Severe Intraoperative Myocardial Ischemia Following Manipulation of the Heart in a Patient Undergoing Esophagogastrectomy

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Ischemia of the myocardium occurs whenever there is inadequate delivery of oxygen to the myocardium secondary to reduced perfusion. In patients undergoing coronary artery bypass grafting, intraoperative myocardial ischemia has been shown to increase the incidence of postoperative myocardial infarction. Patients with known or suspected coronary artery disease undergoing noncardiac surgery may also develop intraoperative myocardial ischemia, although it is unknown whether this increases the incidence of postoperative myocardial infarction (MI).

We report a case of severe intraoperative myocardial ischemia complicated by MI in a patient with risk factors for coronary artery disease undergoing esophagogastrectomy for carcinoma of the esophagus. We describe the clinical presentation of this ischemia and discuss our approach to its treatment.

REPORT OF A CASE

A 60-yr-old, 72-kg man presented with a 2-wk history of nonexertional, atypical chest pain. A thallium exercise stress test to 89% of the maximal predicted heart rate was negative for coronary artery disease and "M" mode two-dimensional, and Doppler echocardiograms revealed dilatation of the aortic root and concentric hypertrophy of the left ventricle. Upper gastrointestinal series with radiographic contrast material demonstrated a distal esophageal mass. Past medical history was significant for hypertension treated with hydrodiuril, cigarette smoking, chronic obstructive pulmonary disease (COPD), and a 4-cm abdominal aortic aneurysm. Arterial blood pressure was 150/100 mmHg, heart rate 80 beats/min, and respirations 16 breaths/min. The rest of the physical examination was unremarkable. Routine laboratory investigations were within normal limits. The forced expiratory volume in 1 s/forced vital capacity ratio was 0.52. The chest radiograph was consistent with COPD. The preoperative ECG revealed sinus rhythm with nonsignificant Q-waves inferiorly and minor T-wave abnormalities (fig. 1). Esophagogastroduodenoscopy confirmed the diagnosis of esophageal adenocarcinoma, and the patient was scheduled for esophagogastrectomy. Preanesthetic medication consisted of 6 mg morphine and 0.4 mg IM scopolamine. After application of a 7-lead ECG (Marquette Electronics 7000) standardized to 10 mm/mV and pulse oximeter, an iv catheter was inserted. The radial artery and right internal jugular vein were cannulated for continuous pressure measurements. The initial central venous pressure (CVP) was 3 mmHg. An epidural catheter was inserted at the L1–2 interspace for postoperative pain control. After negative aspiration, 4 ml of 1% lidocaine with 1/200,000 epinephrine was injected to verify catheter placement. This resulted in transient hypotension (systolic blood pressure 80 mmHg), which responded promptly to 100 µg phentylephrine and 1 L lactated Ringer's solution iv. During this episode the patient was asymptomatic and no electrocardiographic changes occurred. There was no evidence of subarachnoid block. Anesthesia was induced with thiopental (7 mg/kg), fentanyl (2 µg/kg), and pancuronium (0.15 mg/kg) with 100% oxygen. A 30-Fr left robershaw double-lumen endotracheal tube was inserted under direct vision, and its position was verified with auscultation and fiberoptic bronchoscopy. Anesthesia was maintained with isoflurane 1.0–2.0% inspired, and the patient's lungs were ventilated with an N2O/O2 mixture (FIO2 = 0.4) to maintain an arterial PCO2 between 55 and 40 mmHg. Nitroglycerin 50–100 µg/min was infused for blood pressure control (mean arterial pressure of 80–90 mmHg), and dopamine (2.5 µg·kg⁻¹·min⁻¹) was infused to maintain urine output. The patient was placed in the right lateral decubitus position, and the table flexed to open the intercostal spaces on the upper hemithorax. One-lung ventilation was managed with an FIO2 = 1.0. Five-centimeter continuous positive airway pressure was applied to the unventilated lung (FIO2 = 1.0), which maintained the oxygen saturation at 98% and the arterial PO2 between 150 and 240 mmHg. During dissection of the proximal esophagus, the heart and pericardium were manipulated to various positions to attain improved visualization of the line of proposed resection. Approximately 90 s following this manipulation, severe ST-segment elevation (4–6 mm) was noted in the inferior ECG leads with reciprocal changes in the lateral and chest leads (fig. 2). There were no dysrhythmias. The heart rate was 80 beats/min, with a mean arterial pressure of 80 mmHg. The CVP was 15 mmHg. Two nitroglycerin boluses of 400 µg were immediately given iv. Isoflurane was discontinued. The nitroglycerin infusion was increased to 200 µg/min and 20 mg nifedipine was administered sublingually without resolution of the ECG changes. Hematocrit was 50%; arterial blood gas was as follows: pH 7.39, PCO2 59 mmHg, PO2 240 mmHg, FIO2 1.0; and serum potassium was 3.6 mEq/l. Two units of packed red blood cells were transfused. A cardiology consultant agreed that the ECG changes were consistent with acute inferior wall myocardial current of injury and suggested continuing 10 mg nifedipine sublingually every 2 h along with iv nitroglycerin at 100 µg/min as tolerated. Because of the hemodynamic stability and lack of dysrhythmias, surgery was able to be completed under enflurane–fentanyl anesthesia. The ST-segments remained elevated throughout the procedure, although the degree of elevation was not as great (2–3 mm above baseline, fig. 3). Near the conclusion of surgery, 5 mg epidural morphine diluted in 20 ml 0.9% saline was administered.
A flow-directed pulmonary artery catheter was inserted at the end of surgery, and the thermodilution cardiac output was 5.0 l/min with an index of 1.6 l\cdot min^{-1} \cdot m^{-2}. The pulmonary artery pressure was 27/15 mmHg and pulmonary capillary wedge pressure was 15 mmHg, without a significant v-wave. The CVP was 11 mmHg, mean arterial pressure 85 mmHg, heart rate 80 beats/min, and nasal temperature 34.6°C. Dobutamine and sodium nitroprusside were infused, and the patient was transferred to the cardiothoracic intensive care unit where his trachea remained intubated and ventilation controlled. Cardiac index increased over the next 2 h to 3.4 l\cdot min^{-1} \cdot m^{-2}. However, the patient developed a sinus tachycardia (130–140 beats/min), which was unresponsive to 10 mg morphine iv and 10 mg diazepam iv administered in 2.5-mg increments. Dobutamine was discontinued, and esmolol was infused at 50 μg \cdot kg^{-1} \cdot min^{-1} with resolution of the tachycardia within 20 min. Heart rate was 72 beats/min, mean arterial pressure 75 mmHg, pulmonary capillary wedge pressure 15 mmHg, and cardiac index 3.0 l\cdot min^{-1} \cdot m^{-2}. Following 48 h of esmolol therapy, metoprolol was started and esmolol was discontinued. Serial postoperative ECG revealed persistent ST-segment and T-wave abnormalities; however, significant Q-waves did not develop (Figs. 4 and 5). Creatinine kinase MB fraction peaked at 89 IU/l (absolute creatinine kinase = 1571 IU/l). The patient was diagnosed as having had an inferior subendocardial infarction. Echocardiogram 2 wk postoperatively showed concentric left ventricular hypertrophy with normal left ventricular systolic function. The postoperative course was complicated by respiratory failure, pneumonia, and cerebral encephalopathy. The patient was discharged from hospital 4 months after his surgery in good condition. Follow up visits have revealed only mild irregularities in swallowing mechanism.

