

Coronary Artery Spasm and ST-Segment Depression

To the editor:—The recent report by Skarvan *et al.* describes 10 patients with coronary artery spasm after coronary artery bypass surgery.¹ All their patients had concomitant ST-segment elevations. We would like to stress that perioperative coronary spasm also may be associated with ST-segment depression, as demonstrated in the following case report.

A 53-year-old man with good left-ventricular function received two coronary artery bypass grafts. Anesthesia was induced and maintained with morphine (total dose 3 mg · kg⁻¹) and 50% N₂O. The prebypass period was uneventful except for one episode of hypertension (170/80 mmHg) during sternotomy, which was treated successfully with intravenous nitroglycerin (NTG). Myocardial protection during aortic cross-clamping was accomplished with cold hyperkalemic cardioplegia. Weaning from cardiopulmonary bypass was uneventful. But 30 min later, suddenly ST-segment depression (more than 2 mm) occurred in the V₅ lead, followed by a decrease in blood pressure from 120/60 to 100/50 mmHg. Heart rate remained unchanged at 85 beats · min⁻¹. Cardiac output decreased from 6 to 4 l · min⁻¹ without a change in pulmonary artery pressure (25/10 mmHg) or pulmonary capillary wedge pressure (12 mmHg). Inspection of the coronary grafts revealed no air or torsion. Phenylephrine infusion, in an attempt to increase myocardial perfusion pressure, did not change the ST-segment pattern or cardiac output. Intravenous NTG (0.2–0.4 mg · min⁻¹) also was unsuccessful. Because coronary spasm was suspected, intracoronary NTG (0.4 mg) was administered in the LAD graft. This immediately relieved the ECG signs of ischemia, and cardiac output returned to 6–7 l · min⁻¹. Fifteen minutes later, during closure of the chest, ST-segment depression again occurred suddenly, this time treated successfully with sublingual nifedipine (10 mg). NTG 0.15–0.2 mg · min⁻¹ infusion was maintained in the immediate postoperative period. The patient was discharged 6 days later, without recurrence of myocardial ischemia.

We would like to mention several other important aspects in the presentation and management of patients with coronary artery spasm. Severe classic coronary artery spasm will cause ST-segment elevation because of transmural ischemia and is associated with significant cardiac depression. However, a moderate degree of coronary spasm will depress the ST segment as a result of subendocardial ischemia.² Global myocardial function then will not be impaired to a major degree, and therefore a significant elevation of PCWP will not occur.³

Coronary artery spasm also may present itself as refractory dysrhythmias, AV-conduction block, and even sudden cardiac arrest.³ One should suspect coronary spasm as the cause when these symptoms are resistant to conventional treatment, such as inotropic drugs, vasopressors, and antidysrhythmic drugs. Intracoronary administration of nitroglycerin (0.1–0.4 mg) has been always successful in relieving coronary spasm,⁴ and it also can be considered a diagnostic tool. Intravenous use of NTG is not always effective, as this frequently fails to produce adequate plasma concentrations to relieve the spasm without causing severe systemic vasodilation.⁴ Calcium channel blocking agents, especially nifedipine and diltiazem, also are very effective.⁵ In the rare occasion when nifedipine and intravenous NTG are not effective, NTG should be injected directly into the spastic coronary artery or graft, either in the operating room or in the catheterization laboratory.⁶

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