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Case Report

A rare cause of intraoperative hypercarbia in the prone position: Cuff herniation versus internal damage of a reinforced flexometallic endotracheal tube

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Abstract

An armoured tube is preferred in securing airways where kinking or obstruction is expected due to positioning or surgical procedure. We report an incident of intraoperative hypercarbia in an elective spine case in prone position with armoured tube. The various reasons for intraoperative hypercarbia include: kink in the breathing circuit, obstructed expiratory valve, exhaustion of soda lime, obstructed airways and ventilator malfunction. In this case, the above mentioned reasons were ruled out. The possibility of cuff herniation was anticipated and 1 ml of air was removed from the ETT cuff and ventilation improved immediately and ETCO2 was normalised. In this patient, cuff herniation may be the probable cause of hypercarbia. Hence the cuff of the endotracheal tube should be checked for any manufacturing defect before intubation although it is new. Fibroptic was done and endotracheal tube was found to be patent.

Keywords: Armoured Endotracheal tube, Cuff herniation, Prone position

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1. Introduction

Endotracheal tube (ETT) cuff herniation is an infrequent yet potentially serious cause of airway obstruction and ventilation difficulties during general anaesthesia. Its subtle presentation can make diagnosis particularly challenging, especially during lengthy surgeries in non-supine positions. In this report, we describe a case of rising end-tidal carbon dioxide (hypercarbia) observed intraoperatively during an elective spinal surgery performed in the prone position using an armoured ETT. The ventilatory issue emerged approximately an hour after induction and was likely due to partial occlusion of a mainstem bronchus by the inflated cuff. Notably, the obstruction was promptly relieved following a slight deflation of the cuff.

2. Case Report

A 36-year-old female, presented to the orthopaedic unit with a one-year history of low back pain radiating to the right lower limb. She reported associated numbness and a sensation of reduced feeling over the same leg. Her symptoms were consistent with neurogenic claudication. There was no preceding trauma, and her past medical and surgical history was unremarkable.

On admission, the patient was alert and oriented. Her vital signs were stable, with a supine blood pressure of 130/90 mmHg in the right arm and a heart rate of 82 beats per minute with a regular rhythm. Examination of the cardiovascular, respiratory, and abdominal systems was within normal limits. Neurological assessment revealed tenderness at the L4–L5 vertebral level and paraesthesia over the right leg.

Routine laboratory investigations were normal. Chest radiography and a standard 12-lead ECG revealed no abnormalities. Transthoracic echocardiography demonstrated preserved left ventricular function with an ejection fraction of 73%. Magnetic resonance imaging (MRI) of the lumbosacral spine showed Grade I anterolisthesis of L4 over L5 with bilateral spondylosis and disc dehydration at L4–L5 and L5–S1 levels. There was a broad-based posterior disc bulge with central protrusion at the L4–L5 level, resulting in mild narrowing of the spinal canal and bilateral

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neural foraminal compromise, with compression of exiting nerve roots.

A diagnosis of L4–L5 spondylolisthesis was established, and the patient was scheduled for elective posterior spinal stabilisation and interbody fusion at L4–L5. Airway evaluation did not reveal any anticipated difficulty, and she was classified as American Society of Anesthesiologists (ASA) Physical Status I.

The patient received standard preoperative medications the evening before and on the morning of surgery. In the operating theatre, an intravenous line was secured in the left arm, and standard monitoring was instituted, including ECG, pulse oximetry, non-invasive blood pressure, capnography, respiratory rate, and temperature monitoring.

Premedication was administered with intravenous glycopyrrolate (0.04 mg/kg), midazolam (0.04 mg/kg), and fentanyl (2 mcg/kg). Preoxygenation was performed with 100% oxygen for three minutes. Anaesthesia was induced with IV propofol (2.5 mg/kg), and muscle relaxation was achieved with IV suxamethonium (2 mg/kg) to facilitate intubation.

An attempt to pass a 7.5 mm internal diameter (ID) flex metallic (armoured) endotracheal tube (ETT) encountered resistance at the subglottic level and was not forced. A 7.0 mm ID armoured ETT was successfully inserted and secured after confirming equal bilateral air entry. The cuff was inflated with 7 mL of air.

Anaesthesia was maintained with a 50:50 mixture of nitrous oxide and oxygen, intermittent doses of vecuronium (0.08–0.1 mg/kg), and 1% isoflurane. The patient was positioned prone with appropriate care to protect pressure points. Bilateral breath sounds were rechecked post-positioning. Mechanical ventilation was initiated with a tidal volume of 8 mL/kg and a respiratory rate of 14 breaths per minute. End-tidal carbon dioxide ($ETCO_2$) was monitored continuously.