**DISCUSSION**

The incidence of cardiac complications following esophagogastrectomy is unknown. Ellis and Gibb found that three of 72 patients (4%) suffered cardiac dysrhythmias following surgical resection of their esophageal or gastroesophageal carcinomas. These dysrhythmias were said to be minor in nature and did not prolong hospitalization. Bluett et al. reported a 10% incidence of cardiac complications following esophageal cancer resection in their series of 104 patients, although they did not report what type of complications occurred or whether any of these patients had preexisting cardiac disease.

Perioperative myocardial ischemia has been shown to occur in 24–74% of patients with known or suspected ischemic heart disease undergoing noncardiac surgery. There were several major risk factors for coronary artery disease in our patient (hypertension, cigarette smoking, aneurysmal dilation of the abdominal aorta) despite the negative thallium stress test. Moreover, it is known that thallium exercise testing may fail to detect up to 16% of patients with severe coronary artery disease. Thus, there was a high probability of our patient having coronary artery disease. Perioperative myocardial
ischemia can be precipitated by numerous inciting factors. These include atheroembolization, coronary thrombosis, coronary artery spasms, air embolism, and other physiologic aberrations that result in an imbalance between myocardial oxygen supply and demand.\textsuperscript{14,15} Vasospasm may aggravate atherosclerotic obstructions by further narrowing the vessel lumen or by initiating formation of thrombus with subsequent MI.\textsuperscript{16-19} Moreover, coronary artery vasospasm may be induced by alpha-adrenergic drugs\textsuperscript{20} and respiratory or metabolic alkalosis.\textsuperscript{21} It is hypothesized that in the present patient manipulation of the

\textbf{Figure 3.} ECG immediately postoperatively. The ST-segments remain elevated in leads II, III, and aVF, although the degree of elevation is not as great as intraoperatively.

\textbf{Figure 4.} Twelve-lead ECG on postoperative day 2. The ST-segments still remain elevated in leads II, III, and aVF.

