Early graft failure after heart transplantation due to coronary artery embolization with myocardial tissue

Abstract Myocardial infarction due to myocardial tissue embolism of the right coronary is a rare cause of early graft failure. We report one case with such a complication, in a 44-year-old man who was transplanted with a graft from a 32-year-old male donor. Prolonged refractory cardiogenic shock developed after orthotopic heart transplantation. A right coronary artery bypass graft was performed and the patient was supported with a biventricular assist device for 69 h. Despite that, no improvement of the graft contractility was observed and the patient could not recover from shock. Pathologic examination showed inferior and septal acute infarction. An embolus of myocardial tissue was completely occluding the first portion of the right coronary artery. The tissue probably came from suture lines of the left atrium.

Key words Early graft failure - Coronary embolism - Myocardial tissue

Introduction

Acute graft failure is one of the most disastrous events occurring after heart transplantation and it increases the operative mortality [1]. The reasons for this complication are multiple, but usually they are related to prolonged ischemic time, unsatisfactory myocardial protection and poor previous donor cardiac function. Coronary artery disease (CAD) is one of the most important causes of late graft failure and death after transplantation, but it is also a common cause of early graft failure. Pre-existing coronary vessel atherosclerosis is frequent in old donors and may induce severe graft dysfunction after transplantation [4]. Although extended myocardial infarction with rapid hemodynamic deterioration rarely occurs immediately after transplantation, this situation has been described in cases of intraoperative embolism of the coronary arteries.

In this article we report a case of acute heart failure after heart transplantation due to embolic occlusion of the right coronary artery. To our knowledge there are no cases described in the literature of post-transplant acute infarction caused by muscular tissue embolism of the coronary arteries.

Case report

In April 1994, a 44-year-old man with end-stage ischemic cardiomyopathy was accepted for heart transplantation. He had a prior history of anterolateral myocardial infarction in 1980 with impaired ventricular function because of left ventricular aneurysm. Catheterization at this time showed poor ejection fraction (EF=0.32), high end-diastolic left ventricular pressure (EDLVP=14 mmHg) and single 99% obstruction of the left descending artery (LDA) without criteria for surgical revascularization. He remained stable (NYHA functional class II until January 1993), when he was readmitted to the hospital because of ventricular tachycardia and flutter refractory to pharmacologic treatment. One year later the patient presented with a new episode of tachycardia and flutter refractory to pharmacologic treatment. He was accepted for heart transplantation after new coronary angiography, isotopic ventriculography and echocardiography. His EF had dropped to 0.22 and EDLVP was 19 mmHg. Mean pulmonary
artery pressure (PAP) was 34 mmHg and pulmonary wedge resistance (PWR) was 232 dyne • sec • cm⁻⁵.

A suitable donor was proposed 1 month later. He was a 32-year-old man who died due to cerebral hemorrhage. He had no history of pathologic events and was in good conditions, hemodynamically stable and without the need of inotropics. An earlier echocardiogram proved good contractility of the heart with normal cardiac valves and cavities. Routine exploration of the potential graft revealed no alterations except mild indurations over the right coronary artery and LDA. Graft procurement was undertaken in the usual manner and cold crystalloid cardioplegia followed by multiple doses of cold blood cardioplegia were used to preserve the myocardium. The ischemic time was not too long (180 min) because of the proximity of the donor hospital.

Heart transplantation was performed in accordance to the classical Shumway procedure without complications. Once the anastomoses were completed, the heart cavities were carefully de-aired and purged. Warm blood reperfusion was administered immediately before the aorta was unclamped. After removal of the aortic clamp, the monitor ECG showed ventricular fibrillation that was cardioverted to achieve nodal rhythm; ST elevation was observed in inferior derivations with slow progressive decrease. The graft never returned to sinus rhythm and 35 min after the unclamping, it again presented ventricular fibrillation. After a 70-min period of cardiopulmonary bypass (CPB) assistance, no improvement was observed and the implantation of an intra-aortic balloon pump (IABP) was decided on. At this time clear right ventricular failure had developed with cardiac distention. Ischemic changes in the ECG remained unchanged and digital re-exploration of the coronary arteries revealed patent industrations on the proximal portion of the right coronary artery. After a 3 mm arteriotomy, performed distally to the induration, no flow coming from the right coronary artery was visible and, consequently, coronary artery bypass grafting (CABG) was performed using a saphenous vein. Although mild amelioration of the ventricular contractility was observed, attempts to wean the patient from CPB failed repeatedly. There was refractory severe biventricular failure.

