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Acute Myocardial Ischemia during Thoracotomy in a Patient with Previous Coronary Artery Bypass Grafting

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PREVIOUS coronary artery bypass grafting (CABG) may be protective for patients undergoing subsequent noncardiac surgery.[1–3] Patients with previous CABG may, however, be susceptible to acute intraoperative ischemic events when surgical manipulation occurs in the vicinity of functioning bypass grafts.

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Case Report

A 66-yr-old man was to undergo resection of a 1.6-cm non–small-cell carcinoma of the right middle lobe. Medical history was significant for CABG 7 yr and bilateral carotid endarterectomies 6 yr before admission. The patient had done well after CABG, although he had been evaluated for palpitations 1 month earlier with ambulatory electrocardiographic (ECG) monitoring. This 24-h recording showed two premature ventricular contractions, 14 supraventricular beats, and no episodes of significant ST-segment depression. He complained of no other symptoms referable to the cardiovascular system. Physical examination revealed only a soft systolic ejection murmur. Medications included verapamil, pravastatin, and aspirin. Results of the preoperative laboratory evaluation were normal. The patient was instructed to take his usual dose of verapamil the morning of surgery. The preoperative ECG (fig. 1) showed a normal sinus rhythm, left atrial abnormality, and nonspecific T-wave changes, and was unchanged from previous ECGs.

After the patient's arrival in the operating room, intravenous access was established, a radial arterial catheter inserted, and 0.5 mg intrathecal morphine administered *via* lumbar puncture at L3–L4. ECG monitoring was performed with a critical care monitor (Series 7010, Marquette Electronics, Milwaukee, WI) with leads placed on the shoulders and lower thorax bilaterally and in the V_5 precordial position. Anesthesia was induced with intravenous fentanyl 250 μg and

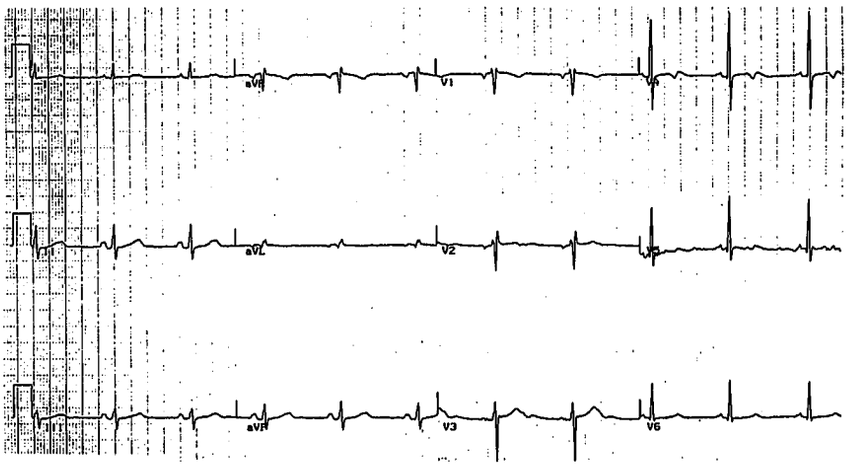

Fig. 1. Preoperative electrocardiogram shows a normal sinus rhythm, left atrial abnormality, and nonspecific T-wave changes.

thiopental 250 mg. Muscle relaxation was achieved with pancuronium, and the trachea was intubated with a 39-French left-sided double-lumen tracheal tube. Correct positioning of the tracheal tube was confirmed by fiberoptic bronchoscopy. Anesthesia was maintained with nitrous oxide in oxygen and isoflurane 0.6–1.0% end-tidal. The patient was positioned in the left lateral decubitus position.

Surgical incision and dissection into the right hemithorax were well tolerated by the patient, with no significant change in heart rate or blood pressure. One-lung ventilation with an inspired oxygen fraction of 1.0 was maintained, with an oxyhemoglobin saturation of 100%. When lateral traction was applied to the middle lobe to mobilize its medial adhesions, the blood pressure suddenly decreased from 130/70 to 80/40 mmHg, concomitant with a 3–4-mm increase in the ST segments in the inferior (II, III, and aVF) ECG leads. Phenylephrine 100 μg was given immediately, and blood pressure returned to 125/70 mmHg, but the ST segments remained elevated. Traction on the middle lobe was released within seconds of the fall in blood pressure. Mechanical manipulation of the right saphenous coronary bypass vein graft was suspected immediately and was confirmed by dissecting out the right coronary vein graft and demonstrating adhesion of this graft to the medial aspect of the right middle lobe. A nitroglycerin infusion was started at a rate of 0.5–1.4 $\mu g \cdot kg^{-1} \cdot min^{-1}$. A segmental resection of the middle lobe mass was performed, and the remainder of the surgical procedure was completed without difficulty. The patient remained hemodynamically stable for the duration of surgery, requiring no vasopressors for support of blood pressure.

