A perilous course following myocardial infarction: ischaemic ventricular septal defect in a transplanted heart

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Abstract

Coronary artery disease in the donor heart is an established cause of early graft failure. However, identification of this before implantation is difficult. Cardiogenic shock associated with significant myocardial infarction during the early postoperative period is rare. Here, we report a case of a 42-year-old man who presented acutely with cardiogenic shock; he was supported by short-term extracorporeal support as a bridge to transplantation. Following successful orthotopic heart transplantation, he sustained coronary artery atheromatous plaque rupture, resulting in acute coronary artery occlusion, and subsequently developed an ischaemic ventricular septal defect on the third postoperative day.

Keywords: Ventricular septal defect • Heart transplant • Atheromatous plaque • Myocardial infarction

INTRODUCTION

Cardiac transplantation is an established procedure that can be achieved with relatively low morbidity and mortality. Coronary artery disease (CAD) is an established cause of late graft failure. Atheromatous CAD in the donor heart is difficult to evaluate at the time of organ retrieval and, as more marginal hearts are utilized to maintain heart transplantation volumes, the risk of transplanting significant unrecognized CAD increases. Centres in the UK do not routinely investigate potential donors by coronary angiography. Severe cardiogenic shock post-transplantation because of CAD is rare. Here, we report the case of ischaemic ventricular septal defect (VSD) secondary to plaque rupture and myocardial infarction within days of cardiac transplantation.

CASE PRESENTATION

A 42-year-old man was admitted to the emergency department following a large ST segment elevation myocardial infarction (STEMI) and, despite primary coronary intervention, he progressed towards heart failure necessitating veno arterial extra corporeal membrane oxygenation (VA-ECMO) support. VA-ECMO was discontinued 4 days later; however, he required a Levitronix extracorporeal left ventricular assist device (LVAD) to support his circulation. A gradual improvement in haemodynamic status made him eligible for transplantation, and he underwent an orthotopic heart transplant 49 days post-STEMI (the total warm and cold ischaemic time was 120 min). The donor was a 58-year-old woman who had had a subarachnoid haemorrhage following a fall, with occipital, C1 and C2 fractures. She had no known risk factors for heart disease and no history of cardiac or respiratory arrest preceding explantation. Twelve-lead electrocardiogram (ECG) on the donor was unremarkable and a transthoracic echo-cardiogram showed an ejection fraction of 50% with no regional wall motion abnormalities, valvular pathologies or septal defects. Postexplantation, there was no external evidence of any cardiac pathology and no palpable CAD.

Following transplantation, despite requiring an intra-aortic balloon pump (IABP) to improve haemodynamic performance during the early postoperative period, by Day 1 the patient was extubated and inotropes weaned and, by Day 2, the IABP was removed and inotropic support was discontinued, following which he remained haemodynamically stable for the next 24 h on low-dose isoprenaline (CIs of 2.5–3.0 l/min/m²). Sixty-nine hours post-donor heart reperfusion, a sudden haemodynamic collapse occurred with sustained severe hypotension (CIs consistently <1) despite significant escalation of inotropes and subsequent worsening metabolic acidosis. This required central VA-ECMO support. An intraoperative transoesophageal echocardiography (TOE) alluded to a possible ventricular septal defect (VSD) (Fig. 1). Repeat TOE demonstrated an unequivocal anterior VSD. Day 4 post-VA-ECMO he was retransplanted; however, this heart failed to wean from cardiopulmonary bypass despite maximum inotropic support, and repeat ECMO was thought to be futile. The histopathology of the first donor heart showed a thrombus in the left anterior descending artery (LAD) 3 cm from the origin, extending over a length of 1 cm, in conjunction with an antero-septal infarct (mid-septal level and extending to the apex) and rupture of the ventricular septum at the mid-septal level (Fig. 1). Microscopically, there was mild patchy atherosclerosis in the coronary arteries. In
the LAD proximal to the level of the thrombus, there was a lesion suggestive of an unstable plaque. In the region of the thrombus, there was disruption of the underlying atheromatous plaque. These findings confirmed an acute myocardial infarction (MI) resulting from plaque rupture of an unstable plaque in the LAD with resultant thrombotic occlusion. Examination of the specimen confirmed that, macroscopically, the atherosclerosis was not apparent and, therefore, could not have been detected in the donor at retrieval.

**COMMENT**

To our knowledge, this is the first reported case of a VSD post MI on a transplanted heart. The patient rapidly descended into cardiogenic shock 2 days post-transplantation. There are a limited number of cases in the literature reporting cardiogenic shock resulting from plaque rupture of an unstable plaque in the LAD with resultant thrombotic occlusion. Examination of the specimen confirmed that, macroscopically, the atherosclerosis was not apparent and, therefore, could not have been detected in the donor at retrieval.

VSDs can complicate a severe MI with a 30-day mortality of 74% [4]. However, postinfarct VSDs are uncommon, with an incidence of 0.2% post-thrombolysis [4]. VSDs typically occur within the first week of infarction, with a mean time from symptom onset of 3–5 days [5]. In this patient, clinical deterioration was remarkably
rapid: catastrophic haemodynamic collapse with rapid onset of cardio-genic shock on Day 3 post-transplantation, within 24 h of weaning IABP and inotropic support. Therefore, the VSD must have developed within 3 days of the infarct. Clinical suggestion of an infarct, let alone a VSD, was not apparent postoperatively; therefore, an indication to perform an angiogram with a view to thrombolysis, to reduce the risk of a mechanical complication, was not evident.

Although this was an anterior infarction complicated by a VSD, and thereby should have a better prognosis than inferior infarcts and VSDs, this case was further complicated by the infarct occurring on a transplanted heart, recognized to be particularly dependent upon RV performance. Therefore, the clinical course was acute in presentation associated with extensive septal infarction and severe RV dysfunction. The transplanted heart, having already been exposed to the stressors of ischaemia reperfusion and extensive infarction, was not suitable for a VSD repair. If this was attempted, biventricular support would have been inevitable. Furthermore, recovery from the infarct was unlikely because of the extensive infarct area and, therefore, long-term LVAD support was not suitable. This necessitated post-transplant ECMO support to maintain an adequate circulation and urgent retransplantation as a definitive option.

The demand to transplant a marginal heart in an era of donor scarcity risks facing morbid sequelae associated with undetectable CAD. This risk is greater in countries where angiographic assessment of the donor heart is not conducted. In these centres, clinicians should be aware of such potential complications and be prepared to utilize the necessary adjuncts to salvage a transplanted heart. This case demonstrates a rare early complication associated with existing CAD in the donor heart and, to our knowledge, is the first-described postinfarct VSD in a transplanted heart; its likelihood should not be ignored.

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REFERENCES