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A Rare Reason of Kidney Failure in a Renal Transplant Recipient: Atrial Fibrillation

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Abstract

The main causes of renal dysfunction in renal transplant recipients are acute or chronic allograft rejection, recurrent and de-novo glomerular diseases, BK virus nephropathy, renal artery stenosis, and rarely ureter obstruction. Renal infarction is a rare condition which is caused by renal artery occlusion and it might be resulted in renal parenchymal damage.The most common etiology of renal infarction is atheroembolic diseases. Other causes are trauma, autoimmune diseases and hypercoagulability. Renal infarction of the kidney is very rare and its incidence is not clear in patients with kidney transplantation. Herein we presented a renal transplant recipient with renal dysfunction due to renal infarction as a result of embolism due to atrial fibrillation, resulting in permanent renal dysfunction.

Keywords: *Kidney transplantation, atrial fibrillation, renal infarction.*

Introduction

Kidney transplantation is the best treatment option in patients with end-stage renal disease. Thecommon reasons for kidney dysfunction in renal transplant recipients are drug induced nephrotoxicity caused by certain drugs such ascalcineurin inhibitors, ischemic reperfusion injury, recurrent glomerular diseases,BK or cytomegalovirus (CMV) virus infections, acute orchronic allograft rejection. Diagnostic work-up such as therapeutic drug concentrations, complete urinalysis, renal imaging, and diagnostic biopsy should be performed before irreversible nephron damage occurrence. Interventions to be performed depending upon the results , the reduction or cessation of calcineurin inhibitors if drug-induced nephrotoxicity is detected, strengthening immunosuppression to prevent chronic rejection, as well as ensuring control of blood pressure, proteinuria, dyslipidemia, diabetes, smoking and other comorbidities [1].

Renal infarction is a rare condition that is caused by renal artery occlusion and usually causes permanent renal parenchymal damage [2-7]. It has been shown to be seen at a low level of incidence approximately 0.007% in all emergency department admissions [8], therefore, the diagnosis is often delayed. The mean age of the patients with renal infarction is 40 years old;however it may vary in different etiologies. [9]. The main causes of renal infarction in general population are; cardioembolic diseases, renal artery injury and hypercoagulable states [2,4,6,9,10]. However, the incidence of renal infarct leading to allograft dysfunction in renal transplant patients is not well known. It is very rare that renal embolism may underlie the acute allograft dysfunction. In this case report, we present a patient who progressed to end stage renal failure due to atrial fibrillation-related allograft infarction in a renal transplant recipient.

Case Report

A 53-year-old man was admitted to the emergency department with complaints of nausea, vomiting, chills, shivering, fever and palpitations in December 2017. He had been under follow-up for 10 years with the diagnosis of chronic kidney disease and hypertension. Kidney transplantation had been performed five years ago from a living donor. The serum creatinine level after transplantation was in the range of 0.7-0.9 mg / dL. On the admission the patient's physical examination revealed mild suprapubic and right inguinal tenderness,pulsusparadoxus and tachycardia. Vital signs were as follows; fever: 38.4 ° C, heart rate: 160 / minute, pulse 88/ minute, respiratory rate: 26 / minute, blood pressure: 120/80 mmHg. In laboratory tests, serum creatinine level was 4.3 mg / dL, white blood cell count (WBC) was 26.78* 10^3/µL (4.8-10.7), neutrophil count (NE) 24.17*10^3/µL (2.2-4.8), lactate dehydrogenase (LDH) level of 2380 U / L (135-250), erythrocyte sedimentation rate : 6 mm / h (0-20), C-reactive protein (CRP) was 327 mg / L (0-1). Urine dipstick analysis showed 125 leukocytes /HPF. Leukocytes were observed in the microspoic evaluation of the urine. No casts were revealed in the urine evaluation. The chest X-ray showed normal findings.Emprical antibiotic treatment with piperacillin-tazobactam was started with a diagnosis of urinary tract infection due to presence of suprapubic tenderness, fever ,pyuria and elevated CRP levels. Tacrolimus level was 7.3ng / mL. Electrocardiography (ECG) showed atrial fibrillation withhigh ventricular response. Previous ECG had been shown normal sinus rhythm a month ago in his last visit. He was diagnosed with acute atrial fibrillation (AF)(Figure-1). Doppler ultrasonography of kidney showed normal arterial flow in the middle and lower pole of the transplanted kidney, while arterial flow in the upper pole could not be visualized. CT angiography has not been preferred due to acute kidney dysfunction and risk of contrast induced nephropathy. The patient has been undertaken renal scintigraphy .It has been reported the relative decreased concentration of the radionuclid substance given in the upper half of the transplanted kidney. The appearance was consistent with the loss of parenchymal function. It has been shown minimal extension and decrease in the circulation and concentration function of transplanted kidney (Figure-2).Transesophageal echocardiography (TEE) was performed because of AF, nointracardiac cardiac thrombus was observed. Cardiac rhythm did not return to normal sinus rhythm after amiodaroneinfusion ,cardioversion was performed but normal sinus rhythm could not be achieved.Diltiazem 90 mg twice daily tablet was started for heart-rate control. Since the patient's CHA2DS2-VASc score was calculated as 3, anticoagulant therapy (warfarin)was started. The patient's CRP and WBC regressed. CMV-PCR and BK-DNA results were negative. Blood and urine cultures were all negative. The antibiotic treatment was discontinued after seven days. He was evaluated as renal ateroembolic disease due to AF. The patient's creatinine level was 4.5 mg / dL after one week later of the admission. After discharging hospital renal replacement therapy was planned due to estimated glomerular filtration rate calculated by creatinine clearance was 10 ml / min at his visit in July 2018. The patient is admitted to the hemodialysis program three days in a week.

