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Gastric Mucosal Lacerations: A Complication of Cardiopulmonary Resuscitation

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Although rarely recognized clinically, laceration of the gastric or esophageal mucosa occurs in approximately 9–12% of patients who receive cardiopulmonary resuscitation (CPR).[1–3] We treated a patient who developed life-threatening hemorrhage from gastric mucosal lacerations secondary to CPR after he had an acute myocardial infarction and cardiac arrest. General anesthesia for emergent exploratory laparotomy then was required.

REPORT OF A CASE

A 64-year-old man was admitted to a local hospital with an acute inferior wall myocardial infarction. Despite a continuous 2 mg/min iv infusion of lidocaine, initiated because of frequent premature ventricular beats, ventricular fibrillation ensued. The patient received a single precordial thump, followed by external cardiac massage and mouth-to-mouth ventilation for approximately 3 min. Direct current defibrillation with 400 watt-s restored sinus rhythm. A continuous 2 mg/min iv infusion of procainamide was initiated. Three hours later, the patient vomited a large volume of dark red blood and was transferred to our hospital. Initial laboratory analysis revealed a hemoglobin concentration of 15.4 g/dl, K^+ of 4.0 mEq/l, and Pa_{O_2} of 63 mmHg with an FI_{O_2} of 0.2. Endoscopy revealed clotted blood at the gastroesophageal junction but no active bleeding. When bleeding recurred several hours later, a flow-directed, 7 French, quadruple lumen pulmonary artery catheter and radial artery cannula were inserted percutaneously. Twelve units of packed erythrocytes were administered over the ensuing 36 h to maintain a hemoglobin concentration of 11 g/dl. Fiberoptic endoscopy was repeated and revealed active bleeding at the gastroesophageal junction. Multiple attempts to obtain hemostasis with a laser coagulator were unsuccessful. Endoscopy was terminated because of progressive electrocardiographic S-T segment depression in lead II consistent with increased myocardial ischemia. Enzyme analyses revealed a creatine phosphokinase concentration of 1,890 IU/l, 35% of which was myocardial–brain isoenzyme (CPK-MB). Immediate surgical exploration was deemed necessary. Three additional units of packed erythrocytes were administered, increasing the hemoglobin concentration to 13 g/dl. His hemodynamic profile revealed a cardiac index of 2.6 $l \cdot min^{-1} \cdot m^2$, a mean arterial pressure of 92 mmHg, a left ventricular stroke volume index of 23 $ml \cdot m^2$, a left ventricular stroke work index of 25 $gm \cdot m \cdot m^2$, a mean pulmonary artery occlusion pressure of 14 mmHg, a mean pulmonary artery pressure of 21 mmHg, and a central venous pressure of 10 mmHg, with a heart rate of 115 bpm. Systemic vascular resistance was 1,230 $dynes \cdot s \cdot cm^{-5}$, and the pulmonary arteriolar resistance was 105 $dynes \cdot s \cdot cm^{-5}$.

Following inhalation of oxygen and gastric suctioning, the patient's nasal mucosa was anesthetized with cotton pledgets saturated with 4% cocaine solution, and an uneventful nasotracheal intubation was performed without laryngoscopy. Immediately after placement of the nasotracheal tube, thiopental 75 mg was given iv, followed by fentanyl 4 mg, scopolamine 0.5 mg, and pancuronium bromide 11 mg, iv. Nitrous oxide 60% was administered for several minutes and then terminated several minutes later when approximately 10 μg/kg fentanyl had been infused. Fentanyl infusion was maintained throughout the operation to a total dose of 50 μg/kg. Continuous monitoring of the II and V5 leads of the electrocardiogram revealed no evidence of ischemia at any time. The initial arterial blood pressure in the operating room was 170/85 mmHg and heart rate 115 bpm. Arterial blood pressure rose transiently to 180/80 mmHg and heart rate to 122 bpm. Both returned within 5 min to 160/80 mmHg and 112 bpm, respectively. PAOP and PAP did not change at any time.

Exploratory laparotomy revealed a small sliding hiatal hernia and two linear 2-cm mucosal tears of the gastric mucosa at the lesser curvature immediately adjacent to the gastroesophageal junction. Intraoperatively the patient remained stable, with hemodynamic measurements essentially unchanged from the preoperative values. Prolonged central nervous system depression and partial neuromuscular blockade necessitated postoperative ventilation for 6 h. We chose not to reverse the neuromuscular blockade or narcotic depression to avoid possible hemodynamic effects that might increase cardiac ischemia. No evidence of extension of the myocardial infarction was detected with postoperative electrocardiograms and serial CPK-MB measurements. The postoperative course was complicated by a single episode of ventricular fibrillation that was treated successfully with electrical cardioversion. Three weeks later the patient was discharged to his home, doing well on a cardiac rehabilitation program.

