Gastroesophageal Perforation after Intraoperative Transesophageal Echocardiography

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THE use of transesophageal echocardiography for intraoperative monitoring of cardiac function is increasing. Complications are rare. Oropharyngeal injury and hypopharyngeal perforation during difficult insertion of the echoprobe in anesthetized patients have been reported. [1,2] One case of perforation of the cervical esophagus in an awake patient after multiple attempts at insertion of the echoprobe has been reported. [3] Esophageal perforation from an echoprobe in an anesthetized patient, however, has not been published. We observed and report a case of perforation of the gastroesophageal junction in an anesthetized patient after an easy insertion of the echoprobe that yielded good quality transesophageal echocardiograms.

Case Report

The patient was a 66-year-old man who presented with a cold and pulseless left foot. Angiography on admission revealed that the left limb of an aortobifemoral graft placed 3 yr before admission was occluded, along with the left superficial and deep femoral vessels. His medical history was remarkable for a diminished ejection fraction (0.41) measured 3 yr before admission. Intravenous heparin infusion was begun, and elective surgical endarterectomy of the left limb of the aortobifemoral graft and left femoral-popliteal revascularization was planned.

On the day of surgery, an arterial cannula and a pulmonary artery catheter were inserted before induction of anesthesia. The following data were obtained: blood pressure 175/75 mmHg, pulmonary artery pressure 55/20 mmHg, mean pulmonary capillary wedge pressure 22 mmHg (with a prominent V wave peak measuring 45 mmHg), and cardiac output 4 l/min. Anesthesia and paralysis were induced uneventfully, using fentanyl, etomidate, and succinylcholine, the trachea was intubated, and anesthesia was maintained using isoflurane and fentanyl. After tracheal intubation, a 5-MHz Omniplane transesophageal echoprobe (Hewlett-Packard, Waltham, MA) was inserted to investigate the etiology of the V wave on the pulmonary artery catheter trace. The probe was inserted with ease by an attending anesthesiologist with more than 5 yr experience in cardiac anesthesia and transesophageal echocardiography, including monoplane, biplane, and Omniplane probes. Echocardiographic examination showed normal valvular anatomy without evidence of mitral regurgitation, akinetic posterior wall of the left ventricle, and ejection fraction estimated at 0.35 using end-diastolic and end-systolic cross-sectional areas. At the time of this examination, the following data were also obtained: blood pressure 120/70 mmHg, pulmonary artery pressure 28/15 mmHg, mean pulmonary capillary wedge pressure 12 mmHg without a V wave, and cardiac output 3.1 l/min. Another echocardiographic examination shortly thereafter, when the systemic blood pressure was 160/80 mmHg and a prominent V wave appeared on the pulmonary capillary wedge pressure tracing, showed moderate mitral regurgitation. Blood pressure

was, therefore, targeted and maintained at approximately 120/80 mmHg throughout the operation, which was subsequently uneventful. During retention of the echoprobe in the esophagus, when echocardiographic examination was not being performed, the acoustic power to the transducer was reduced to the minimum level, and the tip of the echoprobe was left in the unlocked position, free of any deflection. Because the patient remained hemodynamically stable, without evidence of mitral regurgitation, the echoprobe was removed after approximately 3 h. At the conclusion of the operation, which lasted approximately 8 h, the patient was awakened, and, after tracheal extubation, transferred to the intensive care unit.

At 4 AM the morning after the operation, the patient complained of nausea, followed 2 h later by severe abdominal pain and a single episode of coffee ground emesis. A nasogastric tube was inserted, and 600 ml coffee ground gastric contents was aspirated, with no bright red blood. During nasogastric tube insertion, the patient experienced tachypnea, hypotension, tachycardia, and agitation, and suffered an episode of arterial hemoglobin oxygen desaturation despite high-flow oxygen via a face mask. The trachea was then intubated. Physical examination showed hypoactive bowel sounds and diffuse abdominal tenderness. Chest and abdominal radiographs showed bibasilar atelectasis, inflammatory infiltrate of the left lung base, diffuse gaseous dilation of small bowel loops, no free intraperitoneal gas, and a mediastinal gas shadow outlining the aortic arch and the descending aorta, consistent with esophageal rupture. A limited gastrointestinal contrast study showed hiatal hernia, with leakage of contrast medium at the gastroesophageal junction. The postoperative hematocrit was decreased from 36 to 28.

An esophageal perforation was diagnosed on the afternoon of the first postoperative day, and the patient was returned to the operating room for a left thoracoabdominal esophagogastrectomy. Gross operative findings were a large perforation at the gastroesophageal junction and a possible esophageal stricture. Pathologic examination of the excised surgical specimen showed a large, 1.2 x 0.7 cm hemorrhagic

perforation at the gastroesophageal junction, adjacent to a much smaller hole measuring 0.3 cm in diameter (Figure 1). The larger perforation showed gross and microscopic evidence of a thermally burned lateral edge, and evidence of acid injury. The esophageal epithelium was pale. Microscopic examination showed extensive necrosis of the esophageal mucosal surface, with underlying lymphohistiocytic inflammation and epithelial sloughing extending 3 cm proximal to the perforation. The perforation at the gastroesophageal junction was surrounded by diffuse lympho-plasmacytic and focal suppurative infiltrations. The microvasculature showed considerable congestion. The gastric mucosa appeared normal, but dilated glands and focal lymphocytic infiltrates suggested mild preexisting gastritis. The pathologic diagnosis was chronic gastritis and esophageal perforation, with ulceration, necrosis, and suppurative inflammation. The extensive necrosis was said to suggest that an element of ischemia was present, in addition to trauma due to instrumentation. Whether the ischemia was secondary to instrumentation or underlying pathology could not be determined.

