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Coronary Angioplasty Following Acute Perioperative Myocardial Infarction

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In the early hours following an acute myocardial infarction (AMI), coronary angioplasty and/or thrombolytic therapy may be indicated in an attempt to restore blood flow to ischemic myocardium.[1] Such therapy is increasingly available to hospitalized patients. Unfortunately patients who suffer an intraoperative AMI are often deprived of this form of treatment due to difficulties in detection of the infarction and problems of interdisciplinary communication. We describe a case in which intraoperative electro- and echocardiographic changes suggested AMI during noncardiac surgery, and balloon angioplasty was carried out immediately postoperatively with a successful outcome. Our purpose in presenting this case is to alert anesthesiologists to the logistics involved in carrying out angioplasty postinfarction and to summarize the importance and benefits of invasive therapy for an acute intraoperative myocardial infarction.

CASE REPORT

A 47-yr-old man with diabetes mellitus, hypertension, and a long history of smoking underwent wide excision of the left anterior knee structure and gastrocnemius flap reconstruction for a poorly differentiated sarcoma of the patella. Medications included propranolol 40 mg twice a day, chlorpropramide 500 mg daily, and enalapril 5 mg daily. He had occasional chest pain at rest. He weighed 92 kg, his lungs were clear, and no heart murmurs or S3 were audible. Arterial blood pressures of 170/90–115 were recorded preoperatively. The ECG showed inverted T waves in lead III and left ventricular hypertrophy by voltage criteria.

The morning of surgery the patient received 10 mg diazepam, 40 mg propranolol, and 5 mg enalapril orally. Preoperative arterial blood pressure was 180/105 mmHg with a heart rate of 70 bpm. The an-

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esthetic technique was continuous lumbar epidural analgesia (T8 level using 15 ml 3% chloroprocaine) combined with general anesthesia (0.25–0.5% isoflurane and 50% nitrous oxide-50% oxygen and pancuronium). One hour after induction of anesthesia, the arterial blood pressure decreased to 100/50 mmHg, heart rate was stable at 90 bpm, and nearly 3 mm ST segment elevation was noted in leads II, III, AVF, and V_5, though a formal 12-lead ECG was not performed. Oxygen 100%, nitroglycerin 200 μg iv bolus, and neosynephrine 200 μg (100 μg iv bolus repeated twice) were administered, resulting in restoration of normal ST segments. The arterial blood pressure was stable at 120–140/75–85 mmHg after this event and nitroglycerin was continued. A two-dimensional transesophageal echocardiograph probe (2-D TEE) was then inserted, and short-axis cross-sectional views at the level of the papillary muscles showed a diffusely hypokinetic left ventricle without regional wall motion abnormalities. Due to the transient, reversible nature of the ECG abnormalities, we decided, in consultation with the surgical team, that surgery should continue. Three hours later, with the arterial blood pressure 100–110/70 mmHg, 5 mm ST segment elevation was noted, localized to leads II, III, and AVF. Simultaneously, severe inferior wall dyskinesis was evident on 2-D TEE. These changes persisted despite the cessation of surgical stimulation. Administering 10 mg verapamil, 50 μg neosynephrine, increasing doses of nitroglycerin, 40 mg esmolol, and 100% oxygen failed to reverse the changes despite an increase in blood pressure to 140/80 mmHg and a heart rate of 60 bpm. Frequent ventricular premature contractions were noted, and a lidocaine infusion was begun. A pulmonary artery catheter was inserted; the cardiac output was 3.5 l/min, pulmonary artery pressure 23/17 mmHg, pulmonary wedge pressure 14 mmHg. The dyskinetic inferior wall motion and absence of wall thickening in that region on 2-D TEE persisted. The operation was rapidly completed, and the patient taken to the recovery room, where his arterial blood pressure was 110/72 mmHg and heart rate was 63 bpm. Ventilation was controlled and sedation induced with iv morphine and diazepam. A 12-lead ECG revealed 5-mm ST segment elevation in leads II, III, and AVF, ST segment depression in leads V_2 and V_3, and Q waves in leads III and AVF, all different from his preoperative electrocardiogram. Within 30 min, the patient was transported to the Cardiac Catheterization Laboratory for emergency angioplasty (PTCA). Angiography revealed a normal left main coronary artery, greater than 70% stenosis of the proximal and 60% stenosis of the distal left anterior descending artery, and 50% distal stenosis of the circumflex artery. The right coronary artery was dominant and totally occluded in its mid-portion. PTCA of the right coronary artery was attempted following the administration of 10,000 units of heparin. Three successive inflations with a balloon dilatation catheter at 3–5 atmospheres lasting 42–120 s resulted in a reduction of the ST segment elevation from 5 to 3 mm. Repeat angiography showed a residual 20% stenosis at the previous site of total occlusion in the right coronary artery. The patient was transferred to the Coronary Care Unit and 8 h later, serum creatine phosphokinase (CPK) was 2050 iU/l with a 5% MB fraction. Over the next 24 h, mechanical ventilation was discontinued, and the ST segment elevation resolved. The second postoperative day, repeat CPK was 356 and precordial echocardiography showed an overall mild decrease in left ventricular function with inferior wall hypokinesis. He recovered fully from his perioperative complications and was discharged on the 21st postoperative day. A wound debridement performed 1 month later requiring general anesthesia was uneventful.

