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A Complication of Nasogastric Intubation: Pulmonary Hemorrhage

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Although insertion of a nasogastric (NG) tube is usually an innocuous procedure, both trivial and life-threatening complications have been described. Inadvertent intubation of the tracheobronchial tree with an NG tube has occurred most frequently in obtunded, uncooperative, or anesthetized patients with occasionally resulting hypoxia, atelectasis, pneumothorax, pleural effusion, or bronchopleural fistula.[1,2] We describe below a massive intrapulmonary hemorrhage following the insertion of an NG tube into the tracheobronchial tree of an awake, alert, and cooperative patient.

REPORT OF A CASE

An 82-year-old man was admitted electively for resection of the left distal clavicle to alleviate chronic pain secondary to an acromioclavicular separation sustained in a motor vehicle accident. Medical problems included mild chronic obstructive airway disease and a hiatal hernia. Analysis of arterial blood gases preoperatively with a $F_{I_{O_2}}$ of 0.21 reveal a *p*H of 7.38, Pa_{CO_2} of 35 mmHg, and Pa_{O_2} of 87 mmHg. Pulmonary function testing revealed a diminished forced vital capacity of 48% and peak air flow of 37% of the predicted value. After an uneventful operative procedure, the trachea was extubated in the recovery room when the patient became agitated and hypertensive with adequate ventilatory variables. Shortly thereafter, cyanotic mottling, diminished air movement with bilateral wheezes, and rhonchi necessitated reintubation of the trachea and controlled ventilation. Immediately before reintubation of the trachea, the pH_a was 6.98, Pa_{CO_2} 75 mmHg, and Pa_{O_2} 42 mmHg. A chest roentgenogram revealed massive bilateral pulmonary edema. Copious quantities of frothy, endotracheal secretions had a colloid oncotic pressure of 8.0 mmHg, compared with the plasma COP of 20.3 mmHg. A 12-lead electrocardiogram ws remarkable for 2.5 mm ST segment depression in leads V_3–V_6. The overall clinical picture was one of acute cardiogenic pulmonary edema associated with hypoxemia, metabolic and respiratory acidosis, and myocardial ischemia.

The patient was treated with controlled ventilation with 10 cm H_2O PEEP, 5 $\mu g \cdot kg^{-1} \cdot min^{-1}$ of dopamine and 20 mg iv furosemide. The EKG reverted to the baseline, and cardiac enzymes subsequently proved negative for myocardial necrosis. A chest roentgenogram taken 8 h after the initial decompensation revealed substantial clearing of the pulmonary edema. Gastric distension was noted, and a NG tube was inserted with return of gastric fluid. Twenty-four hours after the development of pulmonary edema, the patient was weaned from controlled ventilation and the trachea extubated.

Approximately 90 min after tracheal extubation, the patient removed his NG tube. He was awake, alert, and fully oriented. An experienced intensive care unit nurse reinserted the NG tube with the patient in the sitting position. Tracheal placement was immediately suspected when initial passage evoked coughing, and the tube was withdrawn. A second attempt elicited no cough; the patient remained able to converse; and gurgling sounds were heard over the left upper abdominal quadrant with insufflation of air through the tube. However, immediately upon applying suction to the tube, 1 l of bright red blood was drained within 5–10 min. Tube placement was verified again by auscultation over the left upper quadrant, and no blood was found in nares or oropharynx. There was no cough. The patient remained conversant, alert, and oriented.

The apparent diagnosis was an acute GI hemorrhage, most likely from a gastric stress ulcer. Iced saline lavage via the nasogastric tube was begun. The magnitude of the hemorrhage rapidly decreased, but the returning lavage fluid remained blood tinged. Over the period of 90 min after the initial hemorrhage, while lavage was continuing, the patient became increasingly agitated and hypertensive. Adequate oxygenation was documented by serial analysis of arterial blood gases, although a progressive increase in the alveoloarterial O_2 gradient was noted. The patient reported left anterior chest pain and shortness of breath, with no relief obtained from either sublingual nitroglycerin (repeated three times) or intravenous morphine. The stress of hemorrhage and subsequent events was suspected to have precipitated a recurrence of cardiac dysfunction. To arrest further deterioration and relieve the patient's respiratory distress, controlled ventilation was reinstituted. Laryngoscopy was facilitated with thiopental and succinylcholine, iv, but intubation of the trachea was unexpectedly difficult, in part because of inadequate visualization of the larynx and also because of resistance encountered in passage of the tube into the trachea. A chest roentgenogram taken immediately after endotracheal intubation revealed a left pleural effusion, with the NG tube traversing the left main stem bronchus and extending to the pleura at the left base (fig. 1).

