CASE REPORT

A rare cause of complete airway obstruction caused by a defective pilot tube of a reinforced endotracheal tube

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Abstract

Background: The common causes of failure to inflate the lungs after placement of an endotracheal tube include mechanical obstruction of the tube by tight gauzes, tongue blade, tube kinking, patient biting on the tube, obstruction of the lumen by blood or foreign body, bronchospasm, haemothorax, tension pneumothorax or equipment malfunction.

Case presentation: We report a case of complete airway obstruction due to herniation of the pilot inflation line into the lumen at the proximal end of a reinforced tube when the tracheal cuff was inflated. Prior to use, the endotracheal tube was tested and functioned normally. The patient was uneventfully reintubated with a new endotracheal tube. With the endotracheal tube, device problems often involve the pilot balloon and cuff assembly and leaks are more common.

Conclusion: This case report highlights an unusual cause of complete proximal lumen obstruction due to a manufacturing defect which could have caused a critical incident in the theatre.

Keywords: Airway obstruction, Capnography, Endotracheal intubation, Equipment failure, Peak airway pressure

Background

When adequate oxygenation or ventilation is not achieved after an endotracheal tube (ETT) is placed, the causes have to be rapidly identified and reversed to avoid respiratory acidosis, hypoxemia, respiratory arrest and eventually cardiac arrest. One has to consider a tight bronchospasm, intraluminal obstruction by foreign body, dislodged tooth or mucus plug, ETT obstruction due to biting, tube kinking by position or tight gauze packs, oral retractors such as the Doughty's blade, and external lung compression from pneumothorax, haemothorax, or diaphragm rupture. In this case, after ruling out other possibilities such as tube kinking, patient biting or capnography side stream blocking, with a ETCO₂ of 0 mmHg, elevated peak airway pressures, reduced tidal volumes and failure to pass a suction catheter into the lumen, a complete obstruction of tube was suspected. Cuff herniation and blockage of distal lumen has been

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Ward et al. 1978). We highlight this case which is very rare cause of airway obstruction due to a manufacturing defect in the endotracheal tube which was diagnosed promptly and treated before a catastrophic event occurred. To troubleshoot acute deterioration in a mechanically ventilated patient, remember the DOPE mnemonic as outlined by the Paediatric Advanced Life Support guidelines. As a reminder, D is for displacement of the ETT from the trachea, O is for obstruction of the ETT, P is for pneumothorax, and E is for equipment or operator problem (Brady et al. 2012). In the mechanically ventilated patient, obstruction of the airway should be suspected by observing abnormally high peak inspiratory pressures, a decrease in compliance of the lungs, an increase in resistance to inflation and a notable difference between measured inhaled and exhaled tidal volumes. Obstruction in the intubated patient may also be identified by passing an appropriate size suction catheter to assure patency of the ETT. Other things to consider are the absence of breath sounds upon auscultation, review of the chest radiograph to assess appropriate ETT position, the observation of

reported in literature (Davis et al. 2011; Kao et al. 2005;





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dampened chest rise with manual or mechanical ventilation and a change in vital signs and saturation.