Discussion

The rationale behind invasive therapy of an acute myocardial infarction is related to the extremely high incidence of occlusion of the diseased coronary artery. DeWood *et al.* demonstrated that the early period (first 4 h) after transmural myocardial infarction is characterized by a complete absence of blood flow in the diseased coronary artery in 87% of patients.[2] Thus, the angiographic demonstration of occlusion of a coronary artery supplying an infarcted area has led to attempts to reestablish blood flow with the goal of improving patient outcome.[2] These attempts to increase flow have been accompanied by reduction in chest pain, return to normal ST segments, an early peak of CPK, limitation of infarct size, and improved ventricular function.[3–6] Methods utilized for reestablishing perfusion include perforating the occlusion with a guide wire,[1] infusing thrombolytic agents,[3–6] performing balloon angioplasty,[7] or emergency coronary artery bypass surgery.[8–9] These options are all potentially available for invasive treatment in the surgical patient who has suffered an intraoperative AMI, and in addition, temporary measures such as intra-aortic balloon counterpulsation may also be considered. Emergency coronary artery bypass surgery is associated with a low mortality rate, salvage of myocardial function, and improvement of long-term survival when performed within 4–6 h of infarction,[8–9] but requires availability of still another surgical team, as well as perfusionists. Streptokinase therapy has been reported to dissolve thrombi and restore coronary blood flow in 70–90% of acutely obstructed arteries.[10] Unfortunately, this therapy is inappropriate in an immediately postoperative patient due to the possibility of life-threatening hemorrhage. In the postoperative surgical patient who has sustained an infarction, angioplasty seems to offer several advantages. Balloon angioplasty has been demonstrated to be a highly effective therapy for acute myocardial infarction in patients treated within 3–5 h postinfarction. In addition to avoiding bleeding complications which may occur with thrombolytic therapy, the success rate for recanalization of the infarct vessel is nearly 85% using angioplasty.[11] Residual stenosis of 70% or more was present in only 4% of patients. Furthermore, there were significant increases in left ventricular ejection fraction and improvement in regional wall motion abnormalities.[11] There is evidence of improved coronary blood flow and a reduction in recurrent ischemic events.[7,12] Disadvantages include a high frequency of arrhythmias and a 12–15% incidence of re-occlusion upon followup angiography.[13] Abrupt closure and re-occlusion are more common following angioplasty in patients who have sustained an MI as opposed to those undergoing the procedure on an elective basis.[13]

The approach we used in treating an acute myocardial infarction, *i.e.*, direct coronary angioplasty, would probably only be possible in a minority of hospitals in this country, since only 12% have facilities for coronary angioplasty.[14] Furthermore, a 24-h call team, experienced personnel, and a commitment to using angioplasty immediately result in there be being few centers that could

perform direct coronary angioplasty after myocardial infarction. Therefore, even if such therapy were contemplated following an intraoperative MI, it is doubtful that at present many patients would have immediate angioplasty as an available therapeutic option. It should be noted that at present there are no randomized studies comparing immediate angioplasty alone to other therapies for acute myocardial infarction.

Did our patient, with an inferior wall MI, actually benefit from angioplasty? There is controversy regarding the benefits of angioplasty in patients who have sustained an inferior wall MI. Most patients with inferior wall infarctions have preserved ventricular function and their mortality is lower than in comparable patients with anterior wall infarctions. One could argue, therefore, that the risk of angioplasty in this situation exceeds the risk of mortality from the infarction. Furthermore, compared with the left anterior descending and circumflex arteries, the right coronary artery is more likely to reocclude after angioplasty.[15] However, there is justification for using angioplasty in patients similar to ours, as immediate reperfusion therapy results in an equivalent improvement in regional wall motion abnormalities in patients with left or right coronary artery occlusions, and the risk of death appears to be the same in the two groups as well.[16] We do not know if our patient's outcome was altered as a result of PTCA. There was an improvement in ST segment elevation and in inferior wall motion and therefore we can state that he did derive some benefit from the procedure.

