Stenting of unprotected left main stem stenosis in cardiogenic shock after orthotopic heart transplantation

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Abstract

We report the spectacular case of a young patient who developed cardiogenic shock caused by significant stenosis of the left main stem 8 years after orthotopic cardiac transplantation. Immediate percutaneous coronary intervention was a life saving procedure. The report illustrates the importance of early coronary angiography for the clinical management of heart transplant patients in cardiogenic shock as a relevant differential diagnosis to acute rejection. Allograft vasculopathy can develop unexpectedly even after years of uncomplicated follow-up.

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1. Introduction

Transplant allograft vasculopathy is a major contributor to morbidity and mortality after cardiac transplantation. As clinical signs like angina pectoris are often subtle, coronary angiography and myocardial biopsies are regularly scheduled in order to identify patients at risk. This case report describes a patient who was under routine control, in stable clinical condition, 8 years after orthotopic heart transplantation and developed severe left main stenosis that could be successfully treated by emergency coronary stenting.

2. Case report

The 29-year-old male patient had orthotopic cardiac transplantation for terminal heart failure due to dilated cardiomyopathy in 1995. He was on the waiting list for 4 months. The periprocedural course was uneventful and immunosuppression was started on cyclosporin A 250 mg/day, prednisolon 50 mg/day and azathioprine 100 mg/day. Systemic Cytomegalie viral infection occurred in 1995 and was successfully treated by ganciclovir. Twelve months later steroids were withdrawn according to the local protocol. In 1998, the patient had one episode of acute cardiac rejection that was graded IIb SHLT and treated with steroids. Outpatient control of risk factors, immunosuppression and infections were managed by the transplant center.

Chronic medical therapy included simvastatin (20 mg/day), aspirin (100 mg/day) and diltiazem (180 mg/day) as prophylaxis against cardiac allograft vasculopathy (CAV). Coronary angiograms were performed once a year and no relevant allograft vasculopathy was identified conventionally. Diagnostic intracoronary ultrasound (IVUS) was not performed routinely. Cardiac function was normal, ejection fraction (EF) was 55% and regional wall motion abnormalities were not detected.

Five months after the last normal angiogram the patient developed tachycardia, weakness and shortness of breath on exertion. Within two days he was admitted to a local hospital in respiratory failure. Mechanical ventilation was initiated with the diagnosis of severe opportunistic pulmonary infection and the patient was referred to the transplant center. At admission the patient was in progressive cardiogenic shock (blood pressure 85/65 mmHg). Intravenous catecholamine infusion had to be increased continuously (epinephrine <0.5 μg/kg/min). Twelve-lead surface EKG showed sinus rhythm at 110 bpm and no characteristic ischemic alterations. Echocardiographic assessment showed a severely reduced LV-function (EF 20%). Lab results: C-reactive protein 15.8 mg/dl, (normal range: 5.0–10.0), Troponin T: 0.04 ng/ml (normal range: <0.01). In the emergency setting, it was decided to rule out transplant vasculopathy by coronary angiography and to obtain endomyocardial biopsies to identify acute rejection immediately as Troponin T was slightly elevated. At coronary angiography, an injection of contrast agent in the left coronary system showed a 95% concentrical stenosis of the main stem and a 60% stenosis of the left anterior descendent coronary artery (LAD) located distally to the first septal

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branch (see Fig. 1). Although blood pressure was extremely low, intracoronary nitrate (0.05 mg) was injected to rule out coronary spasm. After consulting the cardiac surgeon in the catheterization laboratory, to discuss all invasive options, the decision for acute coronary stenting was made because of the extremely rapid progression of cardiogenic shock within minutes and increasing catecholamine support. Systemic thrombolytic treatment was considered, but declined as morphology of the target stenosis was less suggestive for thrombus formation than for a lesion caused by graft vasculopathy (Fig. 1). Coronary spasm was ruled out by local low dose nitrate infusion via the guiding catheter. Intraaortic balloon pumping was initiated via the left femoral artery to improve coronary perfusion. A guiding catheter 6F JL 4 SH was placed near the LM ostium and a coronary stent (Cypher® 3.5 × 13 mm) was directly implanted into the left main stem. Abciximab (ReoPro®) was infused at recommended standard dose for 12 h.

After intervention the patient could be weaned from inotropic support within 24 h and was discharged home one week later. His ejection fraction was 40%. At his scheduled control angiography 6 weeks later the LAD stenosis was successfully stented (Fig. 2).

Three years later the patient is well and clinically stable. He had repeated coronary angiograms with normal findings.

3. Discussion

Cardiac allograft vasculopathy, as one manifestation of chronic rejection, is the major cause of morbidity and mortality among up to 50% of patients after heart transplantation [1,2]. Compared to conventional atherosclerosis, narrowing is more diffuse and less focal [3]. The left main stem is rarely involved significantly and acute plaque rupture seems to be rare as compared to conventional atherosclerosis. Predisposing factors for the development of cardiac allograft vasculopathy are initial endothelial injury by ischemia and reperfusion [4], immunosuppression, episodes of rejection, older donor age, cytomegalic viral infection, hypercholesterolemia [5], and others [3]. Our patient experienced one episode of allograft rejection proven by endomyocardial biopsy, one cytomegalic viral infection and hypercholesterolemia as predisposing condition for accelerated graft vasculopathy. As patients do not exhibit typical anginal symptoms regularly scheduled coronary angiograms are recommended during elective follow-up. Doshi documented a series of percutaneous interventions for allograft vasculopathy and found a mean interval of 7.8 years between transplantation and the need for revascularization [6] as in the presented case. The role of percutaneous coronary balloon angioplasty, and stenting, and bypass surgery for revascularization of patients with significant graft vasculopathy has been described before [6–8]. However, stenting of the unprotected left main stem in cardiac transplant patients has only been published in very few case reports [9,10]. This is the first report documenting direct emergency stenting of unprotected left main stenosis in cardiogenic shock for transplant vasculopathy. For the patient, it proved to be a successful strategy in a 2-year follow-up.

4. Conclusions

Cardiac allograft vasculopathy can develop unexpectedly in stable transplant recipients. In patients presenting with acute heart failure after cardiac transplantation, it is crucial to identify allograft vasculopathy as differential diagnosis to acute rejection early in the clinical course. As complex atypical coronary pathology might be identified, a well reflected interdisciplinary surgical or interventional strategy must be developed immediately.
References