ST Elevation Myocardial Infarction Early After Heart Transplantation

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Introduction

Heart transplantation is the last option to improve survival in end-stage heart failure. Unfortunately, the increased need for organs far outweighs the availability of donors. To overcome this situation and to increase the number of available hearts for transplantation, donor selection criteria were made less stringent[1-3]. Nowadays, the one-year survival rate after transplantation is close to 90%[4-6], with 50% of the patients surviving for more than 10 years[6]. The main factor limiting survival is allograft vasculopathy (AV), which occurs in up to 10% and 50% of the patients at 1-year and 5-year after transplantation, respectively[5].

ST elevation myocardial infarction (STEMI) is rare in these patients and is even more uncommon shortly after transplantation. Despite being an uncommon manifestation of AV, STEMI is usually related to AV[6,7].

Case Report

We describe the case of a 65-year-old man, former smoker with diabetes, hypertension, dyslipidaemia, and end-stage ischaemic cardiomyopathy who underwent orthotopic heart transplantation. He received the organ from a 55 year-old obese male with hypertension and dyslipidaemia. The cold ischemic time was approximately 1 hour; the donor was under dopamine perfusion (8mg/kg/min) for 12 hours. The donor heart had normal left ventricular ejection fraction. The heart was harvested without angiographic screening (not mandatory according to the local policy) and was therefore not tested for the presence of coronary heart disease (CAD).

After transplantation, the patient remained under amine support for 36 hours and mechanical ventilation for 12 hours. He was maintained on a regimen of cyclosporine, azathioprine, and prednisolone.

At the fourth day after transplantation without any clinical signs or symptoms (namely chest pain), a rise in the ST segment was monitored, associated with an elevation in myocardial lesion markers. Subsequently, an ECG was performed in the emergency setting and showed an ST elevation from V2 to V6 (Figure 1). The transthoracic echocardiogram revealed moderate left ventricular dysfunction associated with akinesia of the anterior wall, septum, and apex. The patient underwent urgent cardiac catheterization that revealed an occluded left anterior descendent (LAD) in the mid segment, without significant lesions in the remaining vessels. Angioplasty with implantation of a bare metal stent was successfully performed (Figure 2). The patient evolved in Killip Kimball class II with a peak troponin of 86 μg/L (normal range, < 0.5 μg/L) and an early post-infarct left ventricular ejection fraction of 40%. Despite being under optimal medical therapy (including dual antiplatelet therapy, ACE inhibitors, spironolactone and statins) he experienced several medical complications during his hospital stay, including progressive heart failure (NYHA III), multiple infections and sudden death after three months. The autopsy showed areas of infarct of different evolution stages in the anterior wall and septum, including areas of recent infarct, which was assumed as the cause of death.

This is a very rare case of an early STEMI in a transplanted heart not associated with AV, but probably due to "traditional" coronary artery disease of the donor's heart.

Discussion

There are only few epidemiologic and clinical data on MI following heart transplantation. This lack of data is likely due to the rarity of the event and due to the fact that the diagnosis is missed in the case of asymptomatic MI[7]. Moreover, data come from different types of sources such as autopsy registries, clinical trials of percutaneous intervention, and imaging cohort studies, making the estimation of the overall incidence and the characterization of the event difficult[7]. The clinical manifestations include typical angina symptoms (in those 10–30% who have nerve regrowth), fatigue, heart failure, arrhythmias, and death. However, most patients are asymptomatic or have an atypical presentation as a result of cardiac denervation[7]. Therefore, these patients can experience multiple silent MIs at any time after transplantation, resulting in loss of allograft function, heart failure, or sudden death.

STEMI in heart transplant bearers can occur early or late after the transplantation, probably reflecting different pathophysiologic mechanisms. Intravascular ultrasonography of the coronary circulation of transplanted hearts showed the presence of two types of lesions: 1) focal, non-circumferencial and proximal, donor-transmitted or *de novo* lesions and 2) a diffuse, concentric pattern that is related to AV[6-8].

Late STEMI is more often related to AV, an accelerated arteriosclerosis characterized by diffuse, longitudinal, and concentric proliferation of intimal smooth muscle and extracellular matrix[8].

Keywords

Myocardial Infarction / complications; Heart Transplantation/ mortality; Amines/therapeutic use; Respiration Artificial; Cyclosporine/therapeutic use; Survivorship (Public Health).

