Cardiovascular disease is a major contributor to morbidity and has become the leading cause of death among renal transplant patients. This paper presents a case of acute myocardial infarction occurring three weeks after a renal transplant. A 45-year-old male recipient experienced acute chest pain, hypotension, and atrial fibrillation, leading to a diagnosis of inferior-posterior wall myocardial infarction. Thrombolysis with Tenectaplace was administered, resulting in the development of a perinephric hematoma six hours later. The patient underwent transfusions and pigtail drainage of the hematoma. Following hematoma resolution, the pigtail catheter was removed, and the patient was started on dual anti-platelets. Upon discharge, the patient exhibited stable renal function.

In conclusion, thrombolysis in the early post-transplant period poses significant risks. This case highlights the use of thrombolytic therapy during this period and the successful management of associated complications.

Keywords: myocardial infarction, renal transplant, thrombolysis.

Conflict of interest. The authors declare no conflict of interest.
Тромболізис в ранньому посттрансплантаційному періоді у пацієнта з гострим інфарктом міокарда: клінічний випадок

Медичний коледж Шрі Рамачандра, Ченнаї, Індія.

Резюме. Серцево-судинна захворювання є основною причиною смертності пацієнтів після трансплантації нирки. У цій статті представлено випадок гострого інфаркту міокарда, який стався через три тижні після трансплантації нирки. У 45-річного чоловіка-реципієнта виник гострий біль у грудній клітці, артеріальна гіпотензія та фібриляція передсердь, було діагностовано інфаркт міокарда нижньої та задньої стінки. Пацієнту проведено тромболізис тенектаплазою, що призвело до розвитку навколониркової гематоми через шість годин. Після розрішення гематоми на фоні її дренування та гемотрансфузії пацієнту призначено подвійну антиагрегантну терапію. Виписаний зі стаціонару зі стабільною функцією нирок.

Ключові слова: інфаркт міокарда, трансплантація нирки, тромболізис.

Introduction. Cardiovascular disease is a significant cause of morbidity and is now the leading cause of death in patients with renal transplants [1, 2] and, consequently, one of the leading causes of renal allograft failure [3]. Diabetic renal transplant recipients have a 3-5 times higher risk of cardiovascular disease than non-diabetic recipients [4]. End-stage kidney disease confers a higher risk of developing cardiovascular disease (CVD) because of the well-known “traditional” risk factors [5], as also the non-traditional factors such as oxidative stress, hyper-homocysteinemia, calcium-phosphate derangement, and others. Candidate risk factors for post-transplant myocardial infarction (PTMI) include characteristics of the recipient, the kidney donor, transplantation management, and transplant course. Time on dialysis before transplantation [6], donor history of hypertension [7], immunosuppressive regimen [8], quality of allograft function [9], and post-transplantation diabetes [10, 11] are also implicated as mediators of cardiovascular risk in this population. However, managing acute myocardial infarction (MI) in the post-transplant period remains challenging. Herein, we report a case of a patient who underwent deceased donor renal transplantation (DDRT) and developed AMI three weeks post-transplant.

Case Report. A 45-year-old man presented with sudden onset left-sided chest pain associated with vomiting and diaphoresis. He was a known case of Type 2 Diabetes Mellitus and hypertension. He was diagnosed with chronic kidney disease two years back when he was initiated on continuous ambulatory peritoneal dialysis (CAPD). His cardiac function was evaluated three months before admission and found normal with left ventricular systolic ejection function (LVSEF) of 63%, normal treadmill test, and a normal myocardial perfusion — single photon emission computed tomography (SPECT) scan. In addition, a repeat electrocardiogram and echocardiography were found to be normal before the transplant. He underwent a Deceased Donor Renal Transplant (DDRT) three weeks before admission, with Basiliximab induction and triple immunosuppression protocol (Tacrolimus, Mycophenolate mofetil, and Prednisolone). He had no postoperative complications and was discharged in two weeks with a creatinine of 0.7mg/dl.

On examination, he had a pulse rate of 110/min, irregularly irregular, with a blood pressure (BP) of 90/60 mmHg and a pulse deficit of 19/min. Systemic examination revealed signs of atrial fibrillation, and the transplanted kidney was palpable in the right iliac fossa, non-tender with a clean and healed surgical wound.

ECG was done, which revealed tachycardia, Atrial fibrillation (AF), and ST elevation in leads II, III, aVF, and in the chest leads v4-v6, with ST depression in leads I, aVL, and v2. In addition, a 2D-Echocardiogram showed concentric left ventricular hypertrophy, trivial mitral and tricuspid regurgitation, normal pulmonary artery pressure, and hypokinesia of the inferior-posterior wall with a left ventricular ejection fraction (LVEF) of 55%.

