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Perioperative myocardial infarction: diagnostic clues and prevention

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ABSTRACT

The diagnosis of perioperative myocardial infarction can be missed if the pain is masked by postoperative analgesia and the possibility is not considered. This report is the case of a patient with a missed diagnosis of perioperative myocardial infarction. Myocardial injury and infarction from noncardiac surgery is currently the subject of intense interest and research. This report illustrates the importance of the diagnosis and suggests clues that can be used to make the diagnosis.

Keywords
Diagnostic errors; Cognitive dysfunction; Myocardial infarction; General surgery; Autopsy

CASE REPORT

This 57-year-old man had severe degenerative joint disease, morbid obesity, hypertension, hyperlipidemia, gastro-esophageal reflux disease, depression and bilateral carpal tunnel syndrome. The patient underwent right hip replacement 14 months prior, which he described as the most painful experience of his life. The hip replacement was complicated by a postoperative pectoral muscle rupture secondary to supporting his weight with a walker. He had a 40 pack-year history of smoking (one pack a day for 40 years). He drank minimal alcohol but was a former heavy drinker. He denied use of recreational drugs. He was a truck driver on disability leave. He had a history of exposure to asbestos for three years. His mother had died of breast cancer at age 42. His father had died at age 67 of a heart attack. The patient's chronic medications were celecoxib, tramadol, sertraline, atorvastatin and omeprazole.

During pre-operative evaluation for right knee replacement, chest x-ray showed a 3.6 cm left lung mass in the lingula. Transbronchial biopsy showed moderately differentiated squamous cell carcinoma. He underwent a complete metastatic work-up including computed tomography (CT) scan of the chest and upper abdomen, which revealed normal mediastinal nodes, no adrenal masses and no liver masses, but a 2.5 cm lingular mass abutting the pericardium at the apex of the heart. Bone scan was negative for metastatic disease. Pulmonary function tests revealed a first-second forced expiratory volume (FEV1) of 3.42 L (90% of predicted), forced vital capacity (FVC) of 4.79 L (102% of predicted) and diffusing capacity of the lungs for carbon monoxide (DLCO) of 24.7 mL/min/mm Hg (75% of predicted). Liver function tests were within normal limits. Adenosine myocardial perfusion study showed some inferolateral and posterolateral wall thinning on initial images with some improvement around the periphery of the defect on the delayed images, particularly in the posterolateral wall. Gated views demonstrated some hypokinesis in the inferolateral wall but the left ventricular ejection fraction was preserved at 62%.

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The radiologist interpreted these findings as consistent with inferolateral and posterolateral wall scarring with some peri-scar ischemia along the posterolateral wall, but a cardiologist subsequently interpreted them as false positive.

The patient was admitted for left upper lobe lobectomy. On admission at 06:15 his white blood cell count was 21,700/mm^3 (reference range [RR]: 4,500-11,000/mm^3) with left shift, hemoglobin 12.8 g/dL (RR: 13.5-17.5 g/dL), platelets 237,000/mm^3 (RR: 150,000-400,000/mm^3), glucose 99 mg/dL (RR: 70-110 mg/dL), cholesterol 181 mg/dL (RR: <200 mg/dL), with normal renal function tests, electrolytes, bilirubin, liver enzymes, and albumin. At 07:30, the patient's temperature was 37 degrees C, pulse 60/minute, blood pressure 132/73 mm Hg, respirations 20/minute, height 183 cm and weight 136.1 kg (body mass index 40,6). During the surgery from 10:15 to 14:50, the patient's pulse ranged from 80/minute to 108/minute and his blood pressure ranged from a transient low of 95/54 mm Hg to a high of 194/98 mm Hg. Intraoperatively at 12:26, arterial blood showed: pH 7.30, PCO2 51 mm Hg, PO2 153 mm Hg, hemoglobin 13 g/dL, and glucose 121 mg/dL. The estimated blood loss was 600 mL. Following the procedure, the patient was extubated without difficulty. He was placed on a cardiac monitor post-operatively which showed a normal sinus rhythm. At 15:00 the patient complained of incisional and left shoulder pain he rated 7/10, which responded to two tablets of combination oxycodone 2.5 mg and acetaminophen 325 mg, along with 5 mg of morphine. At 15:10 chest x-ray showed the left lung expanded, but poor aeration and an enlarged heart.