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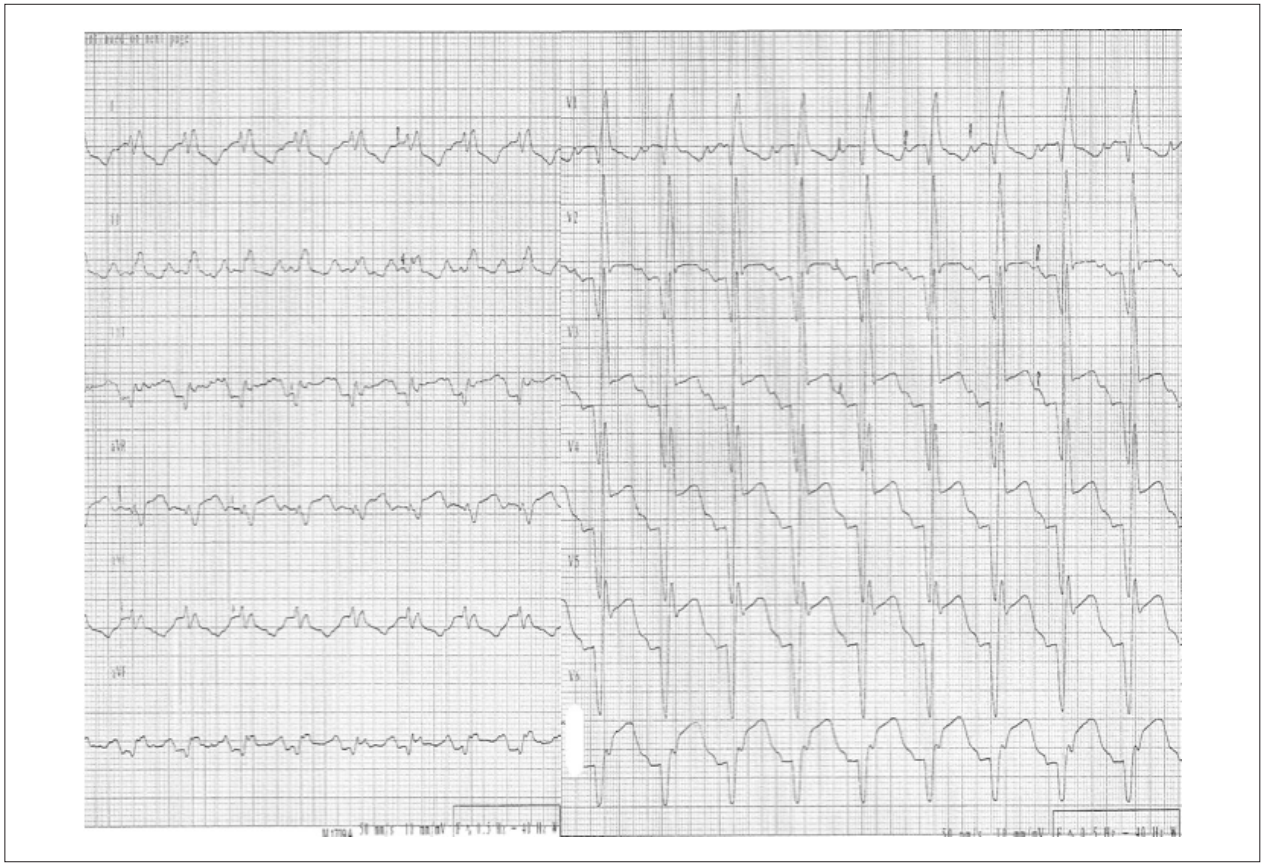

Figure 1 – *ECG during the acute phase.*

Early STEMI is very rare and can be related to procedural features like prolonged organ cold ischemia time, tissue embolization, postsurgical coronary air embolus, and thrombosis. It can also be associated with CAD of the donor's heart[7]. An autopsy registry of patients who died within 2 months after transplantation showed a 30% prevalence of atherosclerosis of the native coronary arteries in early allografts[6].

All transplanted patients have an increased risk of MI. Thrombosis can result from surgery/immune related inflammation and immunosuppressive therapy associated metabolic and hemostasis imbalance. In addition, patients with history of MI may have an underlying basal propensity for thrombosis[6,7]. This effect can also be more pronounced in patients who are already at higher risk, like those with established atherosclerosis.

The widening gap between waiting recipients and donors has given rise to ethical issues concerning the allocation of the organs. The awaiting population includes patients with different orders of priority and borderline candidates (elderly patients, younger patients with significant comorbidities, and patients requiring re-transplantation). In order to satisfy the needs of these borderline candidates, a policy of extending the criteria and acceptance of marginal donors has been adopted.

Several studies have shown that survival with marginal hearts can be similar to that with standard donors[1,9]. Other studies however showed increased early or late postoperative mortality and morbidity[10]. Some of these unfavorable results may be due to the fact that older donor hearts traditionally have been allocated to older or high-risk patients or are used as a biological bridge to transplantation until a younger allograft becomes available[1,9]. Some surgeons transplant borderline hearts to high-risk recipients because they feel that these patients may have the greater marginal benefit from these hearts. Others transplant these organs in low-risk recipients to reduce the overall risk as the recipient risk is the overriding determinant of immediate post-transplant survival[2].