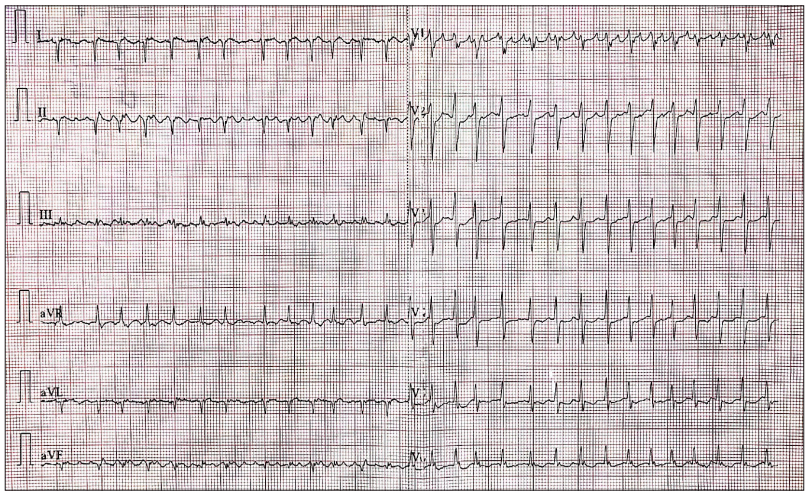

Fig1. Electrocardiogram of the patient illustrates high ventricular response atrial fibrillation.

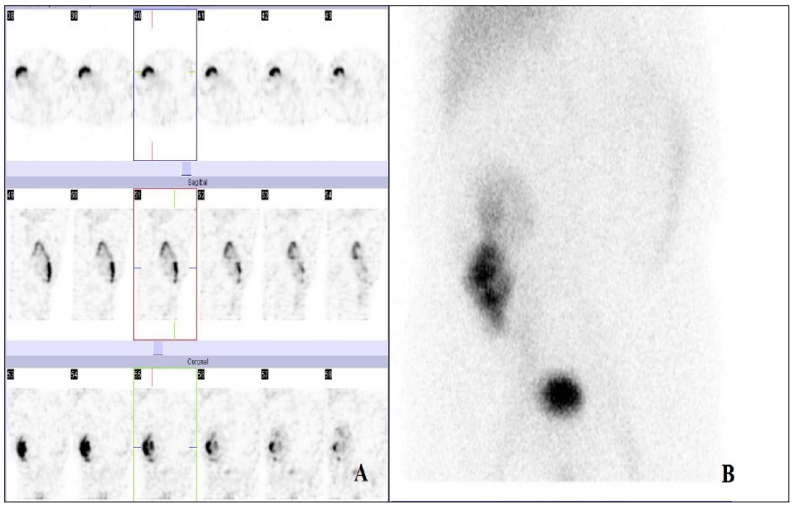

Fig 2. *A) Tc-99m DMSA renal cortical scintigraphy shows the relative decreased concentration of the substance given in the upper half of the transplanted kidney. This appearance is consistent with the loss of parenchymal function. B) The dynamic and static renal scintigraphy performed with Tc-99m MAG3 demonstrates parenchymal loss in the mid-outer segment with minimal extension and decrease in the circulation and concentration function in transplanted kidney.*