On postoperative day 1, at 00:45 the patient had left shoulder pain he rated 9/10, which responded to two tablets of combination oxycodone 2.5 mg and acetaminophen 325 mg. At 08:00, he had recurrent pain he described as chest soreness, which responded to 5 mg of morphine. The patient was hoarse. His temperature rose from 36.2 degrees C at 08:00 to 37.5 degrees C at midnight. His pulse was 86-110/minute, blood pressure 101-136/60-74 mm Hg and respirations 16-24/minute. His chest was clear. He had pulse oximeter readings of 96 to 99%. Cardiac monitoring showed borderline sinus tachycardia at a rate of 100/minute, with 1 mm ST-segment depression. The patient's fluid balance was 5115 mL positive. At 11:20 chest x-ray showed clear lungs and unchanged cardiomegaly.

On postoperative day 2, the patient became dyspneic with walking (with the assistance of 2 people). His maximum temperature was 37.9 degrees C, pulse 86-106/minute, blood pressure 110-160/50-80 mm Hg and respirations 18-22/minute. Cardiac monitoring showed sinus tachycardia at a rate of 109/minute, with T-wave inversion. His fluid balance was 225 mL negative. Chest x-ray showed no change. On postoperative day 3, the patient became dyspneic with minimal activity. He was very diaphoretic. His maximum temperature was 37.5 degrees C, pulse 88-100/minute, blood pressure 120-126/70-78 mm Hg and respirations 18-20/minute. His fluid balance was 245 mL negative. His white blood cell count was 15,200/cu mm, hemoglobin 11.1 g/dL, platelets 263,000/cu mm and glucose 119 mg/dL. Left vocal cord paresis was diagnosed. The left upper lobe lobectomy specimen was signed out showing moderately differentiated squamous cell carcinoma, 3.2 × 2.9 × 2.6 cm, with extension of tumor up to but not through overlying pleura, bronchial and vascular margins of resection free of neoplasm, no angiolymphatic invasion and no metastases in 8 lymph nodes, TNM stage 1B, T2 N0 MX. Enoxaparin therapy was initiated for deep venous thrombosis prophylaxis, given the patient's relative immobility.

On postoperative day 5, the patient's voice was still hoarse, but improving. His temperature was 37.7 degrees C, pulse 80/minute, blood pressure 140/80 mm Hg, respirations 18/minute and fluid balance 550 mL negative. He was discharged to his home.

The following day, at home, the patient became short of breath and an ambulance was called. Upon arrival, paramedics found the patient conscious, alert, sitting on the toilet and hyperventilating. The patient's wife was holding his head up. The patient was in obvious respiratory distress, close to respiratory failure. His skin was extremely pale, with cyanosis around the lips and fingernails. The patient was unable to speak more than one word at a time. He was using accessory muscles of respiration. His pulse was 88/minute and respiratory rate 30/minute. A pulse oximeter showed oxygen saturation of 88% on room air. The patient was immediately given supplemental oxygen at 15 L/minute via a non-rebreather. In the ambulance, the patient

was conscious and alert, stating that he could not breathe. On arrival at the emergency department, the patient was transferred to a bed and reported to the staff, but then almost immediately suffered a cardiac arrest. Resuscitation was attempted for approximately 1 hour. The patient could not be resuscitated.

AUTOPSY FINDINGS

Postmortem examination revealed 420 ml of partially clotted blood in the pericardium, associated with a cardiac rupture in the posterior wall of the left ventricle. The posterior wall of the left ventricle showed a 6.5 × 6 × 1 cm transmural acute myocardial infarction with histological features of an infarction 6 days prior (Figure 1).