Either way, this strategy should be an option only when the survival benefit for the recipient unequivocally exceeds the decrement in early survival due to transplantation of a high-risk cardiac allograft. Conversely, a risk stratification policy with widespread CAD exclusion is still lacking and high-risk allografts are harvested and allocated, contributing to worse outcomes. Therefore, only if properly selected, older donor hearts with negative cardiac history, normal electrocardiogram and echocardiogram, low inotropic support, normal coronary angiogram, and an expected short ischemic time, will fill the void of organs and better outcomes can be expected[1].

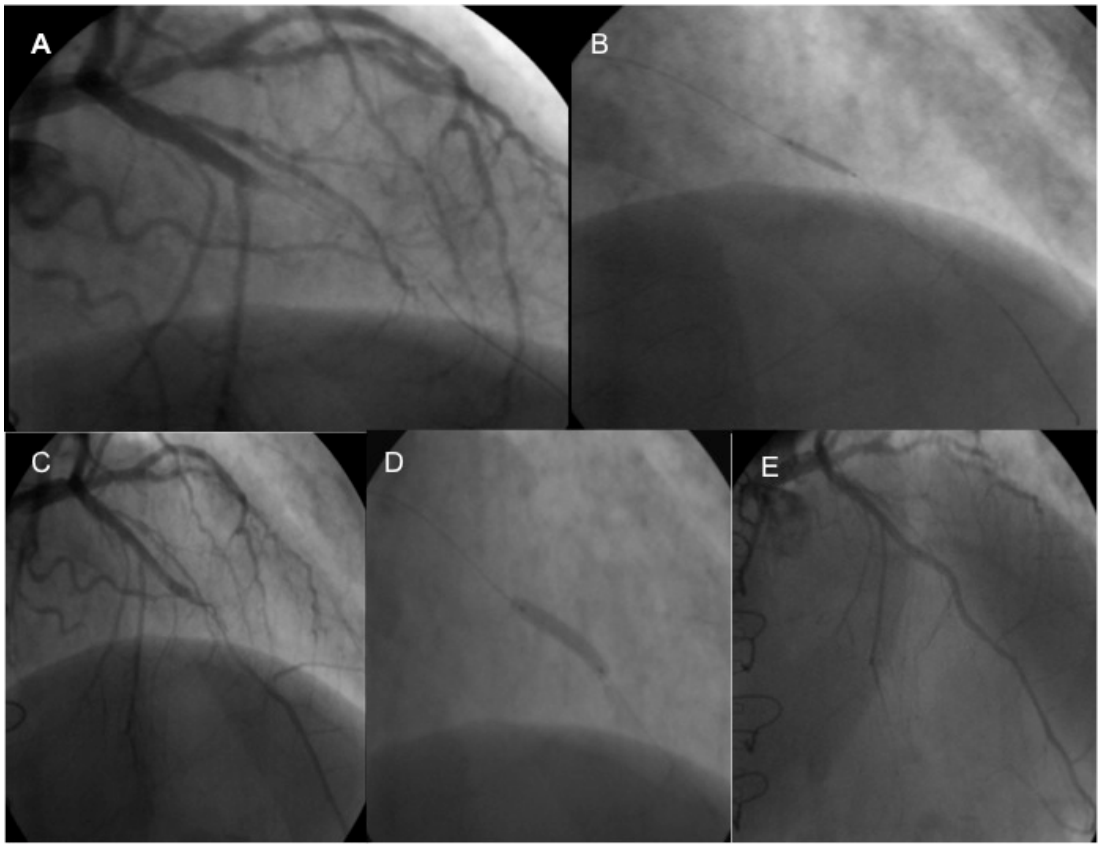

Figure 2 – *Primary coronary intervention. A) Angiogram showing an occluded left descendent artery (LAD) in the mid segment, B) Balloon angioplasty, C) reperfusion of LAD revealing the distal vessel, D) Stent implantation, and E) TIMI 3 flow.*

This case is an example of the issues that can emerge after transplantation of an organ from a marginal donor to a borderline recipient, and illustrates the need for correct stratification of the potential donors and recipients.

Currently, invasive coronary angiography is not mandatory; however, other non-invasive tests (with or without use of contrast dye) may be used to exclude CAD, and to estimate the functional reserve of the donor heart. Cardiac CT using calcium score with or without angiography could be useful to evaluate the presence of significant CAD. Stress echocardiography can as well diagnose significant CAD and appraise cardiac reserve.

Because we are dealing with such priceless resources, it is important to use all the means we have available to select and allocate properly the transplanted hearts.

Conclusion

This is a case of a very early STEMI after transplantation, which highlights the need for special and continuous attention to clinical, laboratory, and electrocardiographic features in the early perioperative period, as manifestations are frequently atypical.

The proper selection and allocation of organs is fundamental for the success of transplantation. Therefore risk should be estimated for all possible matches with the appropriate tests according to institutional resources.

Author contributions

Conception and design of the research and Writing of the manuscript: Madeira SL; Acquisition of data and Critical revision of the manuscript for intellectual content: Madeira SL, Raposo LF, Madeira M, Marques M, Rebocho MJ.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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