Asystole during rigid bronchoscopic stenting under general anaesthesia in a patient with tracheo-oesophageal fistula

Sir,

We present a case of a patient with tracheo-oesophageal fistula (TOF) who required rigid bronchoscopic-guided tracheobronchial stenting. This is a challenging procedure in view of airway, respiratory and cardiovascular related challenges in the perioperative period.[1] Consent was taken for publication of the case.

A 40-year-old woman weighing 60 kg with TOF was posted for tracheobronchial stenting. She was diagnosed as a case of carcinoma oesophagus 2 months back. Upper gastrointestinal endoscopy revealed an ulcerative lesion at 22–28 cm with the opening of TOF within it. She had received three cycles of carboplatin and paclitaxel based chemotherapy and feeding jejunostomy was performed. Computed tomography scan showed a mass of 6 cm × 5 cm in size causing obliteration of the left main bronchus with total left lung collapse. Flexible bronchoscopy revealed a large fistula in the left main bronchus around 2 cm from carina, and the right main bronchus had multiple (3–4) fistulas around 1 cm from carina.

In the operating room, the airway was nebulised with 1% lignocaine in 4 mL saline. Baseline vitals were heart rate (HR) of 67/min, blood pressure (BP) of 134/88 mmHg and oxygen saturation (SpO_2) 96%. Dexmedetomidine infusion was started at loading dose of 1 μg/kg over 10 min. After pre-oxygenation for 3 min, induction of anaesthesia was accomplished with intravenous 1 mg midazolam, 100 μg fentanyl, 100 mg propofol and 25 mg atracurium. Anaesthesia was maintained with oxygen, propofol infusion at rate of 5 mg/kg/h and dexmedetomidine infusion at rate of 0.5 mcg/kg/h. The rigid bronchoscope (RB) was inserted and lungs were ventilated through side port of the RB with 100% oxygen. Five minutes later, HR started decreasing in the range of 40–50/min and reached a nadir of 35/min whenever the RB touched the carina. The HR increased to 50/min when carinal stimulation was stopped. The SpO_2 was maintained between 95% and 98%. The BP ranged from 110 to 150 mmHg (systolic) and 60–90 mmHg (diastolic). The

procedure continued, and 15 min later, at the time of stent deployment, the patient had an episode of bradycardia when the HR decreased from 65 to 10 beats/min and then asystole over around 10 s. The plethysmograph tracing disappeared and BP was not recordable. The RB was withdrawn. The dexmedetomidine and propofol infusions were stopped and intravenous atropine 0.6 mg was administered. The HR returned to 54/min and BP was 98/66 mmHg. The SpO_2 was 96%. Four millilitres of 2% lignocaine was instilled through the RB. Thereafter, the patient remained hemodynamically stable. The procedure was completed in 30 min, and residual neuromuscular blockade was reversed with intravenous neostigmine 2.5 mg and glycopyrrolate 0.5 mg. She had an uneventful recovery and shifted to post-anaesthesia care unit for observation.

Haemodynamic perturbations may happen during the airway intervention under general anaesthesia due to a direct stimulus or due to drug effects. In our case, the bradycardia and arrest appears to be drug related (dexmedetomidine, propofol and fentanyl combination) in addition to direct stimulation of bronchoscope on airway structures which has not been reported earlier. Fentanyl causes bradycardia by inhibiting GABAergic neurotransmission to cardiac vagal neurons in nucleus ambiguous.[2] Propofol has been associated with bradycardia.[3] The combination of propofol and fentanyl has been associated with episodes of severe bradycardia.[4] Dexmedetomidine is a potent alpha-2-adrenoceptor agonist. It has biphasic cardiovascular response, i.e. an initial transient rise in BP followed by a decrease in HR and BP.[5] Up to 42% of patients who have received dexmedetomidine have bradycardia.[6] It appears in our case that probably propofol and fentanyl may have potentiated bradycardia induced by dexmedetomidine in addition to airway manipulation. RB may cause intense vagal stimulation leading to bradycardia and/or asystole. Such stimulation can be also be obtunded by the use of intratracheal lignocaine. The light plane of anaesthesia could also elicit the stress response leading to haemodynamic instability. However, the use of propofol and dexmedetomidine was used for maintenance optimal depth of anaesthesia.

To conclude, although complication rate during RB and stenting is low and bradycardia is a known complication but such a case having asystole during manipulations of RB and stent deployment under general anaesthesia using a combination of dexmedetomidine, propofol, fentanyl has not been reported. Bradycardia during RB can be resolved by withdrawal of the bronchoscope but that occurring during tracheal stent deployment is more problematic, and vagotonic drugs like propofol, fentanyl and dexmedetomidine, may need to be stopped or given in reduced doses.

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Conflicts of interest

There are no conflicts of interest.

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