Pulmonary Aspiration During Induction of General Anesthesia

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Perioperative pulmonary aspiration of gastric contents can induce complications of varying severity, including aspiration pneumonitis or pneumonia, which may be lethal. A 34-year-old man with no significant medical history presented to Okayama University Hospital for extraction of the third molars and incisive canal cystectomy under general anesthesia. He experienced pulmonary aspiration of clear stomach fluid during mask ventilation after induction. After aspiration occurred, the patient was immediately intubated, and suctioning was performed through the endotracheal tube (ETT). An anteroposterior (AP) chest radiograph was obtained that demonstrated atelectasis in the left lower lobe, in addition to increased peak airway pressures being noted, although SpO_2 remained at 96% to 99% at an FiO_2 of 1.0. The decision was made to proceed, and the scheduled procedures were completed in approximately 2 hours. A repeat AP chest radiograph obtained at the end of the operation revealed improvement of the atelectasis, and no residual atelectasis was observed on the next day. Although the patient reported following standard preoperative fasting instructions (no fluids for 2 hours preoperatively), more than 50 mL of clear fluid remained in his stomach. Because vomiting can occur despite following NPO guidelines, the need for continued vigilance by anesthesia providers and proper timely management is reinforced.

Key Words: General anesthesia; Respiratory aspiration; Fasting.

Pulmonary aspiration of gastric contents has remained one of the most feared complications of sedation and general anesthesia because of the potential for lethal consequences secondary to lung injury from particulate matter, acid, and bacteria.[1] Prevention involves strict adherence to preoperative fasting instructions for patients before elective surgery under sedation and general anesthesia. Historically, the median duration of fasting for liquids was reported to be 6 to 9 hours in adults.[2] However, the advantages of shorter fasting times have been established by several randomized clinical trials in which gastric volumes, gastric pH, blood glucose, hunger, and thirst were evaluated after different fasting times.[3] Currently, fasting periods of 2 hours for clear fluids and 6 hours for solid foods are strongly recommended by the Enhanced Recovery after Surgery (ERAS) protocols,[4] American Society for Anesthesiologist (ASA) guidelines,[3] and practice guidelines for intravenous sedation and general anesthesia throughout dentistry.[5] In this case report, an otherwise healthy male patient experienced acute intraoperative regurgitation and pulmonary aspiration of clear gastric fluids during induction of general anesthesia despite reportedly following the fasting instructions for solid food and fluids. This report describes the course of his treatment and a review of the current literature. Written informed consent was obtained from the patient for the publication of this case report and the accompanying images.

CASE PRESENTATION

The patient was a 34-year-old man (height 159 cm, weight 54 kg, body mass index 21.4 kg/m^2) scheduled to undergo extraction of the bilateral mandibular third molars and removal of a nasopalatine duct cyst. He was otherwise healthy with no notable medical or surgical

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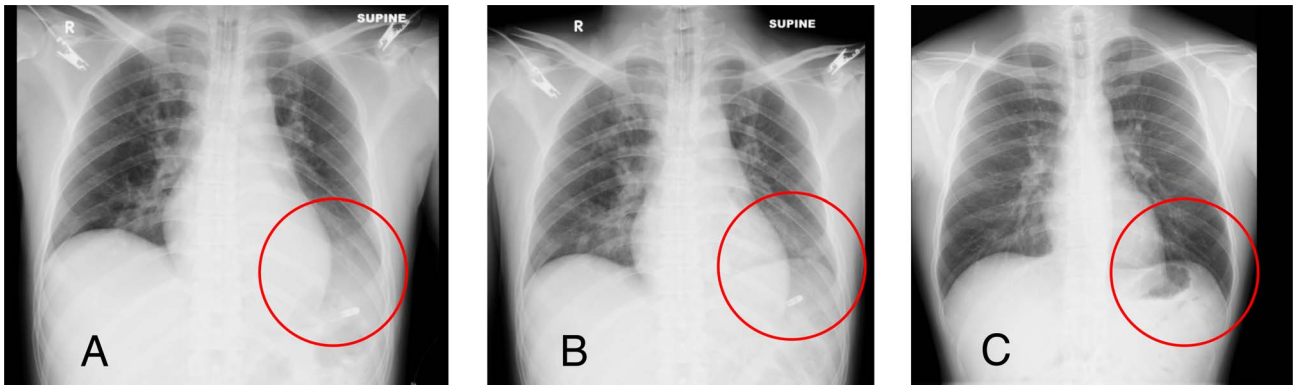

