Coronary Revascularization without Cardiopulmonary Bypass: Use of Ischemic Preconditioning and Adenosine

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ATHEROSClerotic disease of the ascending aorta significantly increases the morbidity and mortality of coronary artery bypass grafting (CABG) by increasing the risk of clamp injury, dissection, embolic coronary debris, and stroke. Therefore, in patients with severe calcific aortic disease, complete avoidance of aortic manipulation is recommended.1 This can be achieved with femoral artery cannulation for cardiopulmonary bypass, combined with the use of the internal mammary arteries for grafting.2 However, femoral artery cannulation may be associated with many complications such as tears, retrograde arterial dissection, stenosis, thrombosis, and an increased incidence of infection.3 We present a patient with severe generalized vascular disease who was treated without aortic or femoral artery cannulation using a combination of ischemic preconditioning4,5 to protect the myocardium and with bolus doses of adenosine to induce temporary cardiac standstill.

Case Report

A 70-year-old man was scheduled for an elective two-vessel CABG procedure. He had a non-Q-wave myocardial infarction 2 days previously. Cardiac catheterization showed 90% occlusion of the left main coronary artery, a 90% ostial occlusion of the circumflex, an ejection fraction of 45%, and mild global hypokinesis. He had bilateral above-the-knee amputations after an aortobifemoral bypass procedure 5 yr previously. His preoperative oral medications included metoprolol, 50 mg twice daily, isosorbide dinitrate 40 mg three times daily, amiodipine 5 mg daily, aspirin 325 mg daily, and lovastatin 20 mg daily.

General anesthesia was induced with intravenous fentanyl, 2 mg, and midazolam, 10 mg, in divided doses. Intravenous rocuronium, 100 mg, was used to facilitate tracheal intubation. Anesthesia was maintained with intermittent bolus doses of fentanyl (total of 5 mg), midazolam (total of 20 mg), and pancuronium, 8 mg. A variable-rate nitroglycerin infusion was administered throughout the surgery. Intraoperative monitoring included continuous electrocardiogram (leads II and V5), 2-channel ST segment monitoring, radial arterial blood pressure, pulse oximetry, temperature, end-tidal carbon dioxide, pulmonary arterial pressure, and cardiac output measurements.

After dissection of the left and right internal mammary arteries and administration of intravenous heparin, 15,000 U, the ascending aorta was determined to be a solidly calcified rigid tube, or 'porcelain aorta,' and cannulation would not be safe. Cannulation of the femoral artery was unfeasible because of significant peripheral vascular disease and an absence of femoral pulses; therefore, coronary revascularization was performed without cardiopulmonary bypass using ischemic preconditioning to protect the myocardium.

The first obtuse marginal branch of the circumflex coronary artery was encircled proximally and distally, occluded for 5 min for preconditioning. During the occlusion, there was no evidence of myocardial ischemia as determined by ST segment and T wave changes, and the patient was hemodynamic stable with no change in heart rate, blood pressure, or pulmonary pressures. The circumflex artery was then reperfused for 5 min and again occluded for bypass grafting. To assist the surgeons during the critical portions of the anastomosis, an attempt was made to decrease the heart rate using bolus doses of esmolol up to 1 mg/kg; however, the heart rate actually increased, from 100 to 110 beat/min, and administration of phenylephrine was required to maintain an adequate mean arterial pressure. The hypertension was thought to be a result of decreased ventricular filling caused by the manual retraction of the heart required to obtain visualization of the circumflex artery and the decreased contractility caused by esmolol. To obtain acceptable surgical operating conditions, we then gave intermittent bolus doses of adenosine during the critical portions of the operation to produce intermittent cardiac standstill. Initially, doses of 18–21 mg provided 30–60 s of asystole, but tachyphylaxis developed, and doses of 30 mg were required to maintain asystole for the same time period. Approximately 2-min recovery period between doses allowed good recovery of the mean arterial pressure. The occlusion time for the circumflex artery was 14 min.

The second anastomosis was also done after a 5-min occlusion period for preconditioning. As the anastomosis to the diagonal branch of the left anterior descending artery does not require manual retraction of the heart, we again administered esmolol 1 mg/kg; however, it was again ineffective in lowering the heart rate. Adequate surgical conditions were achieved with adenosine 30–40 mg with a 2-min recovery period between doses. The occlusion time for the diagonal

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branch was 16 min. The total doses of esmolol and adenosine administered were 400 mg and 300 mg, respectively. The core temperature was 36–37°C throughout the procedure. The surgery was completed without further complications, and the patient was transferred to the intensive care unit in stable condition. The patient was discharged from the intensive care unit on the first postoperative day and discharged home on the sixth postoperative day.