Figure 1. Esophagogastrectomy specimen, opened to show the lumenal surface. The larger perforation measured 0.7 x 1.2 cm.

The postoperative course was complicated by sepsis, coagulopathy, acute renal failure with oliguria and anuria, acute hepatic failure, and persistent hypoxemia. The patient died on the seventh postoperative day.

Postmortem pathologic examination showed extensive abdominal aortic atherosclerosis, characterized by an occluded celiac artery with multiple hepatic infarcts, a congested firm spleen, and ischemic necrosis of the lower esophagus. There was also marked peripheral vascular disease, coronary artery disease with greater than 70% stenosis of the anterior descending and right coronary arteries, an old myocardial infarction involving the inferior and posterior walls of the left

ventricle, and a 1-cm long area of redness in the lateral ventricular wall. Gastrointestinal findings included focal duskiness of the small bowel suspicious of ischemia, hemorrhagic gastric lining with ulceration, and intact surgical anastomoses and stitches. The immediate and underlying causes of death were considered to be multiple-organ system failure and atherosclerotic vascular disease with occlusion of the celiac artery, which compromised blood supply to the esophagus and liver, respectively. The pathologic impression was that the esophagus was unable to tolerate the pressure of the echoprobe during its retention, which led to necrosis and perforation.

Discussion

Reports of complications of transesophageal echocardiography are the results of retrospective analyses of large numbers of diagnostic examinations in awake, sedated patients. [4,5] Such diagnostic examinations are usually brief, lasting less than 30 min. However, intraoperative transesophageal echocardiography in anesthetized patients for monitoring cardiac function results in retention of the echoprobe in the lumen of the esophagus or stomach for many hours. Although there has been speculation that retention of the echoprobe in the esophagus or stomach for many hours might cause complications, no major complications or permanent injury during anesthesia have been reported.

It is our opinion that the perforation in our patient occurred during retention of the probe and not during its insertion. The insertion was easy, and good quality images were obtained on the first passage from both transesophageal and transgastric positions of the echoprobe, indicating that the echoprobe was within the lumen of the upper gastrointestinal tract. The quality of the echocardiographic images did not change during retention of the echoprobe in the esophagus, suggesting that the probe tip did not migrate. The episode of vomiting or nasogastric tube placement may have contributed to the perforation, but the magnitude of this potential contribution is difficult to assess. Nevertheless, there was more than one

perforation, they were larger than the diameter of a nasogastric tube, and one had an apparently burned lateral edge. Together, these do not support nasogastric tube performation and/or emesis as the primary etiology.

The echoprobe can produce both mechanical and thermal effects. In animals, manipulation and retention of the echoprobe within the esophagus for more than 4 h produced no histopathologic changes. [6] Urbanowicz et al. [7] reported that maximum contact pressure between a fully flexed echoprobe tip and the esophagus was less than 17 mmHg in 5 of 6 patients and 60 mmHg in the sixth patient, who had intrathoracic, but no esophageal, disease. Although pressures less than 17 mmHg might not be expected to cause injury to normal esophageal tissues, an element of ischemia due to occlusion of the celiac artery that was seen in our patient might have increased the susceptibility to pressure necrosis. Heat is generated by two distinct mechanisms. The vibration of the piezoelectric crystal that produces the ultrasound can cause heating of the echoprobe itself. As a safety measure, a thermal sensor in the tip of the echoprobe monitors the temperature, and the power to the echoprobe is automatically shut down when the temperature exceeds 44 degrees C. A postoperative electrical test of our echoprobe showed no abnormalities. Absorption of the ultrasound energy by the tissue can also cause its heating at a distance from the echoprobe, depending on the focus of the beam, that is not detected by the thermal sensor in the echoprobe. [6] Heat by both mechanisms is easily dissipated by tissues with normal blood flow. Underlying ischemia of the esophageal tissues that was likely in our patient would have prevented such dissipation of heat, increasing the susceptibility to thermal injury. The finding of an apparently burned lateral edge of the perforation seen on histopathologic examination of the excised surgical specimen lends support to thermal injury as a contributing factor.

Previous reports of pharyngeal [1,2] or esophageal [3] injury have been associated with difficulty in inserting the echoprobe with immediate or early presentation of obvious signs of injury such as bleeding, [1] appearance of the echoprobe in the

surgical field, [2] and subcutaneous emphysema. [3] In the two cases in which there was perforation into the mediastinum, [2,3] early signs prompted aggressive treatment, so that both patients survived the complications. In contrast, in the current case, no difficulty was encountered during insertion of the echoprobe, echocardiograms of excellent and undiminished quality were obtained, and symptoms and signs of injury appeared more than 20 h after probe use. The delay in recognizing the complication perhaps contributed to the magnitude of the postoperative complications.

It is well established that esophageal injury or perforation after instrumentation is more likely in patients with preexisting disease or abnormality than in patients with normal esophagus. [2,4,5,8] For example, Daniel et al. [4] reported a death due to exsanguination from an undetected tumor infiltrating the esophagus that was injured during insertion of the echoprobe. [4] As such, it is common practice to avoid transesophageal echocardiography in patients with known esophageal disease, such as stricture, burns, cancer, and diverticuli. Most often, suspicion of esophageal disease is raised by presenting symptoms such as dysphagia, regurgitation, and hematemesis, or history of past illness. This report illustrates a case in which there was no symptom or sign of subtle but significant esophageal abnormality due to celiac artery occlusion. The possibility of echoprobe complications in patients with severe occlusive disease of the aorta and upper gastrointestinal tract ischemia should be recognized.

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