Approximately one hour into surgery, a progressive rise in $ETCO_2$ from 45 mmHg to 70 mmHg was noted. Other vital parameters, including heart rate, blood pressure, and oxygen saturation, remained stable. The anaesthesia machine alarmed for high peak airway pressure. Initial troubleshooting ruled out circuit obstruction, valve malfunction, soda lime exhaustion, ventilator issues, and inadequate tidal volume delivery.

Despite increasing minute ventilation, $ETCO_2$ levels remained elevated. On auscultation, bilateral breath sounds were diminished. Bronchospasm was suspected and treated with intravenous hydrocortisone (100 mg), dexamethasone (8 mg), and nebulised bronchodilators (Duolin delivered via the inspiratory limb of the breathing circuit). However, hypercarbia persisted.

While the patient was still in the prone position, a 12 French suction catheter was introduced through the endotracheal tube to evaluate for any internal blockage. The catheter advanced without resistance up to the level of the carina, indicating that the tube was not obstructed by secretions or gross kinking at that point. Suspecting a possible cuff-related issue, 3 mL of air was withdrawn from the cuff. This intervention led to an immediate improvement in chest movement and a gradual reduction of $ETCO_2$ to 40 mmHg.

At the end of surgery, a fiberoptic examination was performed in the supine position. It revealed partial obstruction within the armoured tube. The patient was successfully reversed with intravenous glycopyrrolate (0.5 mg) and neostigmine (2.5 mg), then extubated. Postoperative recovery was uneventful, and haemodynamic parameters remained stable. The patient was transferred to the recovery unit for monitoring.

Following extubation, a nasal fiberoptic assessment of the airway was conducted using local anaesthetic nebulisation combined with the Spray-As-You-Go (SAGO) technique. The procedure revealed a pulsatile narrowing along the right lateral wall of the subglottic trachea, approximately 6 to 8 cm proximal to the carina. Inspection of the removed endotracheal tube in cross-section showed a noticeable reduction in its internal diameter. To further evaluate the observed airway abnormality, a contrast-enhanced CT scan of the neck was obtained.