There was severe coronary atherosclerosis with over 75% stenosis of all three major epicardial arteries and an occlusive thrombosis of the proximal right coronary artery, 1.9 cm from the ostium (Figure 2).

Autopsy also revealed a tiny microscopic old subendocardial myocardial infarction of the septum, passive congestion of the liver, mild steatohepatitis with severe mixed macrovesicular and microvesicular steatosis, and severe acute prostatitis.

DISCUSSION

The antemortem and postmortem evidence suggests that this patient most likely suffered a myocardial infarction during surgery. He had hypotension during surgery, but not in the postoperative period. He had ST-segment depression on postoperative day 1 and T-wave inversion on postoperative day 2. Neither cardiac biomarkers for infarction nor 12-lead electrocardiography were done because myocardial infarction was not suspected. The symptoms of the infarction were apparently masked by the expected symptoms following left upper lobectomy. The histopathologic features of the myocardial infarction at autopsy were typical of an infarct nearing the end of the first week, but they are not specific enough to date the infarction to the intraoperative period. They are specific enough to date the infarction to the perioperative period, and the period of surgery, during which blood clotting is activated, is the most likely time that the thrombosis

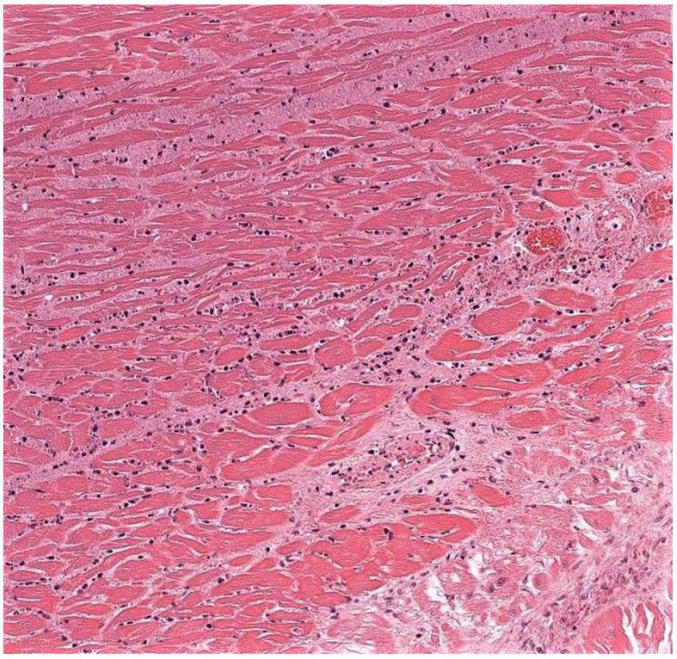

Figure 1. The dead cardiac myocytes (all but lower right corner) show hypereosinophilia and loss of nuclei, associated with an infiltrate of degenerating neutrophils, many of which have lysed, releasing basophilic debris (nuclear dust).

