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CASE REPORT

Bronchospasm or not?

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Summary

Premature closure is one of a list of cognitive biases in decision-making during anaesthesia. Cognitive biases may delay the recognition and correct treatment of a life-threatening event. The authors describe a case of wrongly diagnosed bronchospasm following endotracheal intubation in a 53-year-old female patient who presented for elective peritoneal dialysis catheter re-insertion.

Keywords: bronchospasm, equipment failure, cognitive bias

Case report

A 53-year-old female with end-stage renal disease presented for surgical peritoneal dialysis catheter re-insertion. She had an American Society of Anesthesiologists (ASA) classification of III, was 166 cm tall, and weighed 60 kg (body mass index of 21.77 kg/m^2). She is known to have end-stage renal disease of unknown aetiology, with no further past medical history, and is awaiting a renal transplant. Her previous surgical history included a caesarean section, peritoneal dialysis catheter placement, and an incisional hernia repair. Her cardiovascular and systemic examination yielded unremarkable findings. She was haemodialysed 12 hours before surgery with post-dialysis serum sodium (Na^+)138 mmol/L, serum potassium (K^+)3.8 mmol/L, urea 6.6 mmol/L, creatinine 357 µmol/L, haemoglobin 8.7 g/dl, haematocrit 0.268, and platelets 366 000. Her preoperative chest X-ray and electrocardiogram were unremarkable.

Upon arrival in the theatre, the patient was calm. Standard ASA monitoring was placed, with the first measurements showing a heart rate (HR) of 64 bpm, arterial saturation of 96% on room air, and non-invasive blood pressure (NIBP) at 124/58 mmHg. Anaesthesia was induced with fentanyl 200 µg and propofol 80 mg, after which orotracheal intubation was facilitated by cisatracurium 10 mg. Direct laryngoscopy revealed a grade 3 Cormack–Lehane view. A gum elastic bougie was inserted into the trachea, and a standard size 7.0 mm internal diameter endotracheal tube (ETT) was successfully railroaded. The anaesthetist noticed that it took more effort than usual to remove the gum elastic bougie from the ETT after intubation.

The ETT was secured at 21 cm at the lip after position confirmation on auscultation. Anaesthesia was maintained with isoflurane in oxygen (O_2) and air. Pressure-controlled ventilation was initiated, with a minute volume of 3.5 L/min, peak inspiratory pressure (PIP) of 17 cmH_2O, fixed inspiratory-to-expiratory ratio (I:E) of 1:1.9, and positive end-expiratory pressure (PEEP) of 5 cmH_2O. The initial end-tidal carbon dioxide ($EtCO_2$) was 3.8 kPa.

A significant change in capnography was subsequently noted, characterised by a prolonged expiratory upstroke suggestive of bronchospasm. Concurrently, $EtCO_2$ levels increased to 6.5 kPa, and tidal volumes (Vt) dropped to 200 ml (Table I). In response, volume-controlled ventilation (VCV) was initiated with a Vt of 300 ml, during which the PIP was noticed to be 39 cmH_2O (Table I). At this time, the NIBP was 84/51 mmHg, HR 70 bpm, and arterial oxygen saturation remained at 100% on an inspired oxygen fraction (FiO_2) of 0.8. A 50 µg bolus of phenylephrine was administered intravenously.

On examination, there were no skin changes, but auscultation revealed a silent chest with minimal air entry, further reinforcing the suspicion of severe bronchospasm. This prompted an adjustment in ventilatory strategy, with VCV set to a Vt of 200 ml, a reduced respiratory rate of five breaths per minute, and an I:E of 1:2.5 to prolong exhalation. Following these changes, the

Table I: Case values

	HR (bpm)	BP (mmHg)	SaO_2 (%)	FiO_2	$EtCO_2$ (kPa)	Vt (ml)	MV (L/min)	PIP (cmH_2O)	$PaCO_2$ (kPa)
Pre-induction	64	124/58 (84)	96	.21	-	-	-	-	-
1 min from intubation	63	115/68 (83)	97	.95	3.9	380	3.5	17	-
5 min from intubation	63	93/58 (72)	100	.83	6.5	200	4.4	39	-
6 min from intubation	67	73/53 (61)	100	.48	5.0	215	3.5	25	-
35 min from intubation	93	127/58 (84)	96	.46	7.1	220	2	20	7.2
36 min from intubation	120	111/64 (84)	99	.35	5.9	600	7.9	20	-

BP – blood pressure, $EtCO_2$ – end-tidal carbon dioxide, FiO_2 – inspired oxygen fraction, HR – heart rate, min – minute/s, MV – minute volume, PaC02 – partial pressure of oxygen in the alveoli, PIP – peak inspiratory pressure, Sa02 – oxygen saturation, Vt – tidal volume