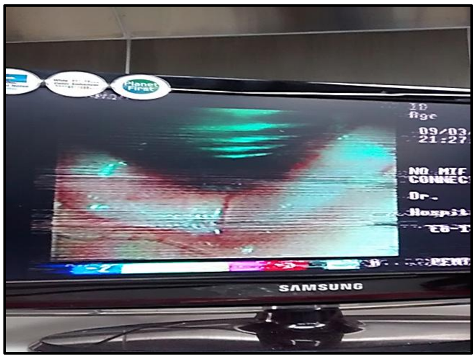

Figure 1: Nasal fibroptic view-a look at ETT position at vocl caord level

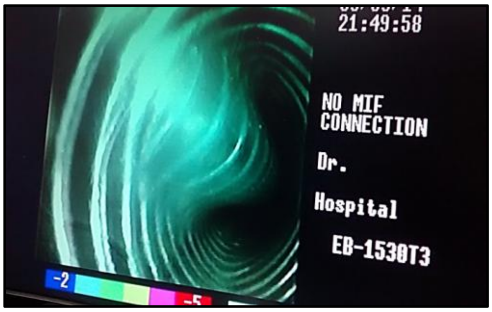

Figure 2: Internal obstruction of ETT- Fibroptic B'scopic view

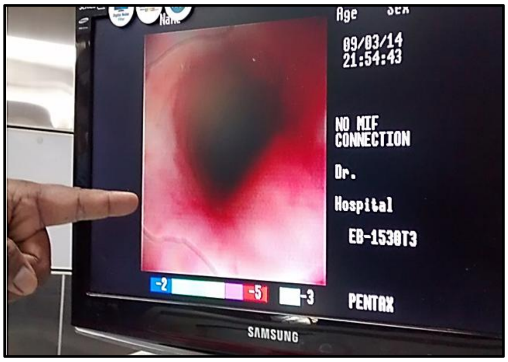

Figure 3: Sub-Glottic stenosis- fibroptic B'scopic view

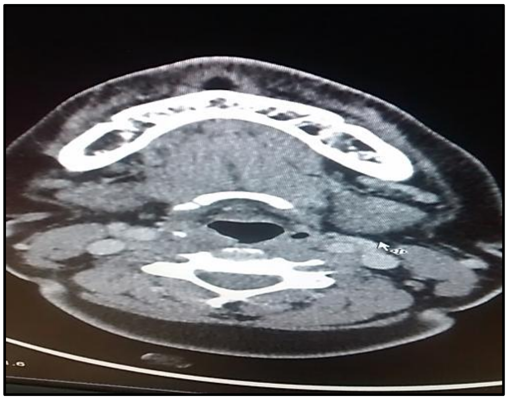

Figure 4: CT scan neck-contrast

Figure 5: Cut end of flexometallic ETT

3. Discussion

Airway obstruction during surgery can arise from a variety of factors. These include bronchospasm, bending or kinking of the endotracheal tube (ETT), issues with the ventilator or its components such as a faulty expiratory valve or blockage of the tube by secretions.[1] Additionally, any attachments placed between the ETT and the breathing circuit may contribute to obstruction. Patient positioning can also affect tube placement,[2] sometimes causing the tube's tip to rest against the tracheal wall, impeding airflow.[3] In armoured ETTs, damage or displacement of the internal metal spiral may lead to kinking and airway compromise.[4]

One commonly used method to detect intraluminal obstruction is inserting a suction catheter or a fiberoptic bronchoscope through the ETT.[5] However, certain conditions, such as cuff herniation, may not be apparent with these tools.[6]

In the presented case, the patient's ventilation improved immediately after partial deflation of the cuff, suggesting cuff herniation as the likely cause of obstruction affecting both mainstem bronchi. A known mechanism involves diffusion of gases like nitrous oxide into the cuff, leading to uneven expansion. This may result in the cuff pressing asymmetrically against the airway wall, bulging over the tube's tip, or narrowing the lumen.[6-9]

Ventilation difficulties were initially attributed to a combination of cuff herniation and inadvertent endobronchial migration of the endotracheal tube (ETT). The delayed onset of symptoms, particularly after approximately one hour in the prone position, raised the possibility of contributing factors such as gas diffusion into the cuff or a manufacturing defect.[4-5] To investigate this hypothesis, the ETT was reinflated with 10 mL of air after removal. The cuff demonstrated uniform inflation without asymmetrical bulging or focal protrusion, effectively ruling out cuff herniation as the underlying cause of the intraoperative airway obstruction.

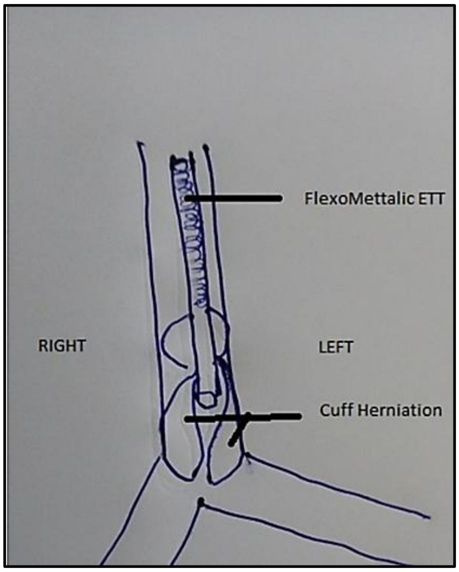

Figure 6: Cuff herniation-Illustration

Research, including that by Ward and colleagues, has demonstrated the possibility of cuff herniation in controlled settings, confirming it as a real cause of airway obstruction. Anatomical changes in vivo, even with initially correct tube positioning, can lead to obstruction over time.[10-11]

Choosing the appropriate ETT is essential in minimizing risk. Clear, single-use tubes with a Murphy eye (a secondary side opening) allow better visualization and reduce the likelihood of complete blockage. Reinforced tubes are beneficial during surgeries requiring head movement but should not be reused, as damage to the spiral support can lead to kinking. It is important to avoid applying any lubricant or liquid inside the lumen that might cause obstruction.[12]

Pre-use inspection of the tube is crucial. The lumen must be checked for patency, often using a stylet to ensure there are no foreign materials. The cuff should be inflated prior to use to assess for symmetry and secure attachment. Inserting a metal connector may help reduce the risk of external compression.

If the tube has a soft or flexible tip, a stylet should be used during intubation to guide placement and prevent bending. After intubation, the cuff should be inflated carefully. During surgeries involving nitrous oxide, regular monitoring and adjustment of cuff pressure is important due to potential gas diffusion into the cuff, which can increase pressure and volume. Radiographic imaging may help assess ETT position and cuff integrity. Withdrawing the tube while the cuff is inflated should be avoided to prevent it from folding over the tip. Bite blocks can prevent occlusion due to patient biting, especially during lighter planes of anesthesia.