Although the inferior ST-segment elevation decreased progressively for the duration of the case, significant changes were still present on admission to the surgical intensive care unit (fig. 2), having the appearance of an acute inferior myocardial infarction. Intravenous nitroglycerin was continued until the morning of the 1st postoperative day, and the patient was separated from mechanical ventilation the evening of surgery. In samples drawn at 7:00 and 11:00 PM the evening of surgery and at 3:00 and 9:00 AM and 12:00 PM on the 1st postoperative day, serial creatine kinase myocardial band isoenzymes never exceeded 3% of the total creatine kinase. By 11:00 PM the evening of surgery the ST segments had returned to baseline (fig. 3). The remainder of the patients hospital course was uneventful, and the patient was discharged on the 6th postoperative day.

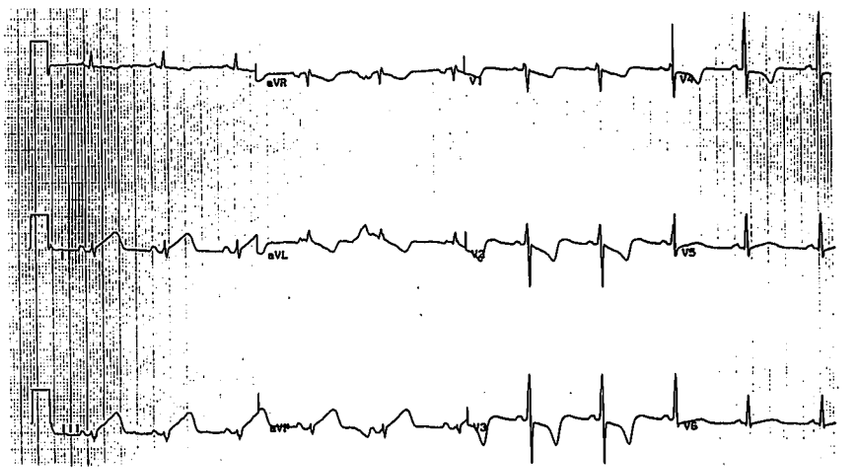

Fig. 2. Electrocardiogram on admission to the surgical intensive care unit, showing inferior ST-segment elevation and anterior ST- and T-wave abnormality.

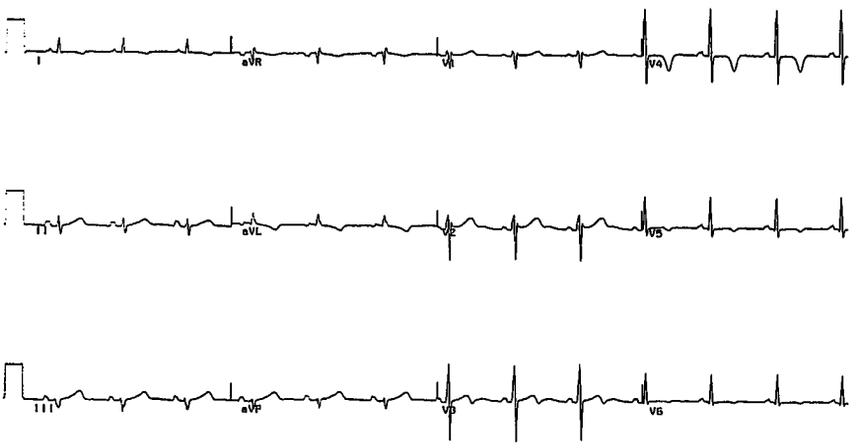

Fig. 3. Electrocardiogram 4 h after admission to the intensive care unit, showing resolution of inferior ST-segment elevation.