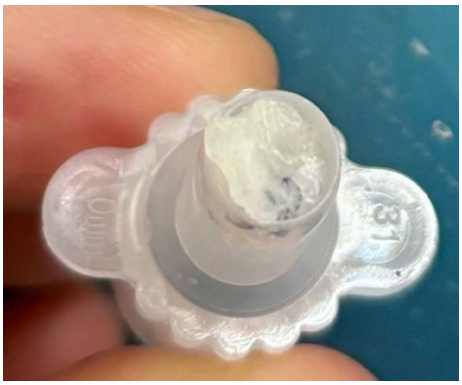

Figure 1: Partial occlusion of the endotracheal tube

Figure 2: Adhesive foreign body in connector

PIP decreased to 20–25 cmH_2O. Five doses of inhaled salbutamol were administered with a metered-dose inhaler via the ETT.

The patient did not respond to the initial phenylephrine bolus, so a second bolus (100 µg) was administered. Two boluses of adrenalin (25 µg) were administered intravenously as a bronchodilator to support right ventricle (RV) contractility due to the acute rise in RV afterload while maintaining a mean arterial NIBP > 65 mmHg. The patient was briefly switched to manual ventilation to evaluate lung compliance, with no change detected on auscultation and minimal air entry.

Further management included 20 mg ketamine and 100 mg hydrocortisone intravenously and a bronchospasm mix consisting of adrenalin 50 µg and lignocaine 3 ml 2% diluted in 2 ml normal saline via the ETT. The HME (heat and moisture exchanger) filter was inspected for any obstructions, while an arterial line was inserted for arterial blood gas (ABG) monitoring. The ABG revealed respiratory acidosis (pH 7.19, pCO_2 7.2 kPa, pO_2 21.0 kPa, HCO_3 20.6 mmol/L, base excess -7.8 mmol/L).

The patient did not respond as expected to the bronchospasm treatment, prompting a re-evaluation of the entire circuit for any other causes of obstruction. A partial obstruction was detected inside the narrow end of the ETT connector. Plastic-adhesive material from the manufacturing, firmly attached to the luminal sidewall, partially blocked the distal end of the connector and could not be removed with a hypodermic needle (Figures 1 and 2).

A new connector was inserted, and the ventilation and haemodynamic parameters immediately improved: NIBP 123/69 mmHg, HR 97, saturation 98% on FiO_2 0.4. PIP was reduced to 17 cmH_2O, and Vt increased to 400 ml, with downward-trending $EtCO_2$. The patient was successfully extubated and transferred to the recovery room. She received postoperative counselling in the ward.

Discussion

Bronchospasm in an intubated patient under anaesthesia typically presents with expiratory wheezing, raised peak airway pressures, a prolonged expiratory upstroke on capnograph, and, in extreme cases, a silent chest with haemodynamic compromise due to the reduced RV preload and an increased RV afterload. It can occur independently or as part of other conditions, such as anaphylaxis, and is often triggered by instrumentation or airway stimulation in patients with pre-existing airway diseases, such as asthma.[1]

This typical bronchospasm clinical presentation is repeatedly emphasised in all anaesthetists' training. Consequently, when this recognised pattern occurs, the result could be "premature closure", one of the cognitive biases that may cause a delay in the correct diagnosis and management of life-threatening conditions in the perioperative period.[2] The correct application of checklists and cognitive aids during perioperative emergencies can decrease cognitive bias and guide management in a best-practice, stepwise manner. The bronchospasm cognitive aid included in the Stanford Emergency Manual recommends early suctioning down the ETT as one of the management steps. This simple step could have resulted in early recognition of the ETT obstruction followed by the correct management.[3]

Many manufacturing defects in ETTs have been observed in the past, including herniation of the pilot inflation line into the lumen of the reinforced tube, dissection of a reinforced tube, overpressure cuff herniation, occlusion of the distal ETT by a plastic meniscus, and a defective connector with narrowed lumen.[4-9] Obstruction may occur anywhere along the patient's airway, airway devices (ETT, supraglottic airways), connectors, breathing circuits, or the anaesthetic machine itself. Therefore, it is vital that all potential causes are considered and a systematic approach is followed to identify the problem correctly. Awareness of the potential for foreign body obstruction from anaesthetic adjunct devices is crucial in this setting, including inspection of all ETTs and connectors before use.

Conclusion

In our patient, an ETT was placed to maintain a secure airway. However, an ETT does not inherently guarantee a patent airway. In this instance, the ETT itself became the source of airway obstruction due to the partial occlusion by adhesive material. This rare complication highlights the importance of vigilance

and readiness to manage unforeseen intraoperative challenges, even with seemingly routine devices like an ETT.

Conflict of interest

The authors declare no conflict of interest.

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Ethical approval

Ethical approval for the research was granted by the Human Research Ethics Committee of Stellenbosch University (reference number: C24/11/033).

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