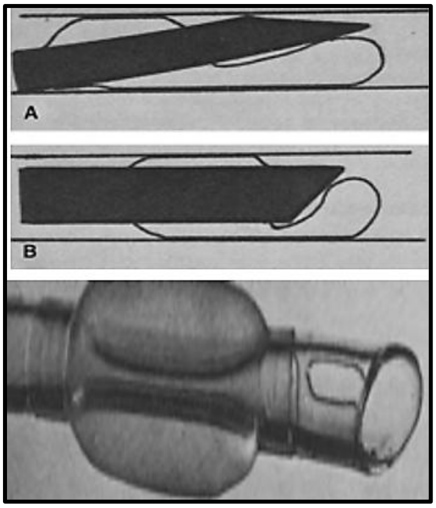

Figure 7: Three causes of tracheal lumen obstruction1 A: Eccentrically inflated cuff pushing the bevel against the wall of the trachea. B: The cuff balloons over the end of the tube and obstructing the tube completely or partially.

Reduction of tube lumen by inflation of a faulty cuff.

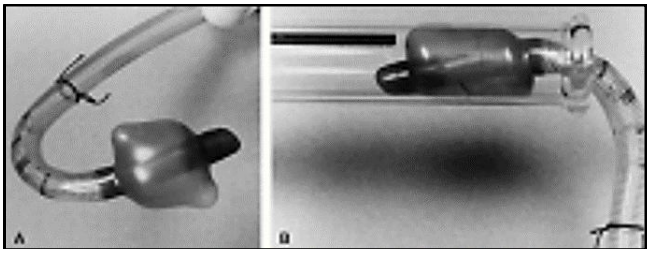

Figure 8: Tracheal tube obstruction secondary to eccentric cuff inflation[1] **A;** The cuff as removed from the patient. B; When placed in a glass tube, the inflated cuff pushes the bevel toward the wall of the tube.

Partial airway obstruction often presents with decreased lung compliance, reduced expiratory flow, elevated peak inspiratory pressures, and a larger gradient between peak and plateau pressures during volume-controlled ventilation. In pressure-controlled modes, clinicians may note reduced tidal volumes. Auscultation may reveal wheezing.[13] Capnography may show a steeper slope in phase III and a larger alpha angle. Severe obstruction may lead to generation of negative intrathoracic pressure, increasing the risk of pulmonary edema.[14] A ball-valve type obstruction allowing inspiration but limiting expiration may result in barotrauma or even cardiovascular collapse.[15]

Diagnosis is often confirmed by advancing a fiberoptic bronchoscope or suction catheter through the tube. If visualization is inadequate, the tube can be inspected directly using a laryngoscope or palpated externally to detect any kinking. Simple maneuvers like altering the patient's head position or deflating the cuff can relieve certain obstructions.

In specific cases, applying pressure to the site of a kink may alleviate the blockage. For narrow tubes, passing a larger ETT over the kinked one may help restore patency. In contrast, a smaller ETT may be passed through a kinked larger tube. If the obstruction is caused by biting, applying a hemostat at a right angle can open the tube temporarily. When thick secretions or blood clots are responsible, devices like Fogarty catheters can be used to clear the lumen. Rotating the ETT over a bronchoscope or using a suction tool designed for in-tube secretion removal are also effective strategies.

Finally, it’s important not to overlook components added between the ventilator and ETT as potential sources of obstruction. These should be considered, especially if introduced after the initial setup or shortly before problems arise.

4. Conclusion

In our patient, a nasal fiberoptic airway evaluation was performed post-extubation using local anesthetic nebulization along with the Spray-As-You-Go (SAGO) technique. This revealed a pulsatile narrowing on the right lateral aspect of the subglottic trachea, approximately 6 to 8 cm above the carina. A cross-sectional examination of the removed flexometallic endotracheal tube (ETT) demonstrated a noticeable reduction in the internal diameter of the lumen. To further assess the airway, a contrast-enhanced CT scan of the neck was carried out, which revealed no significant abnormalities.

During the intraoperative period, an unexpected rise in airway pressure along with hypercarbia led to a provisional diagnosis of cuff herniation. This assumption was supported by a temporary improvement in ventilation following deflation of the cuff. However, upon removal and inspection of the endotracheal tube, internal damage to the flexometallic structure was observed at the cut end. This finding indicated that the true cause of the airway obstruction was deformation within the tube’s internal architecture, either independently or in conjunction with a cuff-related issue.

5. Source of Funding

None

6. Conflict of Interest

None.

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