The anesthesiologist's responsibility during angioplasty was similar to that involved in the intraoperative care of an ill cardiac surgical patient. Ventilation of the lungs was controlled and the electrocardiogram, arterial blood pressure, and pulmonary artery pressures were monitored. Arrhythmias are common during angioplasty, and therefore careful attention was paid to the electrocardiogram and lidocaine was given by continuous infusion. We administered morphine and diazepam to sedate the patient and to thereby decrease myocardial oxygen requirements.

In summary, this case demonstrates that coronary angioplasty can be successful if performed in the early stages following an acute intraoperative myocardial infarction. The use of intraoperative 2-D TEE to verify ECG changes and magnify their significance was important in alerting surgeons and cardiologists to the urgency and importance of the ECG changes in this patient. Where facilities and personnel are readily available to perform cardiac catheterization and angioplasty, and where good communication and coordination is available between the anesthesia/surgery and cardiology teams, this appears to be an appropriate choice of therapy in a similar situation. Anesthesiologists can anticipate an increasing degree of involvement in the care of patients undergoing coronary angioplasty during an acute myocardial infarction.[17]

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General Anesthesia and Hyperkalemic Periodic Paralysis

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The periodic paralyses are a group of disorders characterized by recurrent attacks of weakness, associated with changes in serum potassium concentration. Three distinct groups of periodic paralysis have been described: hyperkalemic, hypokalemic, and normokalemic. Only scanty anesthetic experience has been reported with the hyperkalemic group. In 1959, Egan and Klein described three patients with hyperkalemic periodic paralysis who underwent anesthesia.[1] In each case, upon awakening from general anesthesia, the patient was unable to move for several hours. The anesthetic given in two cases for dental procedures was thiopental. The anesthetic administered in the third case for childbirth was unspecified. One patient delivered a child later under spinal anesthesia without developing paralysis. In 1980, Flewellen and Bodensteiner described a general anesthetic in a 10-yr-old girl for correction of esotropia and gastrocnemius muscle biopsy.§ General anesthesia was induced with inhalation of halothane with 50% nitrous oxide in oxygen. The patient's trachea was intubated and controlled hyperventilation was instituted without the use of muscle relaxants. During the course of the anesthetic, sinus rhythm was interrupted by occasional unifocal extrasystolic beats. Halothane was discontinued and enflurane was begun with return of sinus rhythm. The patient awoke with return of spontaneous respiration and her trachea was extubated without difficulty. We present a description of the first successful management of an adult male patient with hyperkalemic periodic paralysis who received general anesthesia along with muscle relaxants and did not develop muscle weakness on awakening or during recovery.

CASE REPORT

The patient was a 40-yr-old, 85-kg man who began having muscle weakness in his legs at age 9 yr. The weakness occurred shortly after awakening and was more profound following rest after exercise. No myotonia was present and the neurological exam was normal. Hyperkalemic periodic paralysis was diagnosed at age 20 yr by oral administration of KCl which provoked his usual attack. He was receiving hydrochlorothiazide 25 mg/day for 2 yr, and had been free of symptoms. In 1988, he developed numbness and weakness in his left arm and left thumb. After studies, a herniated nucleus pulposus was revealed at C5–6 with spurring of C6–7 and surgery was scheduled for anterior cervical discectomy and fusion.

Admission laboratory values were within normal limits, including a serum potassium of 3.6 meq/l and an EKG that showed normal sinus rhythm, without T-wave abnormalities. The patient received hydrochlorothiazide 25 mg orally 1 h prior to arrival to the operating room. In the operating room, an iv infusion was started and a radial artery was cannulated. EKG, blood pressure cuff, and pulse oximeter were applied. Peripheral nerve stimulator electrodes were applied over the ulnar nerve. Anesthesia was induced with thiopental 250 mg iv. Six mg of vecuronium iv resulted in rapid onset of paralysis and tracheal intubation was accomplished without difficulty. The maintenance anesthetic was nitrous oxide (3 l/min), oxygen (2 l/min), isoflurane (.6–.8%), and fentanyl 750 μg in divided doses over 4 h. Evidence of return of neuromuscular function by train-of-four monitoring was apparent within the first hour of surgery and no additional muscle relaxant was given after the initial dose. Fluids not containing potassium were used throughout the perioperative period (5% dextrose with 0.9% normal saline 500 ml, normal saline 500 ml, 5% dextrose with 0.45% normal saline 1000 ml). Normothermia was maintained using a heated humidifier and fluid warmer. Serial blood gases, serum sodium, potassium, and glucose concentrations were monitored throughout the course. The intraoperative serum potassium concentrations, measured hourly, were 3.6, 4.1, 3.8, and 3.5 meq/l. The surgical procedure was completed in 4 h. The patient was awakened after demonstration of adequate return of neuromuscular function including full train-of-four

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