\textbf{Figure 5.} Twelve-lead ECG 1 month after surgery. The T-waves have become inverted in both the inferior and lateral chest leads.
heart resulted in coronary artery spasm causing total or subtotal coronary artery obstruction with secondary thrombus formation. Heart manipulation could also have caused coronary artery kinking with atheromatous plaque rupture, thrombosis, and occlusion of the artery with or without associated vasoospasm. These theories are supported by the fact that the oxygen content of the arterial blood was adequate during the ischemic episode (oxygen saturation 98%, hematocrit 50%), and there were no significant alterations in blood pressure, heart rate, or CVP. The patient had not received any alpha-adrenergic-stimulating drugs since insertion of the epidural catheter, nor did the administration of phenylephrine at that time cause any discernible change in the ECG. As well, arterial blood gas analysis just after the onset of ischemia revealed normal acid-base status. However, the patient was receiving isoflurane 1.2% inspired at the time of ischemia occurrence. Although this drug has been shown to induce redistribution of coronary blood flow away from areas of borderline perfusion to adequately perfused regions (coronary steal) in instrumented dogs, the phenomenon of coronary steal is unlikely to have occurred in our patient because there was a close temporal relation of the ST-segment changes to surgical manipulation of the heart, and discontinuing isoflurane did not relieve the ischemia.

Although ECG changes may occur relatively late during an episode of myocardial ischemia compared with other monitors (i.e., abnormal left ventricular wall motion or wall thickening on a two-dimensional echocardiographic image, total outward motion during systole on the cardiokymogram), the ECG is still the most widely used monitor to detect myocardial ischemia. Moreover, the Marquette® Series 7000 system used in our patient permitted multiple ECG lead display as well as continuous ST-segment sampling and analysis of leads II, III, and V at the J point + 60 ms. This may have facilitated early diagnosis and treatment of the ischemic episode. The patient was receiving a nitroglycerin infusion, 60 µg/min, at the time of the episode for blood pressure control. Although the inferior lead ST-segments remained elevated despite our therapeutic interventions (nitroglycerin boluses, nifedipine), the degree of elevation was lessened from 4–6 mm initially to 2–3 mm approximately 1 h after institution of therapy, and the patient remained hemodynamically stable throughout the procedure without dysrhythmias. This may be due to the physiologic effects of nitroglycerin, which include relief of coronary artery spasm, abolition of normal coronary artery tone in narrowed vessels, and dilation of collaterals and their parent vessels. Also, the mechanism of action of nitrates may be due to systemic veno, arterial, and arteriolar dilatation, which reduces left ventricular end diastolic pressure, left ventricular end diastolic volume, wall stress, and myocardial oxygen consumption, and improves flow to deep myocardial muscle. However, some patients may be unresponsive to as much as 300 µg/min of iv nitroglycerin and may only respond to intracoronary administration of this drug. Nifedipine may exert its beneficial effects via coronary artery vasodilation and/or reduced myocardial oxygen demand secondary to a decreased arterial vascular smooth muscle tone and systemic vascular resistance. Moreover, nifedipine has been shown to be highly effective in relieving coronary artery spasm and may improve left ventricular function in patients already receiving maximal nitrate therapy. Nevertheless, Muller et al. could not demonstrate any beneficial effects of oral nifedipine in patients with evolving MI in terms of progression of threatened infarction to the acute event and limitation of infarct size in patients experiencing infarction. Although the use of iv and intracoronary thrombolytic agents for reperfusion of coronary occlusions may salvage jeopardized myocardium in the setting of evolving acute MI, this option was dismissed in our patient due to the increased risk of intraoperative and postoperative bleeding associated with anticoagulation in the surgical setting.

In summary, the present patient developed severe intraoperative ST-segment elevation and subsequent subendocardial infarction following manipulation of the heart during esophagogastrectomy for esophageal carcinoma. This ischemia may have been a result of coronary artery spasm or rupture of an atheromatous plaque leading to hemorrhage and thrombosis. It is possible that the early electrocardiographic detection of myocardial ischemia and immediate institution of nitroglycerin and nifedipine therapy may have prevented a transmural MI and limited the extent of the surrounding ischemic zone.

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REFERENCES

Systolic Venous Waves Cause Spurious Signs of Arterial Hemoglobin Desaturation

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Pulse oximetry has found extremely widespread use in the perioperative period, and its routine use has been recommended. During cardiac surgery invasive hemodynamic monitoring supplements oximetry and other noninvasive perioperative monitors. Recently, two patients were identified who had abnormal venous waves and coincident apparent arterial hemoglobin desaturation indicated by ear probe pulse oximetry. The combination of pulse oximetry, blood sampling, and observation of the intravascular pressure waveforms allowed the elucidation of an unusual but clinically important problem inherent to pulse oximetry.

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