The NG tube then was removed and a chest tube inserted on the left, with return of 1,500 ml of serosanguineous fluid. The remainder of the patient's care consisted of controlled ventilation, again with modest levels of PEEP, fluid restriction, broadspectrum antibiotics, and supportive measures. His condition gradually improved, and within 48 hours he was weaned from controlled ventilation and the trachea extubated. The chest tube, through which there was never any sign of air leak, was discontinued 4 days after its insertion. A chest roentgenogram 5 days later revealed only partial atelectasis of the left lower lobe. The patient was discharged home on the ninth postoperative day.

DISCUSSION

The above case represents a previously unreported complication of nasogastric intubation, namely, massive

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intrapulmonary hemorrhage. Unintended intubation of the tracheobronchial tree has been reported in a number of previous publications[1-6] and on one occasion, fluids were infused through the misplaced NG tube, resulting in fatal hydrothorax and empyema.[6] In previous reports, the clinical signs of a NG tube or similar device in the tracheobronchial tree have included agitation, hypoxemia, atelectasis (from bronchial obstruction), decreased peak airway pressure, or air leak during positive-pressure ventilation, pneumothorax, and hydrothorax. These patients were anesthetized, obtunded, uncooperative, or had an endotracheal or tracheostomy tube already in place. Our patient was awake and alert, but the trachea recently had been extubated. He may have had residual laryngeal incompetence following extubation, which allowed him to tolerate unintentional reintubation without signs of laryngeal irritation. Obtundation of airway reflexes can persist for 8 h and longer after tracheal extubation.[7]

With regard to bleeding associated with NG tube insertion, the upper airway is certainly the most common source.[8] Return of bloody fluid also has been reported with accidental intracranial placement of an NG tube.[9] Hemorrhage from the esophagus or stomach as the result of trauma during NG intubation is unreported, although indwelling NG tubes have been implicated as a causative factor in erosion of esophageal varices and gastric ulceration.[8,10,11] Therefore, the sudden appearance of blood following NG intubation, where there had been no previous gastrointestinal bleeding, should alert one to the possibility that the source lies outside the gastrointestinal tract.

In this case, iced saline and tap water were infused into the patient's lung. Parallels may be drawn to salt and fresh water near drowning. Both can cause pulmonary edema, the former due to hypertonicity of fluid, the latter to surfactant destruction and damage to the alveolocapillary membrane. Alterations in $\dot{V}/\dot{Q}$ matching may be induced by both the physical presence of the fluid and the pulmonary vasoconstriction associated with the cold-solution infusion. Warm saline has been therapeutic for pulmonary alveolar proteinosis.

The course of action for verification of proper NG tube position has been discussed in previous reports of tube misplacement.[1,4-6,12-15] In several previous reports, as in our case, auscultation of gurgling over the stomach while insufflating air was misleading.[1,2,6,12] Clinical signs of laryngeal or tracheal irritation certainly may be lacking, even in an awake patient such as ours. Gastric fluid aspiration via the NG tube is not a reliable sign of proper position. After inadvertent insertion of a NG tube intracranially, aspiration of brain has been mistaken for gastric fluid.[12] Earlier radiographic confirmation of tube position would not have eliminated the morbidity incurred in this case but may have led to earlier discovery and reduced the ill effects. Where clinical deterioration or untoward events appear coincident with NG intubation, especially in weak and debilitated patients whose trachea has been extubated recently, a chest roentgenogram definitely is indicated.

