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An unusual case of airway obstruction at the tip of an endotracheal tube caused by insertion of a nasogastric tube

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Abstract
We report an unusual case of ventilatory impediment caused by the obstruction of an endotracheal tube (ETT) by a nasogastric (NG) tube. A 72-year-old woman with bronchial asthma was scheduled for colostomy closure. An ETT of 7.5-mm internal diameter (ID) could not be advanced, and finally a 5.0-mm ID ETT was placed, because she had post-intubation tracheal stenosis. When an NG tube was inserted after endotracheal intubation, ventilation suddenly became nearly impossible. She was treated for an asthmatic attack, but her respiratory condition did not recover. We then exchanged the ETT for a laryngeal mask airway (LMA) and removed the NG tube. It was suspected that the cause of the airway obstruction was that the NG tube in the esophagus compressed the membranous portion of the stenotic trachea and the tip of the ETT was obstructed.

Key words Airway stenosis · Endotracheal tube · Nasogastric tube

Introduction

A nasogastric (NG) tube is frequently used for alleviating gastrointestinal symptoms. Risks of complications are often underestimated because the morbidity associated with the use of NG tubes is low [1]. We present here an unusual case of airway obstruction in a patient with tracheal stenosis whose airway was unexpectedly obstructed by NG tube insertion after tracheal intubation.

Case report

A 72-year-old woman (height, 148 cm; weight, 39 kg) was scheduled for colostomy closure and adhesiotomy. She had received mechanical ventilation for 6 days in the previous year because of multiorgan failure (including acute lung injury) secondary to sigmoid colon perforation. In the previous operation, the trachea had been intubated easily with a 7.5-mm internal diameter (ID) endotracheal tube (ETT), and the cuff volume was 6 ml.

She had a history of bronchial asthma for 30 years and had been treated with oral and inhalational medicines, without steroids. Her preoperative laboratory data, pulmonary function test, including an expiratory flow volume curve, and electrocardiogram results were within normal limits. The tracheal stenosis was overlooked, though it was detected by chest X-ray after the current episode.

No premedication was given. After placement of an epidural catheter in the Th 10-11 interspace, anesthesia was induced with fentanyl, propofol, and vecuronium. An attempt was made to intubate the trachea with a 7.5-mm ID ETT. The tube passed through the glottis easily but could not be advanced further. Tracheal intubation was attemped with smaller ETTs. ETT with sizes ranging from 7.0 to 5.5 mm ID could not be advanced. Finally, a 5.0-mm ID ETT with a cuff was placed through the glottis without being further advanced, and it was fixed at 19 cm from the tip of the ETT to the oral corner. The cuff was inflated with 6 ml of air. Then mechanical ventilation was started. The ventilation went well and the capnometer pattern was normal. The airway pressure was 30 cmH_2O at the time. Lung auscultation did not reveal wheezing. Anesthesia was maintained with 1%–2% sevoflurane and 60% oxygen in air. After the insertion of a 16-Fr NG tube, the peak airway pressure suddenly went up to 50 cmH_2O and ventilation became nearly impossible. The breath sound was barely audible and the capnometer showed marked prolongation of the expiratory phase. Oxygen saturation ($S_{P_{O_2}}$) with a pulse oximeter gradually decreased, and the end-tidal CO_2 (ET_{CO_2}) rose to 60 mmHg. An asthmatic attack was

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suspected because of her medical history, and she was treated with 5% sevoflurane, intravenous injection of hydrocortisone 600 mg, and intermittent ephedrine, and intravenous infusion of aminophylline 250 mg. However, the recovery of her respiratory condition was minimal, and the diagnosis of asthmatic attack seemed incorrect. Then, we attempted to perform bronchofiberscopic evaluations in order to rule out mechanical tube obstruction. Unfortunately, however, we had only a 4.0-mm outer diameter (OD) bronchofiberscope, which was considered too large to pass through the 5.0-mm ID ETT. Therefore, we determined to exchange the ETT for a laryngeal mask airway (LMA) and removed the NG tube. As soon as the NG tube was pulled out, the airway pressure decreased to 20 cmH_2O and the capnometer showed a normal ventilation pattern. Then we exchanged the ETT for an LMA. Bronchofiberscopic evaluation through the LMA showed a circumferential luminal narrowing in a region about 5 cm below the glottis. The lumen was about 4 mm in diameter at its narrowest portion, and showed a smooth surface and normal mucosal color. The operation was postponed. The LMA was removed after adequate spontaneous breathing and clear consciousness were obtained.