Case presentation

A 31-year-old female (ASA II, 60 kg, 155 cm) with a left renal calculus was posted for an elective percutaneous nephrolithotomy (PCNL) under general anaesthesia. She had a history of hypothyroidism which was well controlled with tablet eltroxin 50 µg once daily. Her blood pressure was 120/84 mmHg, and heart rate was 70 beats per minute. She had no prior cardiovascular symptoms and had an exercise tolerance of 6 MET s (metabolic equivalent of task). She had undergone a lower segment caesarian section under a subarachnoid block in the past. Her haemoglobin was 11 g%, serum potassium was 3.8 mEq/L, serum sodium was 141 mEq/L, serum urea was 21 mg/dL, serum creatinine was 0.8 mg/dL and random blood glucose was 100 mg/dL. Her ECG and chest radiograph was normal. Her airway assessment was normal, and no intubation difficulty was anticipated. In the pre-operative assessment clinic, she was explained about the anaesthetic technique. After obtaining a written informed consent, she was posted in the elective surgical list for PCNL. As for any elective surgery, she fasted for 6 h. Morning dose of eltroxin was taken with sips of water 2 h before she arrived in the operating room, and 1 g of cephalexin was given intravenously an hour prior to the surgery. In the operating room, she was connected to a multipara monitor displaying ECG, ETCO₂, gas analysis, SpO₂. NIBP, respiration, temperature and heart rate. The site of surgery was already marked, and through the intravenous cannula on the opposite hand, Ringer's lactate was started. She was premedicated with intravenous glycopyrrolate 0.2 mg, fentanyl 100 µg and midazolam 2 mg. Her baseline heart rate was 70 beats per minute, BP was 120/84 mmHg and SpO₂ was 100%. Following pre-oxygenation with 100% oxygen for 3 min, anaesthesia was induced with intravenous propofol 100 mg. After ensuring mask ventilation with 100% oxygen, intravenous atracurium 30 mg was given. The direct laryngoscopy view was Cormack and Lehane grade 1. Orotracheal intubation was done at ease using a Macintosh 3 blade with a size 6.5-mm-ID (internal diameter) wire-spiral reinforced cuffed ETT (Rusch) guided by a bougie. Tube position in the trachea was confirmed with bilateral equal vesicular breath sounds and an end-tidal carbon dioxide (ETCO₂) tracing. The tracheal cuff was inflated with air to produce an air-tight seal, and the endotracheal tube was fixed with a tape. Anaesthesia was maintained with 50:50 oxygen and nitrous oxide mixture with 1 MAC isoflurane through a closed circuit. She was connected to a ventilator on volume control mode with a tidal volume of 500 mL, respiratory rate of 12 per minute and a PEEP of 3 cm of water. Full muscle relaxation was confirmed by zero twitches present using a train-of-four monitor. Within the next 2 min, the peak airway pressure started increasing gradually up to 32 mmHg and the expiratory and inspiratory tidal volumes decreased and the ETCO₂ trace disappeared and showed a flat line displaying 0 mmHg. Exhaled tidal volumes measured almost less than 10 mL, and the pressure alarm limit set at $30 \text{ cm H}_2\text{O}$ alarmed repeatedly. During this time, the patient did not desaturate. Considering the possibility of a kinked CO₂ sampling tube, it was checked and the position of endotracheal tube was checked again by direct laryngoscopy to rule out oesophageal intubation. The circuit was switched to manual mode, and the APL valve was closed. The reservoir bag showed resistance to ventilation. There was no possibility of patient bite. Auscultation revealed no air entry. The possibility of a tight chest due to bronchospasm or occlusion of tube due to kinking or mucus was considered. The oxygen saturation started to drop below 95%. A sterile 10-Fr suction catheter was attempted to pass inside the ETT which could not pass beyond 3 cm as there was an intra-luminal obstruction. A possibility of complete tube obstruction was made, and the cuff was deflated, and the ETT was removed. The patient's lungs were ventilated manually by 100% oxygen on mask, and a new sterile 6.5 cuffed endotracheal tube was inserted after checking the pilot cuff. Her chest expanded with ease, oxygen saturation increased to 100% with reappearance of capnography trace. ETCO₂ was 55 mmHg initially and normalized to 38 mmHg subsequently. The surgery was carried out successfully, and she recovered well. The malfunctioning tube was inspected again. The blue pilot balloon, the thin tubing running from the blue pilot balloon along the edge of the endotracheal tube and the inflatable cuff itself were intact. But when the cuff was inflated by injecting air into the pilot balloon, the pilot tube at the proximal junction where it joined the endotracheal lumen herniated and inflated into the lumen causing total luminal obstruction. On deflation of the cuff, the lumen was patent again. The tube was packed and sent to the manufacturing company for their information.

Conclusions

Many manufacturing defects in ETT have been observed in the past, which include overpressure cuff herniation (Davis et al. 2011; Kao et al. 2005; Ward et al. 1978), intraluminal tracheal obstruction by kinking (Chua and Ng 2002), occlusion of distal ETT lumen by a plastic meniscus (Sofi and El-Gammal 2010), dissection of reinforced tube (Jeon et al. 2007; Choi and Lee 2018) and breakage of rings in armoured tube causing complete obstruction (Spiess et al. 1991). Similar finding of armoured endotracheal tube obstruction at the level where inflation line enters into the tube was reported by Kumar et al. where the obstruction occurred approximately one and half hours after commencement of surgery (Kumar et al. 2018).