Anteroposterior chest radiographs. Atelectasis was seen in the left lower lobe (red circle) just after tracheal intubation (A), which showed improvement at the end of the 2-hour operation (B). No atelectasis was observed on the next day (C).

history nor any reported allergies or medications. Routine preoperative tests were performed consisting of an AP chest radiograph, electrocardiogram, and blood tests that included a basic metabolic panel, complete blood count, liver function panel, serum lipid panel, and HbA1c, all with no noted significant findings. The patient was admitted the day prior to the scheduled operation. NPO guidelines were followed, with his last solid foods the evening before surgery and clear fluids continuing up to 2 hours prior to surgery. The patient was noted to have consumed 180 mL of water with 18% dextran at 0600 and 0800 hours as well as 150 mL of water at 1000 hours. He was taken to the operating room at 1230 hours.

The anesthetic plan included a nasotracheal intubated general anesthetic. The patient's blood pressure was 127/91 mmHg and pulse was 82 bpm immediately prior to induction. General anesthesia was induced with propofol (100 mg), followed by a continuous infusion of remifentanil (0.4 μg/kg/min) and inhalation of sevoflurane (3%) and O_2 (6 L/min). After loss of consciousness, an intravenous (IV) bolus of rocuronium (35 mg) was administered, and mask ventilation was performed smoothly, without the need for an airway adjunct, at a rate of ~15 breaths/min and a peak pressure less than 20 cm H_2O. No clinically appreciable insufflation of the stomach was noted at this time. During mask ventilation, the patient's heart rate (HR) decreased from 70 to 50 bpm, while his blood pressure was noted to be 125/96 mmHg. Although the bradycardia lasted for only 20 seconds, no significant decreases in blood pressure were observed. Notably, the interval for blood pressure measurements was set at 1 minute throughout this period. At this point, clear fluid was observed overflowing from the patient's mouth. The fluid was immediately evacuated from the oral cavity and upper airway, after which the mask ventilation continued easily. There were no signs of airway obstruction before or after emesis. SpO_2 remained stable at 99% to 100%, and no signs of obstruction were evident via capnography, such as a clear elevation in $EtCO_2$ and/or "shark-fin" appearance of the waveform. The evacuated clear fluid volume was estimated to be >50 mL. The decision was made to intubate the patient, so a preformed 7.5-mm nasal ETT was inserted into the trachea under direct laryngoscopy using a traditional laryngoscope with a Macintosh size 3 blade. The ETT was secured with tape at a depth of 29 cm, and the cuff was inflated with 7 mL of air.

Since pulmonary aspiration and residual fluids in the lower airway were suspected, a 12 French suction catheter (Argyle, Covidien, Dublin, Ireland) and a 12 French orogastric tube (Kangaroo New Enteral Feeding Tube, Covidien, Dublin, Ireland) were inserted through the ETT and the esophagus, respectively. Fluids were suctioned through both the ETT and the orogastric tube, evacuating any residual gastric contents (totaling ~50 mL of clear fluids).

Harsh bilateral breath sounds were then noted upon auscultation, but it was difficult to identify specific areas. No decrease in SpO_2 was observed after intubation, nor were there any notable changes in peak airway pressures or $EtCO_2$ morphology. Direct fiberoptic bronchoscopy was performed by an anesthesiologist, revealing aspiration of clear fluids (~20 mL) into multiple bronchi, which were evacuated as completely as possible. Lung recruitment maneuvers were performed, and an AP chest film was obtained that showed atelectasis in the left lower lobe (Figure A). Elevated peak airway pressures of 28 cm H_2O were noted, at a tidal volume of 500 mL, a positive end-expiratory pressure (PEEP) of 5 cm H_2O, and a respiratory rate of 10 breaths/min. The results of an arterial blood gas analysis obtained from an arterial line that was placed in the right radial artery demonstrated adequate oxygenation despite slightly impaired ventilation and respira-

Arterial Blood Gas Analysis*

	After Intubation	*End of Operation*	*Recovery Room*
pH	7.323	7.339	7.384
PaO_2, mmHg	127.7	297.2	182.5
$PaCO_2$, mmHg	53.5	49.5	42.0
FiO_2	0.5	1.0	—
P/F ratio	255.4	297.2	—