Post operative cervical computed tomography (CT) scan and it's multiplanar recontruction (MPR) image showed degeneration of the tracheal cartilage in the region of the tracheal stenosis (Fig. 1). The stenotic region seemed to be the area where the cuff of the ETT had been located the previous year.

Two months later, the patient was again scheduled for colostomy closure. Because of severe intraabdominal adhesions sufficient muscle relaxation was required. We did not want to use a muscle relaxant, in order to avoid neostigmine, which could be a trigger for an asthma attack. In addition, it was considered that combined spinal and epidural anesthesia (CSEA) might not prevent discomfort caused by the procedure in the whole abdomen. Therefore she was given general anesthesia, with CSEA, and spontaneous respiration was maintained using a LMA-ProSeal (Laryngeal Mask Company, Mahe, Seychelles).

An NG tube was inserted before the induction of general anesthesia, to exclude the possibility that the NG tube itself had caused the previous tracheal obstruction. The NG tube was pulled out and re-inserted after insertion of the LMA. She did not have difficulty in breathing. The operation proceeded uneventfully.

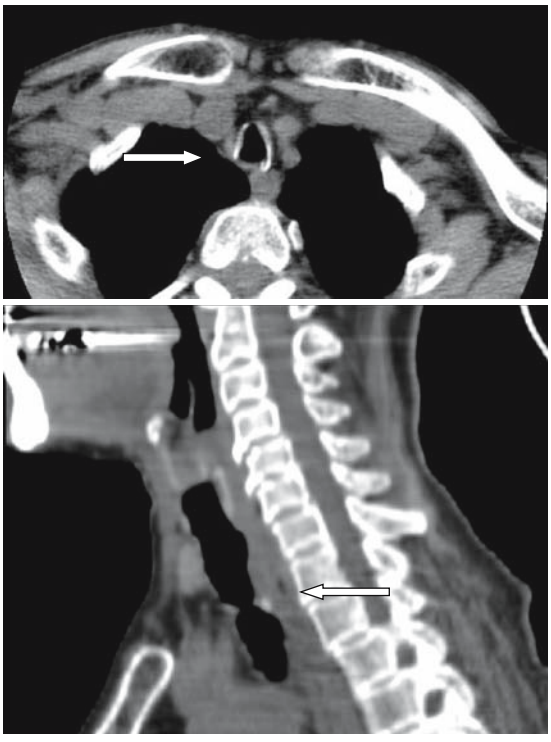

Fig. 1. Postintubation tracheal stenosis. Computed tomography (CT) scan (*upper*) and it's multiplanar recontruction (MPR) image (*lower*) shows focal subglotic stenosis (*arrows*)

Discussion

Mechanical upper airway obstruction is sometimes mistaken for an asthmatic attack because both induce a sudden and marked increase in airway pressure and, sometimes, failure of ventilation. In our patient, an asthmatic attack was initially suspected because the patient had a history of bronchial asthma. However, an asthmatic attack was ruled out because the drugs used for bronchial asthma were absolutely ineffective and the ventilation was smooth and easy after removal of the NG tube. It seems that the mechanical airway obstruction in this patient occurred because the ETT could not pass through the subglottic stenotic region, and the tip of the ETT was located above the stenotic region then the tracheal mucosa compressed by the NG tube obstructed tip of the ETT.

NG tube syndrome (throat pain and vocal cord abductor dysfunction) is uncommon, and is caused by ulceration of the mucosa of the posterior cricoid region [2]. Sofferman and Hubbell [3] describe various mechanisms for the causes of NG tube syndrome; one of these is that gravity pulls the larynx posteriorly, pinching the NG tube between the two rigid structures of the cricoid cartilage and anterior cervical spine in the supine patient. This supports our speculation that an NG tube can compress the membranous portion of the trachea from the esophageal side.

In conclusion, we report here a patient with tracheal stenosis whose airway was unexpectedly obstructed by NG tube insertion after tracheal intubation. The airway

in patients who have a previous history of endotracheal intubation or tracheostomy should be evaluated carefully before intubation. Further, we should recognize that an NG tube can compress the membranous portion of the trachea and cause airway obstruction.

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