A mechanical support device (Abiomed BVS-5000, Abiomed, Inc. 33 Cherry Hill Drive Danvers, MA 01923 USA) was implanted later on. Biventricular support (BVAD) together with IABP and high doses of inotropics (dopamine 20 µg/kg per min, dobutamine 15 µg/kg per min, adrenaline 0.4 µg/kg per min, isoproterenol 0.7 µg/kg per min) allowed weaning from CPB. The patient could be transported to the intensive care unit, but unfortunately he had to be reoperated on 18 h later, because the right ventricular device performances were insufficient. The right output (RO) had decreased severely (2.1 l/min) with increase in superior vena cava pressure (30 mmHg). Obstruction of the right atrial cannula was suspected. After repositioning of the cannula, a clear improvement of the device output was achieved (RO: 3.5 l/min, I.O: 4.1 l/min).

Once the patient was hemodynamically stabilized, he was put on the transplantation list on emergency code. However, he could not be transplanted because he developed severe respiratory distress, acute renal failure and disseminated coagulopathy during the following 2 days. The patient died on BVAD support 69 h after heart transplantation because of multiorgan failure and neurological coma.

Pathologic examination revealed a transmural acute myocardial infarction, about 2–5 days old, of the posterior wall of the left and right ventricles and also of the posterior interventricular septum (Fig. 1). The right coronary artery was dominant and was completely occluded in its first portion by an embolus formed by muscle fibers of myocardium, focally covered by endocardium and surrounded by subepicardial adipose tissue (Fig. 2). The thickness of the myocardium suggested to us that the embolus probably came from the atrial wall. The embolic mass was impacted against the narrowing of the artery caused by the presence of an atheromatous plaque, which was not significantly stenotic (20%) (Fig. 3). Moreover, moderate atherosclerosis was found in the left anterior descending, circumflex and right coronary arteries of the graft. No mural thrombi were found in the heart cavities.

Fig. 1 A transmural acute infarction can be seen in the posterior wall of the left and right ventricles and in the posterior interventricular septum

Fig. 2 Cross-section of the right coronary artery showing a complete occlusion by an embolus of myocardial tissue. We can see the endocardium (E), the myocardium (M) and the subepicardial adipose tissue (A) (×20)

Fig. 3 Post-mortem coronary artery angiogram showing complete occlusion of the right coronary artery in its first portion
Discussion

Since the 1980s, the operative mortality after heart transplantation has remained at approximately 10% [2]. Early graft failure continues to be one of the important reasons for mortality. Multiple factors have been associated with early graft failure in relation to pre-existing conditions of the recipients and/or with myocardial dysfunction of the donor graft. Before harvesting, myocardial dysfunction may be caused by, the hemodynamic instability of brain-dead donors [4] rather than by pre-existing anatomical alterations of the graft, such as coronary artery disease (CAD). Significant vessel atherosclerosis is unusual in young donors but in older ones the possibility of subclinical CAD has led to the policy of performing coronary angiography before acceptance of donors over 45 years of age [2, 7].

Myocardial infarction has been reported among the factors associated with early graft failure [3, 7]. Probably one of the most important factors is myocardial protection, and also the duration of ischemia. Apart from these factors, in the presence of structurally normal donor heart with a low probability of CAD, the reason for acute myocardial infarction after transplantation have been related to complications involving the coronary artery tree. Several different reasons have been reported in the literature [3, 6], such as ligation of the left circumflex artery with the suture from the left atrial anastomoses, traumatic dissection of the LDA or coronary sinus obstruction because of oversewing and coronary embolism.

Embolization in the coronary arteries after cardiac surgery is not usual and few cases of coronary embolism have been reported after heart transplantation [3, 6]. Air bubbles, thrombi fragments, bone marrow fragments, foreign bodies (cotton, wax) and fatty tissue fragments are among the different materials reported as emboli in the coronary arteries. As far as we know, our case is the first one with muscular embolism of the right coronary artery. Probably the embolus came from the left atrial suture lines. Although we usually pay careful attention to left atrial suture lines, that we systematically oversew, little fragments of fatty or muscular tissue can be detached from the suture lines and, if not suctioned, these may produce embolism.

The fact that the donor heart presented some indurations over the coronary artery tree, especially palpable in the proximal portion of the right coronary artery, led us to perform a saphenous vein bypass after failed attempts to wean from CPB. In spite of that, no improvement of the cardiac function could be obtained, probably because the coronary bypass was performed too late. The decision was delayed because the patient presented right ventricular failure with supraventricular rhythm troubles, as usually happens in patients with pulmonary hypertension. Furthermore the ischemic changes in the monitor ECG were not characteristic.

Some cases of concurrent bypass grafting with heart transplantation have been reported [5]. In all cases the CABG were performed electively before graft implantation, as the authors were accepting donor grafts with known CAD. The early results of this series have been good. As stated previously, the CABG in our patient was performed after transplantation when irreversible ischemic changes had probably developed. We were confused by the bad course of the infarction, especially in a graft coming from a totally asymptomatic young donor. We were also surprised when the anatomopathology examination showed embolism of the right coronary artery with a fragment of muscular tissue, although this answered many enigmatic questions.

We believe that this complication can only be avoided by careful attention during the anastomoses and by exhaustive suction of cardiac cavities before unclamping of the aorta.

References