* Arterial blood gas data following intubation, at the end of operation, and 1 hour later in recovery. FiO_2 and P/F were not calculated in recovery since the patient was breathing 4 L/min of oxygen using a face mask.

tory acidosis, as evident by the modest elevation in $PaCO_2$ and decreased pH (Table). The PF ratio (PaO_2/FiO_2), which can be used to identify acute hypoxemic respiratory failure, was 255, which is suggestive of a mild acute lung injury.[6] Despite demonstrating mild respiratory impairment, the patient was otherwise stable. The decision was made to continue after factoring in the small nature of the planned procedure that was expected to only take ~2 hours and the patient being otherwise healthy without any risk factors for atelectasis, such as lung disease, neuromuscular disease, obesity, and age.[7]

General anesthesia was maintained with a continuous infusion of remifentanil (0.15–0.2 μg/kg/min) and sevoflurane (2%) at an FiO_2 of 0.5, using the same ventilator settings as described above. Peak airway pressures remained high (28 cm H_2O), but the harsh breath sounds mildly improved without any bronchodilators. Infiltrative local anesthesia was performed with 1% lidocaine (240 mg) with 1:100,000 epinephrine (0.24 mg). The patient became mildly tachycardic (~110 bpm) roughly 1 hour after starting the operation, which was successfully managed with a continuous infusion of landiolol (0.015 μg/kg/min), subsequently decreasing the HR (~90 bpm). An IV bolus of betamethasone (4 mg) was administered in the middle of the operation for management of postoperative edema. At the end of the surgical procedure, IV acetaminophen (1000 mg) and a diclofenac suppository (50 mg) were administered for postoperative analgesia. The total operating time was 125 minutes. A repeat arterial blood gas (ABG) showed modest improvement in oxygenation upon the increase in the FiO_2 to 1.0 and minimal improvement in ventilation (Table). A repeat AP chest film demonstrated improvement of the aforementioned atelectasis (Figure B). The patient was otherwise deemed stable and fit for emergence and extubation. Sevoflurane and remifentanil were discontinued, and after 10 minutes, the patient was extubated without difficulty, awake and stable, without any appreciable clinical signs of respiratory distress. The patient's HR in the recovery room was ~110 bpm, which was presumed to be due to postoperative pain, although the patient reported well-controlled pain later in recovery. A final ABG drawn in recovery with the patient receiving supplemental oxygen via a simple face mask (4 L/min; FiO_2 ~0.4) indicated further normalization of oxygenation and ventilation (Table).

A repeat chest film taken the next day showed complete resolution of the perioperative atelectasis (Figure C), and there were no abnormal findings upon general examination of the patient. He was discharged from the hospital 2 days after the operation without any complications.

DISCUSSION

Acute intraoperative aspiration is a potentially fatal complication with significant associated morbidity. Several common anesthetic agents, including propofol, volatile anesthetics, and opioids, are known to cause a decrease in lower esophageal sphincter tone.[8] Patient-specific factors, such as systemic diseases and obesity, can also increase the risk of aspiration.[7] In addition, the use of improper clinical anesthetic techniques can be another potential contributing factor, including excessive pressures during mask ventilation. Failure to employ preventive measures, such as the use of cricoid pressure during rapid sequence induction, would be another example.[8]

The ASA has official published guidelines that discuss the importance of adhering to preoperative fasting as well as the use of various pharmacologic agents to help reduce the risk of pulmonary aspiration.[3] Although these guidelines were followed, the patient still regurgitated copious amounts of clear fluid after induction of general anesthesia. One possible cause is persistent residual fluid in the patient's stomach. In this patient, the volume of evacuated fluids based on measuring the suction cannister after clearing the patient's mouth and upper airway plus suctioning from the ETT was estimated to exceed 100 mL (>1.5 mL/kg). It has been reported that 6.2% of the elective surgical population (age >16 years, ASA physical status I-III) have a full stomach (defined as solid contents or a fluid volume >1.5 mL/kg) despite following the recommended fasting guidelines.[9] According to this definition, this patient had a full stomach. Thus, although a 2-hour fasting time for clear fluids has been established as safe for induction of general anesthesia, it should be recognized that large volumes of fluids may be present in the stomach of some patients, even those who are otherwise healthy, at the time of induction. When considering the potentially limited response that may

be available in the office-based environment, it may be advantageous to use a more conservative approach (eg, >2 hour for clear fluids) to NPO guidelines.

