

Case Report

Laryngospasm Caused by Removal of Nasogastric Tube after Tracheal Extubation: Case Report

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Abstract

Background: We report a case of laryngospasm during nasogastric tube removal. Laryngospasm is a severe airway complication after surgery and there have been no reports associated with the removal of nasogastric tubes.

Case Report: After abdominal surgery, the patient was extubated the tracheal tube, and was removed the nasogastric tube. Thereafter patient went into respiratory arrest. We attempted to ventilate using a face mask, and then through a supraglottic device, but both attempts were unsuccessful. Finally, we re-intubated her and stabilized her vitals.

Conclusion: When patients are in emerging from anesthesia, nasogastric tube withdrawal may cause irritation of the vocal cords by gastric acids, and thereby, provoke laryngospasm. This can be avoided by removing it before reversing anesthesia or after the patient is awake.

Keywords: Gastric acid; Gastrointestinal; Intubation; Laryngismus

Abbreviations

mcg/h: Microgram per hour

L/min: Litter per minute

pH: Concentration of protons (H⁺) in a blood

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pCO₂: Partial pressure of carbon dioxide

pO₂: Partial pressure of oxygen

mmHg: Millimeter of mercury

mg/dl: Milligram per deciliter

mmol/L: Millimole per liter

Introduction

Background

Laryngospasm (spasmodic closure of the larynx) is an airway complication that may occur when a patient emerges from general anesthesia. It is a protective reflex, but may sometimes result in pulmonary aspiration, pulmonary edema, arrhythmia and cardiac arrest [1]. It does not often cause severe hypoxemia in the patient, and our review of the literature revealed no previous reports of such cases that cause of the laryngospasm was the removal of nasogastric tube. Here, we describe this significant adverse event in a case and suggest ways to lessen the possibility of its occurrence. We obtained written informed consent for publication of this case report from the patient.

Case report

A 54-year-old woman height, 157 cm; weight, 61 kg underwent abdominal surgery (partial hepatectomy with right partial nephrectomy and colectomy) under general and epidural anesthesia at our hospital. After midthoracic epidural cannulation and pre-oxygenation, anesthesia was induced with 100 mg of propofol and 40 mg of rocuronium intravenously. When bag-valve-mask ventilation was performed, we found its difficulty and the capnography showed phase III, as described by Bhavani-Shankar, et al., [2]. After tracheal intubation using video laryngoscope (McGrath MAC™, Covidien Japan, Tokyo, Japan), anesthesia was maintained with inhaled desflurane, small dose of intravenous remifentanyl (50-180 mcg/h), intermittent epidural 0.375% ropivacaine, and intravenous rocuronium. After induction of general anesthesia, radial arterial cannulation (22 Gauge), right internal jugular vein cannulation (16 Gauge single lumen), and nasogastric tube (16 French) insertion were done. All the cannulation and insertions were done at first trial without any trauma. The position of nasogastric tube was confirmed by the auscultation and visual characteristics of aspirates. After the successful completion of surgery, we performed intratracheal and intraoral suctioning, observed only minimal secretions, and confirmed that the chest and abdominal radiographs were normal. We, then, discontinued the administration of remifentanyl and desflurane, and increased the flow of fresh gas to 10 L/min to rapidly arouse the patient from anesthesia. After confirming a train-of-four count of two on the neuromuscular blockade monitor, sugammadex 150 mg was administered through the central vein. Three minutes after we confirmed recovery from the neuromuscular blockade using the train-of-four monitoring (109%), although she did not obey instructions to move her arms or legs, she moved intensively. Her tidal volume was more than 300 ml consistently, and her respiratory rate was 12 per min. We, therefore, removed the tracheal tube. After extubating, we observed a large quantity of oral discharge and performed intraoral suction.

We, then, removed the arterial catheter at the risk of self-extraction because the patient appeared to be confused and restless. The pupil diameter at this point was 3 mm/3 mm without laterality. Spontaneous breathing was noted, chest examination by a stethoscope was unremarkable, and no paradoxical breathing was observed. Since there was no other obvious cause for her restlessness, we considered the irritation of the pharynx by the nasogastric tube and removed it with active suction. The patient became calm immediately after its removal and her oxygen saturation by pulse oximeter began to decrease about three minutes after removal. Subsequently, the patient stopped breathing and did not react to painful stimuli. We attempted bag-valve-mask ventilation with a nasal airway, but were unsuccessful. We, then, attempted to ventilate the patient using a supraglottic device (i-gel™#3, Intersurgical Limited, Berkshire, United Kingdom). Although the device was inserted smoothly, we were unable to ventilate her. Approximately six minutes after we began attempting positive pressure ventilation (oxygen saturation was extremely low and vital signs monitor, Philips Intellivue MX800 showed 9%), we successfully managed tracheal re-intubation under video laryngoscope (McGrath MAC™, Covidien Japan, Tokyo, Japan) guidance. At the same time to attempt re-intubation, we also prepare rocuronium bromide in a new syringe. During tracheal re-intubation, we noted that the glottis was slightly open. After intubation, her oxygen saturation gradually increased, and we re-cannulated the radial artery. Three minutes after intubation, arterial blood gas analysis showed a pH of 7.358; pCO₂ of 45.9 mmHg; and pO₂ of 544 mmHg. At this time, the patient's glucose concentration was 174 mg/dl; lactate concentration was 1.6 mmol/L; and end-tidal desflurane was 0.8%. On examination, there was laterality in the pupils (left 1 mm/right 2 mm) and slight upward deviation of right eye, and therefore, computed tomography and magnetic resonance imaging of the brain were performed to rule out hypoxic injury and brain diseases that cause seizures, and both were unremarkable. After transfer to the intensive care unit, the patient slowly regained her consciousness, was appropriately oriented, and obeyed commands adequately. The pupillary laterality disappeared, and her Prince Henry Pain Scale score was 0. Six days after surgery, the patient was discharged from the hospital. Nine days after surgery, electroencephalography was performed; no epileptic waves were observed.