DISCUSSION

Gastroesophageal trauma is an increasingly recognized complication of CPR. In its first 4 years of clinical use, external cardiac massage was reported to have caused gastric rupture in three patients. In 1981 Aguilar[6] reviewed reports of 16 cases of gastric trauma complicating external cardiac massage and mouth-to-mouth ventilation. Only three of these 16 patients survived the injury. Two of the survivors died of their primary disease and one recovered completely. Five additional cases of CPR-induced gastric rupture have been reported.[7–11] Even in the absence of transmural rupture, gastric mucosal lacerations can cause fatal hemorrhage.

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Two reports[6,12] describe elderly women who received CPR performed by a lay person for probable syncopal episodes and who died as a result of gastric mucosal lacerations with gastric hemorrhage.

Although clinically recognized gastroesophageal injury secondary to external cardiac massage with assisted ventilation is relatively rare, three autopsy series[1-3] have revealed that gastroesophageal trauma occurs in 9–12% of patients given CPR. Silberberg and Rachmaninoff[1] reported a 2.2% incidence of gastric rupture and a 6.5% incidence of gastric mucosal tears discovered at autopsy following external massage. Lundberg *et al.*[2] and Anthony and Tattersfield[3] reported a 10% and 12% incidence, respectively, of gastroesophageal mucosal lacerations in patients who received unsuccessful CPR.

The mechanism by which mucosal lacerations are created by CPR is unclear. In describing gastric injuries secondary to blunt trauma, Moritz[13] proposed two prerequisites for gastric rupture to occur by compression: ". . . that the stomach not be empty" and ". . . that the external force be so applied as to create a pressure differential between the lumen of the stomach and the peritoneal cavity." Safar[14] reported that mouth-to-mouth ventilation of obese patients or patients with improperly positioned airways could inflate the stomach with as much as 1,900 ml of air. Distention of the stomach with subsequent rupture in the absence of blunt trauma has been reported.[15] Cadaver stomachs will rupture when distended with 4 l of air,[5] but the volume of air required for gastric rupture *in vivo* is unknown.

Lion-Cachet[16] found that unequal distention of the gastric mucosa and musculature was the cause of mucosal tearing in fresh cadaver stomachs distended with water. This observation may explain why most gastric mucosal lacerations occur at the lesser curvature where mucosal elasticity is least.[4,5,8,10]

A high incidence of hiatal hernia and the predominance of lesser curvature gastric mucosal tears in patients with CPR-induced mucosal laceration resulted in the proposal that the intragastric–intrathoracic pressure gradient may be responsible for the laceration, rather than the intragastric–intraperitoneal gradient as proposed by Moritz.[13] Consequently, skillful ventilation, whether mouth-to-mouth or bag-to-mouth, early tracheal intubation, and immediate gastric decompression may decrease the incidence of gastroesophageal trauma in patients requiring CPR.[2,12,17] Postresuscitation gastric suctioning also may facilitate the early diagnosis of gastroesophageal lacerations, should they occur.

Our patient presented with some unusual anesthetic considerations. Because of continued intragastric hemorrhage and intermittent emesis, awake nasotracheal intubation was performed without complication. The primary anesthetic agent, fentanyl 50 μg/kg iv, provided adequate anesthesia without hypotension or clinically significant negative inotropy, as evidenced by absence of change of pulmonary artery occlusion pressure or cardiac output. The combination of fentanyl and scopolamine effectively prevented patient recall of perioperative events, including tracheal intubation. Postoperative evaluation revealed no evidence of extension of the myocardial infarction.

In summary, gastroesophageal trauma occurs in about 10% of patients who receive CPR. Careful airway management, with proper positioning and low-pressure ventilation, early tracheal intubation, and gastric decompression immediately after tracheal intubation may decrease the incidence and severity of this complication. Postresuscitation gastric suctioning may facilitate early diagnosis of gastroesophageal trauma. If surgical therapy is required, invasive hemodynamic monitoring and careful anesthetic management may minimize morbidity and mortality.

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