In this patient, the HR decreased from 70 to 50 bpm immediately before clear fluids began pouring from the patient's mouth, suggesting increased parasympathetic nervous system activity at that time. A decrease in HR may be attributed to vagal reflexes caused by regurgitation of the stomach contents into the esophagus. Swallow syncope is a rare but an established pathophysiological condition that has been associated with life-threatening bradyarrhythmias when swallowing large quantities of solid food or fluids.[10,11] It was reported that balloon inflation in the mid to lower esophagus resulted in a 5.6-second sinus pause in a patient with swallow syncope.[10] Thus, in this case, it is possible that regurgitation of the stomach contents into the esophagus might have precipitated the transient bradycardia through a mechanism similar to that of swallow syncope. This patient did not have a history of swallow syncope, but it is unclear if such a patient has a higher risk for bradycardia caused by regurgitation during induction of general anesthesia. Despite the short 1-minute blood pressure measurement interval, the transient bradycardia lasted for only 20 seconds. Therefore, it is possible that any significant decrease in blood pressure that might have occurred at that time remained undetected.

The vomiting might also have been brought about by activation of the trigeminocardiac reflex (TCR), which can lead to the sudden onset of parasympathetic dysrhythmias, hypotension, apnea, and/or gastric hypermotility.[12] Gastric hypermotility due to increased parasympathetic activity[13] could have resulted in vomiting. However, because TCR stimulation is most often related to invasive surgical manipulation, such as skull-base interventions, ophthalmic surgery,[14] and surgical reduction of facial fractures,[15] stimulation via mask ventilation is not likely to have induced such a response. It is also possible that the decrease in HR during induction of general anesthesia was induced by the anesthetic agents to some degree.

Artificial and assisted ventilation can lead to vomiting if performed with a face mask or other supraglottic airway devices, because air can insufflate the stomach if excessive pressure (>20 cm H_2O) is used or if airway patency is otherwise lost.[16] The risk of regurgitation is also elevated for patients who are difficult to mask ventilate or require higher positive airway pressures secondary to factors such as obesity, micrognathia, and so forth. In addition, the anesthetic agents administered during induction can induce relaxation of the esophageal sphincter,[8] which may have contributed to regurgitation of the stomach contents.[1] As such, anesthesiologists must remain aware that the time between induction to securing the airway carries a higher risk for vomiting and subsequent aspiration. Smooth mask ventilation was confirmed, and abdominal distension was not observed in this patient. However, it is possible that mask ventilation may have contributed to some extent to the patient's regurgitation.

In this case, since the vomiting was clearly witnessed after induction but before intubation, the fluids were promptly suctioned and the airway secured immediately. Pulmonary aspiration was suspected after suctioning fluid from the ETT, in addition to the noted increases in peak airway pressure and course breath sounds. Bronchoscopic evaluation was performed, as it is useful for diagnosing pulmonary aspiration and assisting in clearance of the airway. In this case, suctioning with the bronchoscope was performed through the tracheal tube.[17,18] In addition, an AP chest radiograph was obtained to further assess for evidence of pulmonary aspiration (ie, atelectasis). Once the airway was secured, the patient was ventilated with PEEP, which likely helped improve the atelectasis of the left lower lobe. The patient remained stable despite mild ventilatory impairment, and as a result, increased PEEP levels were not warranted nor was there a need for additional postoperative treatment. Thus, even though the patient vomited and aspirated clear fluid during induction of general anesthesia, this did not affect the patient's outcome. Fasting times for food and fluids have been shortened in the past few decades to promote postoperative recovery and reduce hunger and thirst. The safety of the routine fasting schedule for food and fluids has been well established, but it should be noted that residual gastric contents may persist and become problematic if vomiting occurs during the course of an anesthetic. Clinicians should remain vigilant especially during times when the airway is at risk and be prepared to provide timely appropriate treatment.

CONCLUSION

In the present case, unexpectedly high volumes of residual fluids were regurgitated and aspirated despite adherence to customary NPO guidelines, suggesting that even healthy adults may be at risk for pulmonary aspiration during general anesthesia even when appropriately NPO. Therefore, to prevent serious complications from regurgitation and aspiration of the stomach contents, anesthesia providers must remain vigilant and be prepared to institute proper management, such as immediately securing and clearing the airway. Discontinuing the anesthetic and surgical procedure may be

warranted after such an event, especially if the patient is otherwise unstable.

DISCLOSURE

None of the authors have any conflicts of interest to disclose.

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