Discussion

Laryngospasm (spasmodic closure of the larynx) is essentially a protective reflex to prevent foreign materials from entering the tracheobronchial tree; it has been estimated to occur in 0.87% of general anesthetic administrations [1]. It tends to occur after nasogastric extubation at an insufficient depth of anesthesia or while ventilating via a face mask or supraglottic device, especially when the airway has been irritated by laryngoscopic blade insertion, suctioning, mucous, or blood [3,4]. The standard treatment for laryngospasm is administration of 100% oxygen via face mask under positive pressure ventilation. This is usually sufficient, and only rarely do patients develop hypoxemia requiring emergency intubation [5].

In the present case, we could not achieve ventilation via a face mask or supraglottic device. Therefore, we had to assess the presence of other airway obstructions (including bronchospasm or supraglottic obstruction), opioid-induced muscle rigidity, or apnea. Although we could not visualize the glottic closure using a video laryngoscope, there was no indication of a mechanical airway obstruction, and the

patient could be easily ventilated after tracheal reintubation. Therefore, bronchospasm and supraglottic obstruction were ruled out as possible causes of the patient's respiratory arrest. Although remifentanyl may induce hypoxia when administered intravenously as a push over five seconds, it had not been administered in the ten minutes preceding the patient's respiratory arrest [6]. We had also used an intermittent drug infusion through the central venous line rather than a direct remifentanyl infusion. For these reasons, remifentanyl is unlikely to have caused the respiratory arrest.

Our patient had been restless and became calm after removal of the nasogastric tube, which suggests that the removal may have caused a laryngospasm that impeded her airflow. And desaturation began 3 minutes after extraction also suggests that the extraction was relevant. Recently, it has been reported that gastroesophageal reflux disease may cause patients to develop laryngospasm and syncope, and this may cause sudden unexpected death in patients with epilepsy [7,8]. In our case, removal of the nasogastric tube may have caused irritation of the larynx by gastric acid and induced laryngospasm in the same manner. Superior laryngeal nerve chemoreceptor's are also known to trigger laryngospasm at a low pH [9]. If gastroesophageal reflux occurs while the patient is supine, physicians often recommend that the patient sit up or stand, thereby allowing gravity to move the acid back down the esophagus, causing the larynx to relax. In cases like ours, where the patient remains sedated (and supine) due to unmetabolized desflurane, laryngospasm may go undetected.

Light anesthesia, such as the "Stage 2" anesthesia as described by Guedel in 1927, is a risk factor for laryngospasm [10,11]. In this case, the patient was under light anesthesia when we removed her nasogastric tube in the operating room. This may be attributable to the fact that, after tracheal reintubation of the patient post-respiratory arrest, her end-tidal desflurane volume was still 0.8%. Consensus guidelines for enhanced recovery after surgery for gastrectomy, liver surgery, and pancreaticoduodenectomy, strongly discourage the routine use of nasogastric tubes because they may increase pulmonary complications and delay recovery of normal bowel function [12-14]. In cases involving pancreaticoduodenectomy, it is especially recommended that nasogastric tubes be removed before the reversal of anesthesia [14].

We speculate this patient arrested secondary to laryngospasm. This speculation was based on the fact that we could not perform mask ventilation or ventilation through supraglottic device, i-gel™#3, inserted after we tried mask ventilation. We did not use bronchoscopy to diagnose laryngospasm through supraglottic device, because we were not prepared it nearby. Bronchoscope should be prepared nearby always because it can diagnose laryngospasm with certainty and also can treat it using local anesthetics over the vocal cords from bronchoscope. We also did not attempt the Larson's manoeuvre to relief laryngospasm because of our ignorance, we should acknowledge this valuable technique [15].

Conclusion

In supine patients who are emerging from anesthesia, withdrawing the nasogastric tube can potentially cause laryngeal irritation by gastric acids, thereby provoking laryngospasm. This complication can be potentially avoided by removing the nasogastric tube before the reversal of anesthesia or after the patient is fully awake and no longer supine. We should prepare bronchoscope nearby and know Larson's manoeuvre to relief laryngospasm.

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