ANZASM Case of the Month
January 2024 Edition

(case selected by the ANZASM Committee for your information)

Failure to perform coronary artery bypass graft with redo aortic and mitral valve replacement

Cardiothoracic Surgery

Case summary

An 82-year-old man was admitted to the local hospital with non-ST elevation myocardial infarction. He had developed increasing shortness of breath and chest pain at rest (despite use of glyceryl trinitrate) over the preceding months. He had also had a recent syncopal episode. Approximately 13 years prior he had undergone aortic valve replacement (bioprosthetic) and a coronary artery bypass graft (CABG) to the second diagonal branch of the left anterior descending artery. Additional comorbidities included prostate cancer (treated with radiotherapy 15 years prior), hypertension, paroxysmal atrial fibrillation, amaurosis fugax (16 years prior) and iron deficiency anaemia (likely due to anal canal telangiectasia secondary to radiation of the prostate). The patient was transferred for cardiological evaluation (despite no evidence of a convincing cardiovascular physical examination being found in the medical records).

An echocardiogram demonstrated normal left ventricular systolic function and grade 3 diastolic function. Right ventricular (RV) systolic function was unclear. There was severe prosthetic valve degeneration with a peak velocity of 4.3 m/s and a peak gradient of 75 mm Hg. The mitral valve posterior annulus was heavily calcified with thickened mitral valve leaflets and multiple jets of severe mitral regurgitation. There was also severe tricuspid valve regurgitation with an echo-derived right ventricular systolic pressure of 75 mm Hg. A coronary angiogram indicated 40% stenosis of the left main coronary artery and diffuse luminal irregularities of the left circumflex artery. Stenosis of the right coronary artery (RCA) of 50–60% was also apparent, with a patent graft to D2. A carotid ultrasound showed extensive atherosclerotic plaque at the carotid bifurcations bilaterally without significant stenosis.

Redo aortic and mitral valve replacements (bioprosthetic) without CABG were undertaken. The patient had severe RV systolic impairment after separating from cardiopulmonary bypass (CBP). This was resumed to decompress the right ventricle, and the heart was rested on CPB. Bypass was then discontinued with the patient on high-dose inotropes. The total cross-clamp time was 191 minutes; total CPB time was 296 minutes. No details were provided in the operative notes or the anaesthetic notes of the myocardial protection strategy; however, the perfusion record indicates that cardioplegia was administered anterogradely into the coronary ostia (calculated as every 40 minutes). Transfusion requirements were substantial.

The postoperative course was complicated by low cardiac output, elevated lactate, oliguria and escalating inotropic support. A transoesophageal echocardiogram (TOE) showed minimal RV function and the medical record reported severe aortic regurgitation. Inferior ischaemic changes on the electrocardiogram prompted an urgent coronary angiogram. A note suggested that the coronary lesions may have been more severe than estimated on the preoperative coronary angiogram. It was decided that percutaneous coronary intervention was inadvisable.

The patient was accepted for palliative care (i.e. not for resuscitation). He had progressive low cardiac output and multiple organ failure. A subsequent TOE did not confirm aortic regurgitation. Comfort care was instituted, and the patient died.

Discussion

The surgeon's desire to limit the extent of a redo valvular procedure in an elderly patient, by opting not to perform a CABG (particularly since the coronary angiogram could be interpreted as not demonstrating flow-limiting coronary lesions) can be well understood. However, in retrospect, this decision may have been a driver—likely the major driver—of the severe RV dysfunction observed in the operating room and postoperatively. As a rule, when dealing with valvular procedures where there is coronary artery disease of uncertain severity, it is prudent to graft the vessels. Failure to graft coronary arteries that have obstructive disease, is far more likely to have consequences than is unnecessarily grafting vessels with non-obstructive disease. Furthermore, the fact that chest pain (including pain at rest) was such a major component of this patient's symptomatology, should have been a reason to decide in favour of CABG. When the right ventricle was clearly in trouble in the operating room, this would have been the time to re-evaluate the original decision not to graft the coronary arteries. It is possible that when the patient went back on CPB to rest the heart, placement of a vein graft to the RCA with the heart beating may have resulted in improved RV function.

It is worth noting that the delivery of cardioplegia every 40 minutes seems to be a much longer interval than one would ordinarily accept. This raises the possibility that the myocardial protection strategy may have contributed, in part, to the myocardial dysfunction.

The principal management strategy for postoperative low cardiac output is mechanical circulatory support of some form, usually venous-arterial extracorporeal membrane oxygenation (VA–ECMO), which is superior to high-dose inotropes for myocardial recovery. However, VA–ECMO in an 82-year-old patient was probably a 'bridge too far'. Unfortunately, a patient—particularly an elderly patient—leaving the operating room in low cardiac output, with likely ungrafted obstructive coronary artery disease and on high-dose inotropic drugs for myocardial support, had little chance of survival.

Clinical lessons

This case demonstrates erroneous intraoperative decision-making by the surgeon electing not to perform CABG when confronted with poor cardiac function after aortic and mitral valve replacements.