Perioperative coronary artery spasm leading to myocardial ischaemia after vein graft surgery

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SUMMARY Coronary artery spasm has been recognised recently as a possible cause of perioperative myocardial ischaemia after coronary artery bypass grafting. We report on one case and review the published reports. We emphasise the clinical picture of the patient who is liable to have spasm and the necessity for a prompt diagnosis and correct treatment.

The incidence of perioperative myocardial infarction after coronary artery surgery has shown a progressive fall over the years.¹⁻³ Cold cardioplegia is the single most important reason for this, but it cannot deal with every possible mechanism of perioperative myocardial ischaemia. Coronary artery spasm may indeed constitute such a mechanism,⁴⁻⁶ and its occurrence in a case in our department prompted this report.

Case history

A 42 year old man complained of exertional angina with decreasing threshold over the past six months; on occasion, chest pain occurred at rest. Angiography showed isolated, severe, proximal stenosis of the left anterior descending coronary artery, which persisted unchanged after 0.3 mg glyceryl trinitrate sublingually.

The patient was discharged on oral nifedipine and isosorbide dinitrate, 60 mg each daily, but was readmitted five days later complaining of severe angina on minimal effort and at rest, and we therefore decided to operate. Electrocardiograms showed either a normal pattern or anteroseptal ST and T wave ischaemic changes.

At operation the left anterior descending artery was uneventfully grafted with an autologous saphenous vein under hypothermic cardiopulmonary bypass and cardioplegia. The electrocardiogram in the recovery room was normal.

Five hours after operation the patient complained of acute ischaemic chest pain, and simultaneous ST segment depression was recorded from almost all peripheral and precordial leads. The pain resolved,

but ST changes were still present after sublingual glyceryl trinitrate. Sublingual nifedipine and isosorbide, by hourly increments of 10 and 5 mg, respectively, as well as intravenous glyceryl trinitrate up to 1 mg/h, were not effective in preventing recurrence of chest pain, and the patient was therefore taken to the catheterisation laboratory three hours after the initial episode of angina. Selective angiography of the graft and the native coronary arteries was performed under continuous glyceryl trinitrate infusion at 1 mg/h. The right coronary artery (Fig. 1a) and all branches of the left coronary artery appeared diffusely narrowed. The vein graft was patent despite a very poor run-off (Fig. 1b). Glyceryl trinitrate, 1 mg, was then administered into each coronary artery and into the graft; subsequent injections of contrast medium demonstrated the return of each vessel to its previous size, while left anterior descending artery graft appeared satisfactory (Fig. 2).

Total CK and MB peaked at 576 and 38 units respectively, on the morning of the first postoperative day. The electrocardiogram returned to baseline without any further ST change, or pathological Q waves.

After catheterisation, nitrates were continued at a total daily dose of 306 mg (through combined sublingual, intravenous, and percutaneous routes) which was halved after one week. There were no further episodes of angina.

Six weeks after operation the patient was readmitted for elective stress testing, dynamic electrocardiography, and repeat angiography. All findings were normal, and the patency of the vein graft was confirmed.

(a)

Fig. 1 Postoperative selective coronary angiograms showing (a) diffuse right coronary artery narrowing; (b) patent vein graft to barely discernible left anterior descending artery.

(b)

(a)

Fig. 2 After intracoronary glyceryl trinitrate all vessels returned to normal size.

(b)

Discussion

The appearance of signs of perioperative myocardial infarction in the area of stenosed, grafted vessels has long been regarded as paradoxical.^{2,7,8} This upsetting and occasionally fatal complication has become rare in recent years,³ probably because of the wide adoption of cardioplegic techniques and the improvements in surgical skills.³

Perioperative myocardial infarction in areas distal to normal vessels may constitute an entirely different problem. Bolooki *et al.*⁹ reported one case of anterior wall infarction with patent left anterior descending artery after right coronary artery bypass grafting. Assad-Morell *et al.*¹⁰ reported three cases and Bulkley *et al.*⁷ reported one case, with a normal dominant right coronary artery, patent vein graft(s) to the left coronary system, and perioperative infarction of the

diaphragmatic wall of the heart. No interpretation was offered, and it remained for Pichard *et al.*¹¹ to suggest that coronary artery spasm might be responsible for postoperative ischaemic events. Buxton *et al.*⁴ recently provided angiographic proof of this hypothesis in one case, and reported five more cases with less direct, yet highly suggestive, evidence of the same phenomenon.

Although perioperative "functional" obstruction of the coronary arteries may occur in any vessel, it has been more frequently seen in patients with normal right coronary arteries, which have not been bypassed.^{4,5,12} Patency of the right coronary artery thus seems to be a factor in this condition, as does a history of angina at rest.^{4,6} The relations with variant angina are certainly intriguing, but have not been completely elucidated. Recent reports on this subject, however, do not substantiate the hypothesis of a significantly increased risk of perioperative spasm in patients with this syndrome.¹³⁻¹⁵

All six patients reported by Buxton *et al.*⁴ presented with sudden hypotension and arrhythmias within two hours of termination of cardiopulmonary bypass; two other reports describe a similar sequence of events^{6,12} which was also seen in three additional patients in our unit in whom no angiographic proof of the coronary spasm was obtained. This mode of presentation, when associated with significant ST segment elevation, may be regarded as typical of acute postoperative coronary spasm, and particularly so when the preoperative angiograms showed that the involved vessel was not diseased. The case presented here illustrates a wider spectrum of possibilities for perioperative coronary spasm; not only may a diseased, grafted vessel be involved,^{6,11} but also all coronary arteries may simultaneously undergo diffuse spasm.

The relevance of this peculiar complication is threefold. First, it may affect patients with single or double vessel disease undergoing what usually appear to be simpler surgical procedures at negligible risk. Second, it may evolve in an unfavourable way in the surgical setting, since it is resistant to conventional drug treatment. Third, its correct identification may lead to effective management: that is direct injection of drugs into the spastic coronary artery, either at operation or in the catheterisation laboratory, associated with administration of nitrates and/or calcium antagonists intravenously or sublingually.⁶

We conclude therefore that the possibility of coronary spasm should be promptly considered when analysing perioperative ischaemic events. An aggressive diagnostic and therapeutic approach is warranted in view of the poor prognosis.

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