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–L2 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 phenylephrine and 11 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 thipental (7 mg/kg), fentanyl (2 μg/kg), and pancuronium (0.15 mg/kg) with 100% oxygen. A 30-Fr left röbertshaw 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 (F₂O₂ = 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 F₂O₂ = 1.0. Five-centimeter continuous positive airway pressure was applied to the nonventilated lung (P₀₂ = 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 7.35, pCO₂ 59 mmHg, pO₂ 240 mmHg, HCO₃ 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 instability and lack of dysrhythmias, surgery was able to be completed under enfurane-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.

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A flow-directed pulmonary artery catheter was inserted at the end of surgery, and the thermodilution cardiac output was 3.0 l/min with an index of 1.6 l·min⁻¹·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·min⁻¹·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·kg⁻¹·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·min⁻¹·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). Creatine kinase MB fraction peaked at 89 IU/l (absolute creatine 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 Gibb⁶ 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.⁶ Bluett et al.⁷ 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.³⁻⁵,⁸⁻¹¹ There were several major risk factors for coronary artery disease in our patient (hypertension, cigarette smoking, aneurysmal dilation of the abdominal aorta¹²) 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.¹³ Thus, there was a high probability of our patient having coronary artery disease. Perioperative myocardial

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**Fig. 2.** Intraoperative ECG following manipulation of the heart. Note the marked ST-segment elevation in the inferior leads. Lead V₄ (or V) was located parasternally in the right fourth intercostal space.
ischemia can be precipitated by numerous inciting factors. These include atheroembolization, coronary thrombosis, coronary artery spasm, 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
heart resulted in coronary artery spasm causing total or
subtotal coronary artery obstruction with secondary
thrombus formation. Heart manipulation could also have
casted coronary artery kinking with atheromatous plaque
rupture, thrombosis, and occlusion of the artery with or
without associated vasospasm. These theories are sup-
ported 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 sig-
nificant alterations in blood pressure, heart rate, or CVP.
The patient had not received any alpha-adrenergic-stim-
ulating 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 nor-
mal acid-base status. However, the patient was receiving
isoflurane 1.2% inspired at the time of ischemia occur-
rence. Although this drug has been shown to induce re-
distribution of coronary blood flow away from areas of
borderline perfusion to adequately perfused regions (cor-
nary steal) in instrumented dogs,22 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 car-
diokymogram),3,4 the ECG is still the most widely used
monitor to detect myocardial ischemia. Moreover, the
Marquette® Series 7000 system used in our patient per-
mitted 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 nitrolycerin 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 bol-
uses, 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 hemo-
dynamically 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 nar-
rowed vessels, and dilation of collaterals and their parent
vessels.23–25 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 myocar-
dial oxygen consumption, and improves flow to deep
myocardial muscle.23–25 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.18 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.26,27
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.28 Nevertheless, Muller et al.
could not demonstrate any beneficial effects of oral nif-
edipine in patients with evolving MI in terms of pro-
gression of threatened infarction to the acute event and
limitation of infarct size in patients experiencing infarction.29
Although the use of iv and intracoronary thrombolytic
agents for reperfusion of coronary occlusions may sal-
avage jeopardized myocardium in the setting of evolving
acute MI,30–32 this option was dismissed in our patient
due to the increased risk of intraoperative and postop-
erative bleeding associated with anticoagulation in the
surgical setting.

In summary, the present patient developed severe intra-
operative ST-segment elevation and subsequent sub-
endocardial 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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Pulse oximetry has found extremely widespread use in the perioperative period,\textsuperscript{1-3} and its routine use has been recommended.\textsuperscript{4-6} 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.