Fatal Massive Hemorrhage Caused by Nasogastric Tube Misplacement in a Patient with Mediastinitis

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Nasogastric tube insertion is a routine procedure in medical care. However, misplacement of the tube can cause a variety of complications, which can be life threatening in some instances. We report a case of fatal hemorrhagic shock immediately after nasogastric tube insertion in a patient undergoing debridement by video-assisted thoracoscopic surgery for mediastinitis. Emergency endoscopy showed that the bleeding came from the nasogastric tube which had perforated the esophagus and possibly tore an intrathoracic large vessel. The nasogastric tube insertion was considered to have directly produced the perforation because no esophageal perforation had been found on preoperative endoscopy. Factors contributing to the risk of esophageal perforation in this case included coexisting mediastinitis, surgical manipulation, endotracheal intubation, inability to cooperate during general anesthesia, and repetitive advancement of the nasogastric tube. Prompt clamping of the nasogastric tube or delayed insertion after failed attempts might have improved the outcome. This report illustrates the complication of massive bleeding that can occur immediately after misplaced insertion of a nasogastric tube. Extraordinary care should be taken to avoid misplacement of the nasogastric tube during insertion. [J Formos Med Assoc 2006;105(1):80–85]

Key Words: fatal outcome, hemorrhagic shock, nasogastric intubation, postoperative complications

Nasogastric tube insertion is a routine procedure in medical care. The maneuver is generally performed without visualization, but it is usually safe. A number of complications associated with nasogastric tube insertion have been reported, including tube knotting around a tracheal tube or over the epiglottis, inadvertent passage into the tracheobronchial tree causing pneumothorax, hydrothorax and empyema, intracranial malposition, misplacement into the Eustachian tube, duodenorenal fistula and urinary bladder perforation, and arterial-esophageal fistula and Mallory-Weiss tear leading to fatal upper gastrointestinal bleeding. These complications rarely occur, but when they do, they can be life threatening. We describe a fatal consequence of nasogastric tube misplacement in a patient under general anesthesia for surgical treatment of mediastinitis. The misplacement resulted in esophageal perforation and possible tear of an intrathoracic large vessel leading to acute hemorrhagic shock.

Case Report

A 70-year-old woman, weighing 66 kg, with no remarkable prior medical history presented for emergency debridement of a mediastinal abscess by video-assisted thoracoscopic surgery (VATS). Six weeks previously, she had been sent to our emergency room due to sudden loss of consciousness. She had suffered from two episodes of syn-
cope in the previous 2 months. On arrival at the emergency room, electrocardiogram showed ventricular tachycardia (VT), and cardiopulmonary resuscitation (CPR) was instituted. After recovery, she received temporary pacemaker placement and amiodarone treatment because of recurrent VT. She was then admitted to the intensive care unit (ICU) for subsequent implantable cardioverter defibrillator (ICD) placement. However, she developed intermittent spiking fever and leukocytosis during hospitalization before ICD placement. Blood culture revealed bacteremia of multiple microorganisms. Broad-spectrum antibiotics were given but they were ineffective. Transesophageal echocardiography, colonoscopy, gallium scan, and computed tomography (CT) of the abdomen and pelvis were performed to identify the source of infection, but no definite finding was made. Cough and dyspnea ensued a few days later. Rhonchi were heard on auscultation and desaturation gradually aggravated. Emergency chest CT revealed mediastinal abscess containing air bubbles, with thickened esophageal wall and pleural effusion (Figure). Due to rapidly exacerbating septic shock and respiratory failure, the decision to perform surgical debridement by VATS was made.

On arrival in the operating room, she was breathing with a nasal cannula and the pulse oximeter read only 86%. Respiratory rate was 28/min, blood pressure was 160/72 mmHg, and heart rate was 85 bpm. She had a nasogastric tube in place for feeding because of poor oral intake. Anesthesia was induced with intravenous fentanyl 100 μg, lidocaine 50 mg, propofol 100 mg, cisatracurium 8 mg, and the patient was smoothly intubated with a 35 French left-sided double-lumen tube. A catheter was inserted into the left dorsalis pedis artery for invasive blood pressure monitoring. Anesthesia was maintained with propofol and cisatracurium infusion. Upper gastrointestinal endoscopy was performed first, but no definite lesion was identified. The patient was then turned onto the left decubitus position and the thoracoscope was introduced smoothly. About 20 mL of brownish purulent discharge was drained from the anterior mediastinum. Saturation fluctuated between 87% and 94% during one-lung ventilation. The remainder of the operation was uneventful.