Discussion

Renal infarction is a rare condition which occurs by renal artery occlusion [2-8]. Patients usually present with sudden onset flank or abdominal pain. Nausea, vomiting and rarely fever may be among other findings[8-10]. A sudden increase in blood pressure caused by increased renin release might be the another intriguing finding in the clinical presentation [11,12]. Laboratory work up demonstrateshematuria; proteinuria elevated levels of serum creatinine and LDH if renal parenchymal involvementwas occurred [11-15]. It has been suggested that, while serum aminotransferases are either normal or slightly elevated, elevation in serumlactate dehydrogenase (LDH) suggests renal infarction[11,13,16]. LDH enzyme elevation pattern can also be seen in other conditions, which can easily be distinguished from renal infarction, including late myocardial infarction, hemolysis and kidney transplant rejection [16].

Complete blood count, serum creatinine and LDH, complete urinalysis and urine culture, and also ECG should be performed to evaluate atrial fibrillation in patients with systemic embolization risk and symptoms suggestive of renal infarction [3].The first imaging modality, which is generally preferred in patients presenting sudden flank pain, is a contrast-free spiral abdominal computerized tomography [4]. If there is no evidence of stone disease on CT, a contrast-enhanced CT scan should be performed to assess renal infarction. The classic finding is a wedge-shaped perfusion defect. Magnetic resonance imaging (MRI) with gadolinium is an alternative to CT [17]. However, the use of contrast media or gadolinium in patients with significant impairment of renal function may cause further deterioration of renal function or nephrogenic systemic fibrosis. In such cases, radionuclide scintigraphy of kidney could be an option which reveals renal perfusion.

The main causes of renal infarction are; cardioembolic diseases, renal artery injury and hypercoagulable states [2,4,6,9,10]. However, there is also a group of patients whose etiology is not completely clarified. The most common group of cardioembolic diseases is due to arrhythmias particularly atrial fibrillation [9].In a series of 438 patients with renal infarction cardioembolic causes were found in 244 patients (55.7%), and in 211 cases, atrial fibrillation. 33 cases (7.5%) had renal artery injury and 29 cases (6.6%) had hypercoagulable state and no cause was found in 132 patients (30.1%) [9].AF is the most common arrhythmiathatcanleadtocomplicationssuchasstroke, peripheral embolism due to decreased cardiac output and atrial thrombus formation. Affected patients are at high risk for mortality. Hypertension and coronary artery disease are the most common underlying diseases associated with AF in developed countries. Patients were classified as new onset, paroxysmal, permanent, long-term permanent or permanent AF. Physical examination, electrocardiogram, and transthoracic echocardiogram should be performed at the time of diagnosis. Additional laboratory tests

may be required, such as thyroid function tests and ambulatory ECG monitoring. All patients who have an increased risk of embolization are candidates for antithrombotic therapy. Therapy decision is given by using the CHA2DS2-VASc score [18,19].

Our patient was performed by kidney transplantation from living donor after follow-up with chronic kidney disease due to hypertension for ten years. The patient had acute atrial fibrillation due to hypertensive heart disease with the trigger of urinary tract infection. Contrast computed tomography was not preferred as the first choice because of renal dysfunction, thus kidneyscintigraphy was performed. The patient was diagnosed with acute AF and renal infarction by scintigraphy. Anticoagulant therapy was initiated because of high risk of embolism. Renal replacement therapy was started during the follow-up period because of the progression to end-stage renal disease.

There is not enough data about the development of renal infarctionin renal transplant patients. In conclusion; physicians should consider the possibility of transplant kidney infarction associated with arterial embolism in patients presenting with AF and renal dysfunction.

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