This case will discuss factors that one may consider when adequate oxygenation or ventilation is not achieved after an endotracheal tube (ETT) is placed. Here in this case after ruling out other possibilities such as tube kinking, patient biting or capnography side stream blocking, with a $ETCO_2$ of 0 mmHg, elevated peak airway pressures, reduced tidal volumes and failure to pass a suction catheter into the lumen, a complete obstruction of tube was suspected. The peak airway pressure was 32 mmHg, and the tidal volumes were very low which further supported total luminal obstruction. Moreover, manual ventilation was very difficult, and there was no air entry. Had there been a decrease in ETCO₂ with a partial loss of capnography waveform, one may consider a partial airway obstruction which was not in our case. There was a sudden increase in ETCO₂ after the new tube was placed due to correction of ETT obstruction. In our case, it was a rare case of herniation of pilot tube into the proximal lumen on inflation of the cuff which sealed the lumen completely. In the mechanically ventilated patient, obstruction of the airway should be suspected by observing abnormally high peak inspiratory pressures, a decrease in compliance of the lungs, an increase in resistance to inflation and a notable difference between measured inhaled and exhaled tidal volumes. Obstruction in the intubated patient may also be identified by passing an appropriate size suction catheter to assure patency of the ETT. Other things to consider are absence of breath sounds upon auscultation, review of the chest radiograph to assess appropriate ETT position, the observation of dampened chest rise with manual or mechanical ventilation and a change in vital signs and saturation. End-tidal CO₂ monitor helps in assuring airway patency and the position of the ETT. When there is a partial obstruction of the endotracheal tube, ventilation is possible, although high inspiratory pressures are necessary. In our case, there was a total obstruction of the ETT. Anaesthesia trainers should be aware that perioperative bronchospasm has a similar presentation with complete silence on auscultation suggesting a tight chest, a few quiet rhonchi or loud expiratory noise. Accompanying diagnostic signs may include a rise in pulmonary pressures, falling oxygen saturation and a change in the slope of the end-tidal CO₂ trace. This also may present similar to endotracheal tube obstruction. Management of both these conditions is different. In ETT obstruction, an appropriate size suction catheter cannot be passed inside the lumen. Treatment of bronchospasm requires bronchodilators or deepening the plane of anaesthesia if the patient is light. A drop in end-tidal CO₂ trace may be indicative of mild reactive airways disease, oesophageal intubation, or a catastrophic anaphylactic reaction, reduced pulmonary perfusion, reduced cardiac output, hypotension, hypovolemia pulmonary embolism or cardiac arrest. Anaesthetists must therefore understand the precipitating factors, recognize the presenting signs and be prepared to undertake a logical plan of management. The plan should not only treat the problem but to re-review the patient's evolving condition. Apparatus malfunctioning, disconnection of circuit (note low airway pressures) leaks or disconnection of the sampling tube, ventilator malfunctioning and kinked ETT also cause a sudden drop in end-tidal CO2. The cuff is inflated via a small-bore inflation tube welded to the outside of the tracheal tube or built into its wall. The inflation tube is connected at the proximal end to a small pilot balloon designed to indicate cuff distension. Although armoured tubes are less likely to occlude through kinking, they are not a substitute for a bite block. Reinforced tubes have certain advantages over standard endotracheal tubes in several situations due to their resistance to occlusion when bent and their overall greater flexibility. Whilst it is significantly more difficult for a patient to totally occlude an armoured compared to a regular tube by biting onto it, the armoured tube has a specific disadvantage of not going back to a round diameter once the bite is released. Anaesthesiologists prefer using armoured tubes in head and neck surgeries, neurosurgery and prone position, when access to the airway is limited and chances of occlusion by kinking are less. Reinforced tubes can be useful in patients positioned prone. This case illustrates the potential device malfunction that can develop during a procedure, even when the equipment has been tested and previously functioned well. Even small defects that develop in well-engineered products, such as the manufacturing defect leading to pilot tube



Fig. 1 A patent lumen of the armoured tube when the cuff is deflated



Fig. 2 Complete luminal obstruction where the inflation line joins the tube near the proximal end of the armoured tube, when the cuff is inflated

dissecting into the lumen as described here, can lead to critical emergency situation. This case report shows an unusual reason for complete obstruction of ETT lumen. The illustrations supplied in this manuscript allow for visual identification of the luminal obstruction on inflation of the cuff and a patent lumen on deflation of the cuff (Figs. 1 and 2).

Abbreviations

ETCO₂ : End-tidal carbon dioxide; ETT: Endotracheal tube

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