The options of bypass grafting surgery, primary angioplasty, or thrombolysis were discussed with the cardiologists and attendants. After a multi-disciplinary discussion, the patient was planned for thrombolysis. Thrombolysis with Tenectaplahse was administered on 13/07/2015, within 90 min of the onset of chest pain. There were no complications during thrombolysis, BP reverted to normal, and sinus rhythm was restored. There was also a partial resolution of ST elevation on the ECG.
However, the investigations showed a drop in hemoglobin from 11g/dl to 9g/dl at 6 hours post-thrombolysis. An ultrasound (USG) of the abdomen and pelvis was performed, which showed a perinephric collection measuring 8.7x5.6x8.6 cm (approx 220 ml), suggestive of a hematoma (Fig. 1).

![Fig. 1](image)

Fe. 1. Ultrasound showing collection around the transplanted kidney (a) with thin septations (b).

A repeat USG abdomen and pelvis 14 hours later showed an increasing hematoma size with a collection measuring 11x5.5x11 cm (approx 354 ml) and moderate hydroureteronephrosis (HUN) of the transplanted kidney. His hemoglobin decreased to 6.8 g/dl, and serum creatinine increased from 0.8 to 1.5 mg/dl.

Given the increasing hematoma and HUN, a USG-guided 10F pigtail catheter was inserted in the peri-nephric collection, and about 1600ml of hemorrhagic fluid was aspirated. He was transfused two units of packed red cells. There was no BP drop, and hemoglobin stabilized around 10.8mg/dl. Pigtail drainage decreased gradually to about 30ml by day 3. Repeat USG on day 5 showed a collection of size 4.5x1.5x4.7 cm in the perinephric region, with no HUN. Pigtail drainage ceased, the catheter was removed on day 8 with USG showing no collection, and serum creatinine decreased to 0.7mg/dl with a hemoglobin of 11.2mg/dl. He was started on dual anti-platelets (aspirin and clopidogrel) on day 11 and discharged on day 13 with no further bleeding manifestations, stable renal function, and an LVEF of 59%.

Discussion. The risk of CVD in renal transplant recipients is being increasingly appreciated [12]. However, evidence-based data on the appropriate therapy and the benefits of primary or secondary prevention in such patients is not yet adequate to formulate a guideline [13]. The increasing awareness also raises the issue of whether such a cohort of patients has a different pathophysiological basis for CVD compared to the general population [13]. There is an increasing need for cardiovascular outcome studies in renal transplant recipients. Still, the generalized applicability of the outcomes of such studies is hampered by the size of the sample population. Previous studies have reported aggressive surgical and revascularization attempts in the early post-transplant period, but they could not be employed in our case due to logistic difficulties.

The above case illustrates a few key issues. Firstly, CKD patients may have advanced cardiac disease by the time they are allotted a deceased donor kidney, irrespective of the previous clinical or investigative features. Secondly, pre-operative investigations, especially non-invasive assessment, may not accurately predict the pot-transplant CVD risk in diabetic CKD patients. Thirdly, non-surgical revascularization procedures may have an outcome like the non-renal population but are associated with increased risks. Finally, with the increasing awareness of the risk of CVD in the post-transplant period, the ideal time for initiating a preventive approach is during the early stages of CKD rather than during the post-transplant period. The increased evaluation of CVD risk factors and their adequate management with the use of anti-hypertensives, statins, etc., will contribute to improved survival of the transplant population, as much as the advances in immunosuppression.

Conclusion. Our patient represents a case of severe cardiovascular complication in the early post-transplant period. A detailed assessment of the coronary arteries is essential in high-risk patients, especially diabetic CKDs. There is an unmet need to develop reliable risk estimates for CVD in the post-transplant population [14]. Thrombolysis in the early post-transplant period is considered a very high-risk procedure and is associated with significant risks. Our case illustrates the use of thrombolytic therapy in the early post-op period and the successful management of the attendant complications.

Conflict of Interest: The authors declare no conflict of interest.

Sources of funding: None.

The authors’ contributions.

V.K.B. Bandi: data acquisition, data analysis/interpretation, manuscript writing;
M.S. Shekar: conceptualization, supervision, manuscript review;  
R.E. Elumalai: conceptualization, supervision, manuscript review;  
V.M. Mamidi: data acquisition, data analysis/interpretation;  
V.K.M. Makkena: data acquisition, data analysis/interpretation.

References:


