Perioperative Acute Myocardial Infarction Complicated with Prolonged Delayed Graft Function in A Renal Transplant Recipient

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Abstract

A 61-year-old woman with type 2 diabetes mellitus was admitted for living donor renal transplantation. Sudden onset of acute pulmonary edema developed one day post-transplant and was proved to be due to acute myocardial infarction. Urgent coronary arterial bypass grafting was performed immediately due to failure of percutaneous coronary intervention and cardiogenic shock. This was followed by a prolonged period of delayed graft function (53 days). Fortunately, she had full recovery of graft function with a discharged serum creatinine of 0.8 mg/dL. (J Intern Med Taiwan 2011; 22: 283-286)

Key words: Renal transplantation, Acute myocardial infarction, Delayed graft function, Diabetes mellitus

Introduction

Cardiovascular disease (CAD) is the most important complication in diabetes mellitus (DM) patients, causing significant morbidity and mortality. End-stage renal disease secondary to DM confers an even higher risk of developing CAD, because of the well-known nontraditional factors such as oxidative stress, hyper-homocysteinemia, calcium-phosphate derrangement and others. When such patients are subjected to major operations, subtle change in hemodynamics may elicit critical disturbances in myocardial perfusion and lead to the development of acute myocardial infarction (AMI). Herein, we report a case of a patient undergoing living related donor kidney transplantation who developed AMI one day post-operation. Aggressive management followed by intensive care led to the resumption of both normal activities and the graft function, even after a prolonged period of oliguria.

Case report

A 61-year-old woman had type 2 DM for 20 years and hypertension for 22 years. Six years ago,

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she was diagnosed as having diabetic nephropathy. Meanwhile, she suffered from non-ST elevation MI which was diagnosed via V4-V6 depression over electrocardiography (ECG) and typical chest pain. Percutaneous intervention (PCI) showed left anterior descending (LAD) diffuse 50-60% stenosis at distal part, so one stent was implanted. After that, she did not complain of any chest discomfort. Four years ago, she went into uremic stage and was treated with peritoneal dialysis. Two years later, she was evaluated for living donor kidney transplantation, and received a donor kidney from her younger sister who was 50 years old without any systemic disease, such as DM or hypertension. Pretransplant evaluation for the recipient revealed normal left ventricle (LV) wall motion with mild pulmonary hypertension (peak systemic pulmonary gradient was 32mmHg) and normal ECG tracing without any ST-T change. Renal transplantation was performed without significant drop in blood pressure during 4-hour operation. However, the immediate urine output was suboptimal despite adequate hydration and the use of diuretics. A chest X ray (CXR) showed pulmonary congestion. So 2 liters of ultrafiltration were prescribed on the next day. The process was uneventful and there was no ECG change during the whole course.

Unfortunately, 36 hours after operation, the patient suffered from frank blood in frothy sputum, marked dyspnea and chest tightness. The arterial blood oxygen saturation was only 84% under 100% of FiO_2 mask. CXR showed acute pulmonary edema. The fluid balance was only positive for 590ml without taking into account the insensible loss. ECG showed poor R wave progression and ST segment depression over V4-V6. The cardiac enzyme levels were: creatinine kinase (CK) 623U/L, CKMB 60U/L, and troponin I 15.3ng/mL. PCI revealed CAD, double vessel disease, with left main lesion. CABG (coronary artery bypass graft) surgery was arranged immediately and lasted for 9 hours. The subsequent hospital course was complicated with intrathoracic bleeding on post-transplant day 5, which required reopening of the chest to check bleeding and sudden onset of asystole during dialysis on day 10, which returned to normal rhythm without any sequalae after 28 minutes' resuscitation. She was finally weaned from dialysis on post-transplant day 53 and from ventilator on day 54. During the oliguric phase, graft biopsies were done. Both showed acute tubular necrosis without any sign of rejection even with low dose immunosuppressive agents, which included prednisolone 15 mg once per day, mycophenolate mofetil 500mg twice per day, and tacrolimus 1mg twice per day. She was discharged with a serum creatinine of 0.8 mg/dL. Although she suffered from peripheral arterial occlusive disease over right lower limb which resulted in below-knee amputation 3 years later, her serum creatinine remained at around 0.9 mg/dL at 5 years post-transplant. She did not experience any rejection.