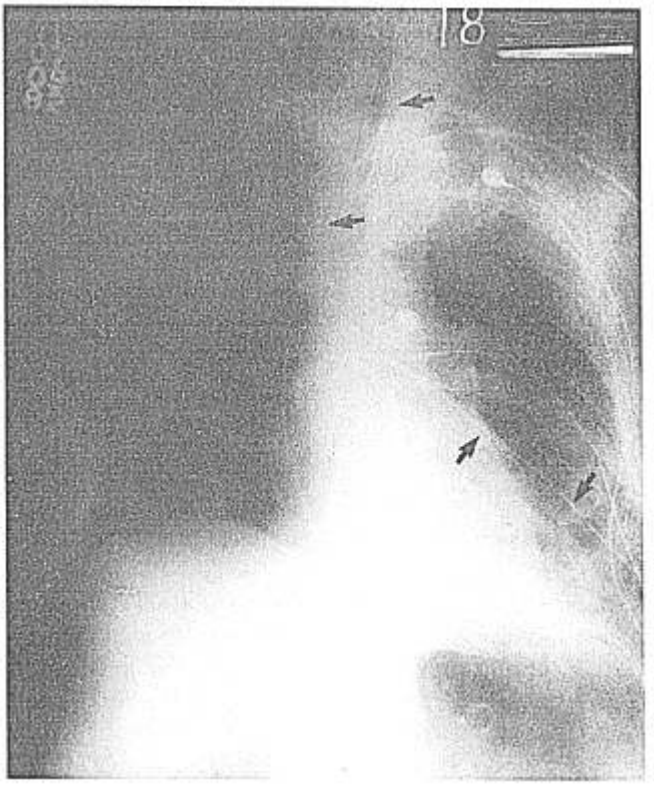

FIG. 1. Nasogastric tube traversing the left main stem bronchus and extending to the pleura at the left base. Arrows indicate the path of the NG tube.

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Prolongation of the Q-T Interval and Sudden Cardiac Arrest Following Right Radical Neck Dissection

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Radical lymph node dissection of the neck with laryngectomy for carcinoma can result in various perioperative complications.[1,2] Among them, prolongation of Q-T interval and severe ventricular arrhythmias with cardiac arrest occasionally have been reported.[3,4] Such a life-threatening complication in a patient who underwent this operation on the right side of the neck and the consideration of published data,[3,5–7] stimulated this study. We investigated on the Q-T interval prolongation with regard to right or left radical neck dissection and the incidence of severe postoperative cardiac arrhythmias.

PATIENTS AND METHODS

Sixty patients scheduled for right or left radical neck dissection consented to be in this prospective study, which covered a period of 9 months. The patients were divided into two groups. Group 1 consisted of 32 patients who underwent right radical neck dissection. Group 2 consisted of 28 patients who underwent left radical neck dissection. Both groups were comparable in age, weight, ASA class, anesthetic technique, and duration of the operative procedure. The preoperative Q-T interval was normal in all patients. We compared preoperative and postoperative Q-T intervals and heart rates. The values were assessed after the first, second, sixth, and twelth postoperative hour, the first postoperative day in all patients and after 2 months in 21 patients. The Q-T interval was measured from the beginning of the Q wave (or the beginning of the R deflection) to the end of the T wave (whether upright or inverted). The Q-T interval then was corrected for heart rate using Bazett's formula ($Q\text{-}Tc = Q\text{-}T/\sqrt{R\text{-}R}$) where Q-T and Q-Tc are expressed in milliseconds (ms) and R-R interval in s. The upper limit of normal of the Q-Tc interval was taken as 440 ms.[4]

Plasma levels of potassium and calcium also were measured 1 h after surgery. All patients stayed in the recovery room for 36 h to have continuous ECG monitoring. Ventilation was controlled via a tracheostomy tube during the first postoperative night.

Intragroup data were analyzed using the paired t test and the intergroup data with the t test for two means. All results are expressed as means ± 1 SE.

RESULTS

Right radical neck dissection resulted in a large and significant increase of Q-T interval (fig. 1) after surgery, as compared with preoperative values ($P < 0.001$). This increase persisted in the 12 patients we were able to follow more than 2 months postoperatively ($P < 0.001$). In contrast, the same surgical procedure on the left side of the neck did not alter significantly the Q-T interval. Significant postoperative increase in heart rate was observed in both groups, as compared with preoperative values ($P < 0.02$).

During the first postoperative days, three of the 32 patients of Group 1 developed severe episodes of tachyarrythmia, causing sudden circulatory arrest. This tach-

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