After returning the patient to the supine position in preparation for transfer back to the ICU, the nasogastric tube slid out. The surgeon decided to re-insert it on the spot. The first two attempts at re-insertion failed as the tube was advanced with great resistance and no sound was heard by auscultation of the left upper abdomen while insufflating air into the tube. The nasogastric tube was advanced smoothly on the third attempt. About 40 mL of air was forcefully insufflated through the tube, but there was still no sound by auscultation. Suddenly, copious fresh dark red blood emerged from the nasogastric tube after disconnecting the tube from the test syringe. Volume replacement with crystalloid fluid was started immediately. Systolic arterial pressure dropped from 145 to 105 mmHg within 1 minute. A central venous catheter was inserted via the left femoral region for aggressive volume administration and transfusions. Systolic blood pressure dropped rapidly to 40 mmHg despite vasopressor use, including epinephrine 20 mg and epinephrine 0.5 mg. Electrocardiogram showed bradycardia followed by VT. Cardiac massage was initiated immediately. Cardioversion with a 200 J shock was given, but the rhythm shifted to frank asystole. CPR was continued while emergency anterolateral thoracotomy was performed for direct cardiac massage. The surgeon decided to perform exploratory laparotomy to identify the source of bleeding, but no intra-abdominal bleeding was found. The descend-

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**Figure.** Preoperative computed tomography of the chest shows a 3.3-cm mass lesion with fluid content and air bubbles at the right mediastinum with esophageal involvement. Thickened esophageal wall and bilateral pleural effusion are also apparent.
ing aorta was temporarily clamped in an attempt to stop bleeding. However, blood continued to flow out from the nasogastric tube, rapidly filling a total of three nasogastric collecting bags. Since cardiac rhythm did not recover after these treatments, extracorporeal membrane oxygenation (ECMO) was established. Sinus rhythm finally resumed after ECMO support. The nasogastric bag was clamped at this time, and total blood loss was estimated to be approximately 4000 mL. No apparent bleeding was found from the mouth or nose after the nasogastric bag was clamped. A total of 12 units of packed red blood cells, 13 units of whole blood and 24 units of platelet concentrate were given. The hemoglobin level was 10.7 g/dL after ECMO support.

Repeat endoscopy showed no signs of recent active bleeding in the upper gastrointestinal tract. The nasogastric tube was found to have produced a false lumen in the posterior lateral wall at the upper third portion of the esophagus and the distal part of the nasogastric tube seemed to have perforated the esophageal wall, which was otherwise intact as seen preoperatively. Without tracing its tip, the nasogastric tube was removed without resistance. After its removal, no more bleeding was found and vital signs were stable without aggressive volume supplementation. After gastrosomy, jejunostomy, chest tube placement and wound closure, the patient was transferred to the ICU. Vital signs were maintained by ECMO and incremental doses of inotropics. Continuous arteriovenous hemodialysis was planned because of anuria. Chest roentgenography showed prominent bilateral lung markings and subcutaneous emphysema over the right chest wall. Pupils were fully dilated without light reflex 2 hours after ICU admission. Because of the dismal prognosis, her family requested against-advice discharge 2 days after the operation.

Discussion

This report describes a fatal case of esophageal perforation resulting from nasogastric tube insertion in a patient with mediastinitis. The cause of death was related to acute massive bleeding, which appeared to have been caused by misplacement of the nasogastric tube into an intrathoracic large vessel. Improper placement of the nasogastric tube can lead to a variety of serious complications. The incidence of malpositioning of the nasogastric tube into the tracheobronchial tree was reported to range from 0.3% to 15%. Penetration of the nasogastric tube into the airway may cause pneumothorax and empyema. Intracranial placement of the nasogastric tube is most commonly seen in patients with craniofacial fracture or surgery of the skull base, and the mortality rate can be as high as 64%. Nasogastric tube placement can cause fatal massive bleeding by the formation of arterial-esophageal fistula. In this case, however, preoperative endoscopy excluded this possibility. Esophageal perforation is another potentially lethal complication of nasogastric tube placement. However, the cause of death is usually related to infection or sepsis. This is the first reported description of acute fatal bleeding after esophageal perforation by a nasogastric tube.

The causes of esophageal perforation include instrumentation, trauma, spontaneous perforation, foreign body, operative injuries, and tumor. The area at the greatest risk for instrumental injury is the posterior wall of the cervical esophagus passing through the cricopharyngeus muscle. The absence of longitudinal muscle and lack of serosa, as well as narrowing of the esophagus at this level, contribute to the likelihood of perforation. The mortality rate from this complication ranges from 16% to 29%. The common causes of death are respiratory failure and septic shock. Massive bleeding can occur in patients with esophageal varices or thoracic aneurysm. Risk factors for esophageal perforation after nasogastric intubation include preexisting esophageal abnormalities, altered mental status, cervical osteophytes, cardiomegaly, endotracheal intubation, infection near the esophagus, and multiple attempts at nasogastric tube intubation.