Discussion

Perioperative myocardial infarction is one of the most important prognostic indicator of short term and long term morbidity and mortality in non-cardiac surgery[1]. In patients with or at risk of CAD, the reported incidence of perioperative myocardial ischemia varies between 20 and 63% and the incidence of postoperative myocardial infarction was between 1.4% and 38%[2]. Perioperative pulmonary edema is not uncommon in renal transplants. "Transplant lung" which was described by Uranga et al in 1968[3], could be due to a variety of pathological processes. In our patient, the absence of infection and overt fluid overload strongly suggested that acute pulmonary edema was secondary to acute heart failure, a situation that was inevitably linked to the past history of CAD. Despite the absence of coronary syndrome, normal ECG and normal echocardiography, acute MI still occurred

one day post-transplant. Several mechanisms may be implicated. Firstly, the stress of operation might trigger the release of catecholamines which could augment the oxygen consumption of myocardium[4]. This increased oxygen demand might be well above what the diseased coronary arteries could supply. In addition to elevated catecholamines, activation of sympathetic nervous system also causes increased blood viscosity, heart rate, and prothrombotic activity accompanied with increased platelet aggregation and decreased fibrinolytic activity. All the above factors can increase susceptibility to myocardial ischemia. Secondly, although there was no appreciable drop of blood pressure during operation, the diseased myocardium might be more sensitive to subtle change of hemodynamics that was not reflected during blood pressure monitoring. Thirdly, ultrafiltration was performed on first postoperation day due to pulmonary congestion. It is well-known that the blood-membrane interaction during hemodialysis can cause a cascade of immune reaction, characterized by the activation of complement system and leading to leukocyte aggregation within the microcirculation of pulmonary vascular bed[5]. The resultant transient hypoxemia might have rendered the diseased myocardium susceptible to further ischemic injury. All the above conditions trigger a mismatch of myocardial oxygen supply and demand.

According to guideline of American Society of Transplantation (AST), pre-transplantation heart survey, our patient was at higher risk (renal disease from diabetes, prior ischemia, and ≧ 2 traditional risk factors (age ≧ 55 years in woman, diabetes, and hypertension)[6]. A stress test may disclose unexpected stenosis of coronary artery and should prompts a coronary angiographic study. The painful experience of the current case has led to the development of a new strategy for our transplant program. All patients who are at high risk of CAD should receive multidetector computed tomography of coronary arteries and stress test. Patients with positive tests will be subjected to PCI and treated accordingly. Our patient recovered and regained a satisfactory graft function. The key to the success was a prompt diagnosis and aggressive treatment. It is interesting that even with severe repeated injuries, the allograft still can achieve a full recovery. Although it has been well-documented that acute tubular necrosis may render the graft more immunogenic by the exposure to HLA antigens and trigger subsequent rejections, our patient is fortunately free from any rejection episodes as of this writing.

Conclusion

Our patient represents an extreme case that was associated with severe cardiovascular complication and accompanied with a prolonged period of delayed graft function. A pretransplant thorough examination of coronary arteries in high risk patient is mandatory to avoid risk of the morbidity and mortality.

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於腎移植之後立即發生急性心肌梗塞造成移植腎功能延遲恢復 - 病例報告

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摘 要

腎臟移植是目前對於末期腎病變病人最好的治療方法，尤其是活體移植有更好的預後。腎移植後病人的心血管問題仍然是最重要的內科問題。我們報告一例六十一歲第二型糖尿病的病人接受活體腎移植，在移植後的第一天，因爲急性心肌梗塞而發生急性肺水腫。心導管檢查爲阻塞三條冠狀動脈，之後發生心因性休克而立即接受心血管外科手術。後續的治療在移植的第五十三天，病人的腎功能開始恢復，而後可以順利出院。幸運的是，她目前的腎臟功能（肌酐酸）仍然維持在 0.9 mg/dL。