pressure ETT cuffs to avoid tracheal ischaemia.

Keane and colleagues\(^5\) suggested that tracheal mucosal destruction could occur by erosion of the ETT tip. Granulation tissue rich in collagen accumulates, later contracting as the wound heals, causing tracheal stenosis. Yang\(^6\) proposed a similar mechanism of injury, caused by excessive movement of a sedated patient that occurred after intubation for 24 h. The present report differs from that of Yang because we obtained paralysis and sedation, which would prevent movement of the ETT caused by the patient.

Poorly controlled IDDM could worsen subglottic stenosis. The association between poor wound healing and IDDM is well recognized. After extubation, stricter control of his IDDM could have led to less serious complications. The effect of poorly controlled IDDM on subglottic stenosis formation may warrant further investigation.

Another reason for this stenosis could be local trauma, either at the time of the head injury itself, or later at intubation. However, the person who performed the first intubation reported normal anatomy, no difficulty in obtaining a tracheal airway and no evidence of airway trauma.

Another potential cause of mucosal damage is the construction of the tube itself. Contoured tubes are designed to conform to the anatomy where they are placed, in contrast to their rigid predecessors. Polyvinyl chloride is the construction material and is thought to be minimally irritant.\(^8\)

The failure of the LMA to provide adequate ventilation in this case should be examined. The LMA is often now used as a means of ventilation, notably as an adjunct in cardiac arrest without operators trained to intubate. Here, the use of an LMA precipitated a near disaster, and although the LMA is a legitimate means of providing an airway in an emergency, its use is not always helpful.

This case report emphasizes a serious but uncommon complication of intubation, which requires a high index of suspicion for diagnosis. Because this patient had few of the risk factors associated with subglottic stenosis, diagnosis was delayed and the consequences were almost disastrous. A young patient, recently intubated and presenting with extreme shortness of breath requiring further intubation, but with relatively little change on chest x-ray, should prompt a search for other causes of respiratory distress. CT and MRI allow early diagnosis and should be considered as part of the initial management of this condition.

**References**