Discussion

The initial plan for coronary artery bypass grafting in this patient was to use cardiopulmonary bypass (after venous and arterial cannulation) combined with aortic cross-clamping and cardioplegic arrest. However, the presence of severe calcific aortic disease made aortic cannulation and cross-clamping unfeasible. As this patient had significant atherosclerosis of the femoral arteries, had had multiple surgical procedures involving his femoral arteries before his amputations, and did not have palpable femoral pulses, femoral cannulation was not an acceptable alternative. Because of the lack of suitable cannulation sites for cardiopulmonary bypass, the decision was made to perform the coronary revascularization without cardiopulmonary bypass. Further, to provide myocardial protection, ischemic preconditioning was used.1,5

Brief periods of ischemia have been shown to paradoxically protect or precondition the heart and to reduce the infarct size caused by a subsequent longer period of coronary artery occlusion.3 Ischemic preconditioning has been demonstrated in animal models, and recent studies suggest that it occurs in humans as well.1-9 The mechanism by which ischemic preconditioning occurs is not completely clear; however, endogenous adenosine is reported to play an critical role. In human and animal preparations, the myocardial protection achieved by ischemic preconditioning can be mimicked by adenosine and specific A1 selective agonists or blocked by the use of A1 selective antagonists.9 Adenosine receptor agonists have been shown to reduce the infarct size; however, there are no reports suggesting that intermittent cardiac standstill produced by administration of adenosine mimics ischemic preconditioning. Although brief occlusion of the coronary arteries does not induce irreversible damage to the myocardium, it can cause reversible damage (e.g., stunning).5

The periods of coronary artery occlusion have varied between 5 and 15 min in the different studies, and the optimal duration of occlusion and reperfusion necessary for preconditioning is not known. Yellon et al.5 used two 3-min periods of occlusion interspersed with 2 min of reperfusion. The preconditioning protocol in the present case (i.e., 5-min occlusion followed by 5 min of reperfusion before occlusion for surgical anastomosis) was determined as it was an middle of the range value in the previously published studies.

Our attempts to decrease the heart rate using esmolol were unsuccessful as it significantly lowered the mean arterial pressure without decreasing the heart rate. Similar to our observations, Lönn et al.10 reported that administration of esmolol, to make the heart flaccid and facilitate the operation, did not decrease the heart rate but did reduce the contractility of the heart. The lack of further attenuation of the heart rate may be related to the inability of esmolol to occupy sufficient additional β-adrenergic receptors to produce added β-blockade in patients on chronic β-blockers.11 On the other hand, adenosine was much more effective than esmolol in controlling the heart rate and providing a motionless field in this patient. The bolus doses of adenosine were well tolerated in this patient with the occasional need for phenylephrine to manage the vasodilatory effects of adenosine.

Adenosine is a naturally occurring purine nucleoside that is rapidly cleared from the circulation by adenosine deaminase, found in vascular endothelial cells, and erythrocytes; its plasma half-life is less than 10 s.12 It has been shown to have potent cardiovascular actions, including sinoatrial node depression, prolongation of the atrioventricular nodal conduction time, attenuation of stimulatory actions of catecholamines, coronary vasodilation, and pulmonary and systemic vasodilation.12,13 Although adenosine usually is used at doses of 3–6 mg for termination of supraventricular tachycardia, the dose required for asystole is larger.13 Adenosine is best administered according to an incremental protocol; the starting dose of 9 mg is increased incrementally until asystole is induced.13,14 Because of adenosine’s ultra-short half-life, the interval between doses can be as short as 1 min without risking a cumulative effect.13

Adenosine should be administered through a central vein if possible, and if a peripheral vein is used, a saline flush is recommended.13 Adenosine should be used with caution in patients with a history of asthma and in those with sinus node dysfunction (e.g., sick sinus syndrome).12,13 In addition, adenosine is not effective in patients receiving treatment with methylxanthines because these are competitive antagonists of adenosine.12 Dipyridamole results in potentiation of adenosine’s clinical effects because it blocks adenosine uptake and metabolism.15 Although the asystole from adenosine usually lasts only for up to 1 min, it would be prudent to have pacing capability available in an event of prolonged asystole after the administration of adenosine.

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Unintended Supraventricular Tachycardia Induced by Extracorporeal Shock Wave Lithotripsy

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DESPITE the development of different modes and newer models of lithotriptors, cardiac arrhythmias continue to be reported as a complication of extracorporeal shock wave lithotripsy (ESWL).1–6 The utilization of an electrocardiographic (ECG)-triggered mode, when compared with respiratory-triggered or nontriggered ESWL, significantly reduces the incidence of arrhythmias.1–2 ECG-triggered lithotriptors eliminate ESWL-induced ventricular tachyarrhythmias as the delivered mechanical shock is synchronized to the terminal portion of the QRS complex during the absolute refractory period of ventricles. However, because of a potential for atrial stimulation, supraventricular tachyarrhythmias may still occur.3 After the performance of more than 3,600 cases of ESWL at our institution, we present a case of a reproducible supraventricular tachycardia caused by the Dornier HM3 Lithotripter (Germaring, Germany).

Case Report

A 71-yr-old man, recently diagnosed with a left renal calculus, planned to undergo cystoscopy and ureteral stent placement followed by ESWL. Other than a history of hepatitis, the medical history was unremarkable. He exercised regularly and denied any history of palpitations, angina, syncope, or dyspnea with exertion. On presenta-

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