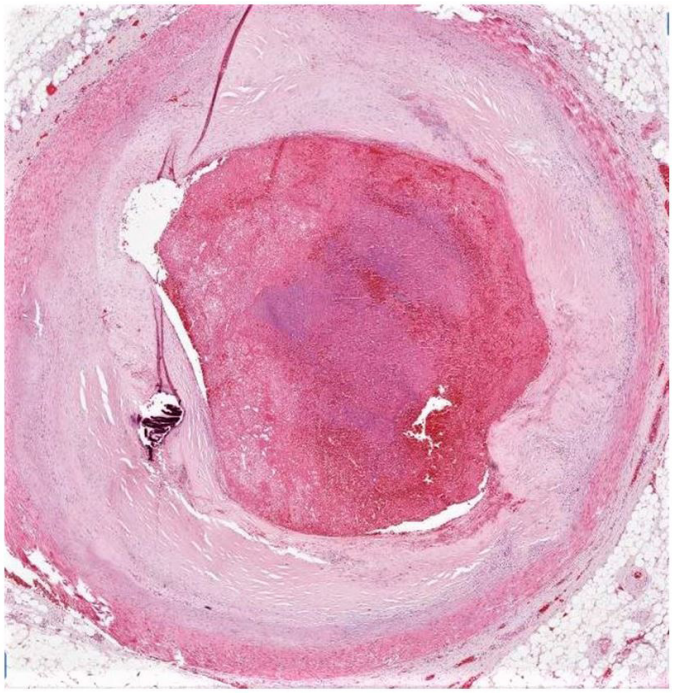

Figure 2. The apparent gaps in this luminal thrombosis are due to artefactual tissue folding, particularly around two calcifications, one of which has been pushed out of the tissue section by the microtome blade.

shown in Figure 2 developed. Ultimately, the infarction led to myocardial rupture and the patient's demise.

Surgery inevitably releases activated clotting factors and platelets into the circulation, so ischemia or infarction of the heart, the brain or other organs at sites of critical atherosclerosis is an inevitable risk of surgery. In an international study of 15,065 patients aged 45 years or older who underwent inpatient noncardiac surgery, among the 1200 (8%) who had a postoperative peak troponin level of 0.03 ng/mL or greater judged due to myocardial ischemia, only 41.8% would have fulfilled the universal definition of myocardial infarction, but 30-day mortality was 3.87 higher.[1] In a follow-up study of 21,842 similar patients, using high-sensitivity troponin T (hsTnT) measurements, 3904 (17.9%) had myocardial injury due to ischemia, but 3633 of these patients (93.1%) experienced no ischemic symptom.[2] In an Indian study of 1075 patients undergoing noncardiac surgery, 188 (17.5%) had a peak 12- or 24-hour postoperative troponin level of 0.03 ng/mL or greater judged to represent myocardial injury, and these patients had a 30-day mortality of 11.7%, compared to 2.5% among the patients without myocardial injury.[3] Myocardial ischemia and infarction are an important complication of noncardiac surgery in older adults, and this complication can be fatal, but symptoms of myocardial ischemia are not often present to prompt the diagnosis.

Clues missed in this case include ST-segment and T-wave changes on the cardiac monitoring for arrhythmias. If one looks at the cardiac monitor only for arrhythmias, changes of myocardial ischemia or infarction can be overlooked.

"The eye sees only what the mind is prepared to comprehend," as the French philosopher Henri Bergson has said. Of course, ischemia must involve a substantial amount of heart muscle to cause changes visible on electrocardiography, and smaller areas of myocardial ischemia could cause a fatal arrhythmia. Another clue that was not so much missed as dismissed by the cardiologist who "cleared" the patient for chest surgery was the cardiac scan interpreted as positive by the radiologist and then reinterpreted as negative by the cardiologist. The patient had many major risk factors for atherosclerotic coronary artery disease, including his history of hyperlipidemia, hypertension, severe obesity and smoking, and his family history of the disease.

Patients with heart rates of 96/minute or higher prior to the induction of anesthesia were significantly more likely to have myocardial injury and postoperative mortality in the Indian study.[3] Another parameter independent of electrocardiographic changes and symptoms of ischemia recently shown to predict perioperative myocardial injury is a widened pulse pressure. The results of one study suggested that a pulse pressure over 62 mm Hg could be a useful clinical sign of risk that could guide strategies to decrease the risk.[4]

Prevention has been the focus of recent studies. Inhibiting platelets was prospectively studied for the purpose of preventing myocardial injury during noncardiac surgery and the administration of aspirin before surgery and in the early postsurgical period was found to have no significant effect on the rate of a composite of death or nonfatal myocardial infarction, but to be associated with an increased the risk of major bleeding.[5] Studies of other preventative measures have had promising preliminary results, but large scale trial results are pending.[6,7] In the meantime, this report can help raise awareness of intraoperative myocardial injury, illustrate the importance of the diagnosis and suggest clues that can be used to make the diagnosis.

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