Several predisposing factors for esophageal perforation existed in the present case. The ad-
advancement of the nasogastric tube could have been blocked by the in-place endotracheal tube. The nasogastric tube was inserted when the patient was still paralyzed so that she could not aid the procedure by the motion of swallowing, a situation resembling that in patients with altered mental status. In addition, preoperative CT scan of the chest showed mediastinal air and paraesophageal abscess, raising the suspicion of preexisting esophageal perforation. Nonetheless, preoperative endoscopy did not identify this condition. The mediastinitis could have arisen from another source of infection, such as granulomatous inflammation of the lymph nodes, and contributed, in turn, to the vulnerability to esophageal perforation. Manipulation during VATS might also have caused esophageal damage, especially in the case of mediastinal abscess adjacent to the esophagus, and further facilitated the breakdown of the esophageal wall by the nasogastric tube. Consequently, extreme caution against the development of esophageal perforation should be taken when nasogastric tube insertion is indicated under a predisposing condition.

The source of bleeding in our patient remained uncertain. Bleeding from esophageal tears usually stops spontaneously or is controlled by nonsurgical treatment, even though blood loss may be considerable. However, the blood loss in our patient was not due to instrumental damage to the esophagus because endoscopy done after the episode did not reveal any bleeding in the upper gastrointestinal tract. The bleeding may have been caused by direct damage to a large vein anatomically close to the esophagus by the nasogastric tube, judging from the color of the blood and spontaneous cessation of bleeding after removal of the tube. The blood might have originated from a large vein returning into the superior vena cava. Because we did not trace the penetrating nasogastric tube by image study before removing the tube, the vessel responsible for the bleeding could not be established. The pre-existing inflammatory status might have decreased the integrity of the wall of adjacent vessels. The 16 French nasogastric tube might have entered into the vein in a way similar to that used for central vein catheterization after passing through the esophagus. The connection of a suction bag to the tube further provided a negative pressure, facilitating the outflow of the blood with a gravitational gradient of about 1 m in height. Therefore, venous return and ventricular preload diminished rapidly. Vigorous fluid resuscitation and transfusion might have been ineffective due to shifting of the volume from the femoral vein to the nasogastric bag before reaching the right atrium. We did not clamp the nasogastric tube immediately due to initial suspicion of bleeding from the gastrointestinal tract. Retrospectively, early clamping of the nasogastric tube might have limited the amount of hemorrhage and improved the efficacy of fluid resuscitation. Moreover, if the bleeding was suspected to be arterial, it would be judicious not to remove the nasogastric tube immediately after clamping it.

Several techniques can be helpful in difficult nasogastric tube insertion, such as lateral neck pressure and forward displacement of the larynx, which aid in nasogastric tube placement by converting 85% of failures into successes. Several basic rules should also be kept in mind, including adequate lubrication, avoidance of force when resistance is met, and consideration of fluoroscopic or endoscopic guidance when risk factors for esophageal perforation are present. The optimal timing of nasogastric tube insertion should also be considered. In the present case, nasogastric tube insertion could have been delayed until after recovery from anesthesia and extubation to enable the patient to cooperate during the procedure and to avoid the endotracheal tube blocking its passage, even though this strategy could cause more patient discomfort. In addition, gastrostomy should be considered early in the case of difficult nasogastric tube insertion.

In this patient, the loss of intravascular volume initially resulted in the development of bradycardia and VT instead of tachycardia as is typically seen. A preexisting cardiac rhythm problem might have played a major role in this manifestation and also the refractoriness to CPR. Air embolism may also have played an important part. Unaware of
the intravascular placement of the nasogastric tube, about 40 mL of air was rapidly injected in order to confirm its correct position by auscultation and which resulted in direct intravascular injection of air into a vessel close to the right heart. The lethal volume of intravascular air injection has not been unanimously agreed upon. The lethal dose of air was reported to be 7.5 mL/kg in dogs and 0.55 mL/kg in rabbits.22 The lethal volume of air in humans has been estimated to be 200–300 mL based on previously reported cases.22 However, the minimum lethal volume was probably less than the estimated amount because some air bubbles were found to be trapped along the venous channels leading to the right side of the heart.22 The volume of air in this case was thought to be smaller compared to that in the previously reported lethal cases. However, the speed of injection and the point of entry close to the heart may also have had adverse effects. Because of the rapid onset of asystole, electrocardiogram characteristic of air embolism was not recorded. Transeosophageal echocardiography is contraindicated in cases of esophageal pathology, and transthoracic echocardiography was hindered by the dressing over the chest. Although the role of air embolism in the present case could not be clearly established, it does illustrate the importance of aspiration first before insufflating air through the nasogastric tube.

In conclusion, this case of fatal massive hemorrhage was caused by misplacement of a nasogastric tube. The nasogastric tube was found to have perforated the esophagus and apparently entered into a large vessel. Contributing factors included coexisting mediastinitis, surgical manipulation, inability to cooperate during nasogastric tube insertion under general anesthesia, hindrance of normal passage by the endotracheal tube, and repetitive attempts at nasogastric tube insertion. Accidental air embolism might also have contributed to the fatal outcome. Awareness of the possibility of nasogastric tube malposition and its associated morbidity is important, especially in patients at increased risk of complication. Prompt identification of the complication and appropriate management may improve the clinical outcome.

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