Discussion

Most anesthesiologists are relieved to know that a patient has undergone previous CABG, although there is a well-defined incidence of venous bypass graft atherosclerosis and occlusion with time.[4] Whether patients with a relatively remote history of CABG with venous grafts are at an increased risk of perioperative ischemia for noncardiac surgery is unknown. Several studies have suggested a low incidence of complications in patients with previous CABG undergoing subsequent noncardiac procedures, although these studies can be criticized for small sample size and lack of control groups.[5,6] Data from the Coronary Artery Surgery Study registry, which evaluated patients with coronary artery disease, bypassed coronary artery disease, or normal coronary arteries also suggests that previous CABG has a protective effect when patients undergo noncardiac surgery.[2] Nielsen *et al.* recently compared 181 patients who had undergone previous CABG to a cohort of age-, gender-, and procedure-matched patients who had not undergone CABG who were having elective major nonthoracic general surgical or vascular procedures. Although the degree of coronary artery disease was unknown in the CABG and cohort groups, no differences were found in the overall incidence of cardiac complications (2.8% and 3.0%, respectively), leading the authors to suggest that patients with previous CABG are not at greater risk than the general population for subsequent noncardiac surgery.[7]

Although our patient had been evaluated for palpitations preoperatively, he was quite active and had no symptoms suggestive of myocardial ischemia. Ambulatory ECG monitoring in the month before surgery showed no evidence of silent myocardial ischemia.

Acute intraoperative myocardial ischemia is usually thought of in terms of an imbalance in the relation between myocardial oxygen supply and demand. Although hemodynamic changes, particularly tachycardia, may provoke intraoperative ischemia in the patient at risk, many if not most episodes of intraoperative ischemia occur in the absence of hemodynamic changes.[8] Fortunately, in our case the cause of the acute event was immediately recognized as kinking of a patent vein graft. Traction on the lung was immediately released. The involved vein graft was then identified and the lung adhesions released under direct vision, leaving the graft intact. Because the patient was hemodynamically stable, the decision was made to continue with segmental resection of the right middle lobe.

The long-term patency of internal mammary artery grafts is better than that of saphenous vein grafts,[9] and internal mammary artery grafts are therefore used whenever possible in CABG. A case has recently been described in which a patient with non–small-cell carcinoma was found at thoracotomy to have tumor invasion of both the left internal mammary artery graft as well as the pericardium and the proximal left anterior descending saphenous vein graft.[10] This patient had transient ST-segment depression during resection of the tumor, but no long-term sequelae ensued. In our patient, the tumor site was remote from the vein graft, which was attached by fibrous adhesions to the medial aspect of the right middle lobe. Our patient's hypotension and prolonged inferior ST-segment changes after manipulation of the graft suggest that the inferior

myocardium was critically dependent on flow through the graft. The reason for the persistent and slowly resolving ST changes after transient kinking of the graft is uncertain. Distal emboli or vasospasm are potential but unlikely explanations. The effect of the intravenous nitroglycerin on the time course of ischemia resolution is also speculative. However, no serial ECG or isoenzyme changes diagnostic of acute myocardial infarction developed.

In summary, we describe a patient with previous CABG who had severe myocardial ischemia during mobilization of the right middle lobe before excision of non–small-cell carcinoma. Mechanical manipulation of CABG grafts can occur with subsequent thoracic surgery, and the possibility of graft manipulation should be immediately considered in the differential diagnosis of intraoperative myocardial ischemia. Once the possibility is considered, manipulation should stop, and the graft should be identified and dissected free under direct vision. In this way a potentially devastating intraoperative complication may be avoided.

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An Anaphylactic Reaction to Topical Fibrin Glue

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Key words: Allergy, anaphylaxis: aprotinin; fibrin glue.

FATAL drug reactions occur in 0.01% of surgical inpatients. Important among serious adverse drug effects are allergic reactions because of their sudden onset and potential for catastrophic outcome.[1] Fibrin glue, a physiologic glue, has been used in a variety of clinical situations including bleeding after cardiac surgery and vascular anastomosis, organ injury, and neurosurgical procedures.[2–4]

Among the extensive clinical experience to date, there are some reports of adverse reaction to fibrin glue.[5] We describe a case of anaphylactic reaction immediately after topical application of fibrin glue (Ber-