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Case Report

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# Acute Renal Infarction and Graft Loss Secondary to Acute Atrial Fibrillation in a Kidney Transplant Patient: A Case Report

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## ABSTRACT

Acute renal artery thromboembolism is a rare clinical condition, and the most common cause is atrial fibrillation. Its' clinical presentation is hypertension and hematuria, accompanied by sudden-onset severe abdominal pain. We aimed to present a case of acute renal allograft thromboembolism and to discuss the diagnostic and therapeutic challenges of this rare clinical entity.

A 68-year-old male patient, who had a living kidney transplant 14 years ago, initially presented with a decrease in urine output, bloody urination, and flank pain lasting for 3 days. On physical examination, widespread tenderness was detected over the transplanted kidney and cardiac beats were arrhythmic. The electrocardiography revealed new-onset atrial fibrillation. In the transplanted kidney, Doppler ultrasound renal artery flow was not observed. Acute renal artery thromboembolism was seen in the renal artery angiography. The patient did not respond to catheter-based heparin and thrombolytic therapy and was accepted as end-stage renal disease.

Although being a rare condition, acute renal infarction should be suspected in a renal transplant recipient with atrial fibrillation presenting with hematuria, acute onset abdominal pain, and acute renal failure.

**Keywords:** Atrial fibrillation, renal artery thromboembolism, renal transplant

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## INTRODUCTION

Acute renal artery thromboembolism is a rare, serious, and challenging clinical diagnosis. It may develop due to in situ thrombosis or a thrombotic embolism from the heart or the aorta. The most common reason is atrial fibrillation (AF).[1] The incidence in hospitalized patients ranges from 0.004% to 0.007%.[2] The majority of patients are in the sixth decade and later in life. It presents with hypertension and hematuria accompanied by sudden-onset severe abdominal or flank pain.[2,3] Since clinical features are not specific, a high clinical suspicion is required for accurate diagnosis in time. Leukocytosis, impaired kidney function, hematuria, and especially lactate dehydrogenase (LDH) increase should draw the attention of physicians. Here, a case of a kidney transplant, who has lost allograft function due to acute renal artery embolism secondary to acute new-onset AF, is presented.

## CASE REPORT

A 68-year-old male presented to our emergency department with complaints of a decrease in urine volume, bloody urine, and flank pain lasting for 3 days. The patient had a kidney transplant abroad from an unrelated living donor 14 years ago due to end-stage renal failure of unknown origin. He was under triple immunosuppressive therapy consisting of prednisolone, tacrolimus, and mycophenolate. The vital signs were as follows: blood pressure was 150/90 mm Hg, body temperature was 36.8°C, respiratory rate was 20/min, and pulse rate was 96/min. In the abdominal examination, widespread tenderness was detected over the

transplanted kidney. There was no circulatory disorder in the end organs and peripheral pulses were clear. Cardiac beats were arrhythmic and the patient was diagnosed as AF by electrocardiography. The patient had not documented any AF previously. Abnormal laboratory tests were as following; urea: 295 mg/dL (normal range (NR): 19-49 mg/dL), creatinine: 11.37 mg/dL (NR: 0.5-0.9 mg/dL), LDH: 1200 U/L (NR: 135-225 U/L), pH 7.1 (NR: 7.35-7.45), bicarbonate: 9 mmol/L (NR: 21-26 mmol/L), white blood cell: 1780 × $10^3$/μL (NR: 3.57-11 × $10^3$/μL). Other results were within normal limits. Urine analysis could not be performed due to anuria. Doppler ultrasonography showed no flow signal in the renal artery of the allograft due to thrombosis. No intracardiac thrombus was detected in transthoracic echocardiography. Renal artery thrombosis secondary to acute AF was suspected. Selective renal artery angiography was performed for the transplanted kidney. No flow was observed in the renal artery. A catheter was placed in this area. After applying bolus heparin (5000 units), tissue plasminogen activator (tPA) infusion was initiated for 24 hours (1 mg/h) 5000 units of heparin were administered intravenously with an interval of 6 hours for three times. A central venous catheter was placed and hemodialysis was performed for 2 hours. Control angiography performed after 24 hours showed that the flow in the renal artery could not be achieved (Figure 1). Treatment was terminated and the catheter was withdrawn. The patient was accepted as end-stage renal failure and was discharged under hemodialysis treatment.



## DISCUSSION

The etiology of renal infarction may show some differences in native and allograft kidneys. While native renal infarction is usually caused by thrombi originating from the heart or aorta, renal artery damage and prothrombotic conditions, and allograft infarction may result from technical error, hypovolemia, antiphospholipid antibodies, high-dose steroid use, long cold ischemia time, delayed graft function, and long-term atherosclerosis.[4,5] On the other hand, infarct pain may be atypical in kidney transplant patients. Allograft denervation may cause pain to be less pronounced than with native kidneys.

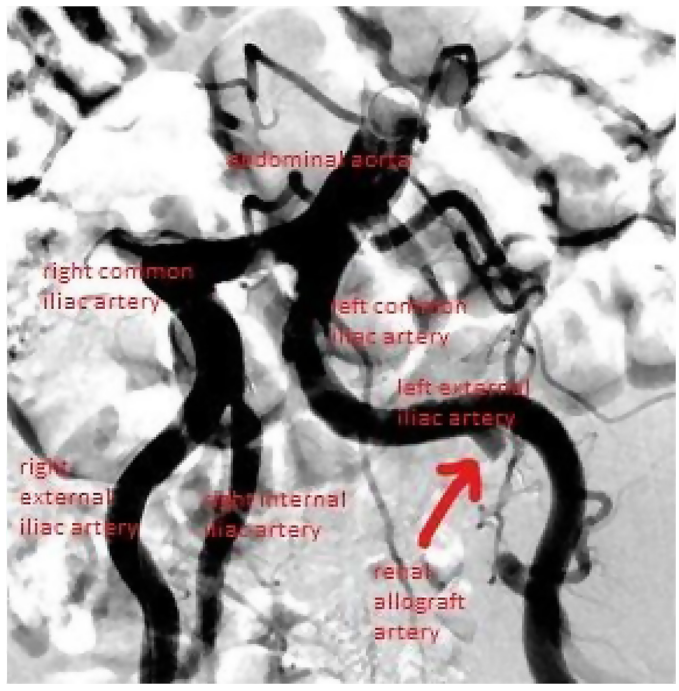


**Figure 1.** Blunt outcome (thromboembolism) of the anastome of the transplanted kidney to the iliac artery in renal angiography.

### MAIN POINTS

- Acute renal artery thromboembolism is a rare clinical condition, and the most common cause is atrial fibrillation.
- Acute renal artery thromboembolism clinical presentation is hypertension and hematuria, accompanied by sudden-onset severe abdominal pain.
- The gold standard diagnostic method for renal artery thromboembolism is renal angiography. However, a carefully performed renal Doppler ultrasonography by an experienced physician can aid in diagnosis.
- In the treatment, heparin, local thrombolytic agents (tissue plasminogen activator, streptokinase, and urokinase), iloprost, and surgical embolectomy can be applied.
- Generally, renal ischemia for more than 3 hours initiates irreversible damage to the kidney. However, rare data show that renal injury can be completely or partially reversed by thrombolytic therapy administered up to 72 hours.

Atrial fibrillation plays the greatest role in the etiopathogenesis of kidney thromboembolism. The incidence of renal artery thrombosis, which is between 0.004% and 0.007% in hospitalized patients,[2] is detected in around 2% of the patients with AF.[6] In small series, acute renal thrombosis is observed to develop principally secondary to AF.[2,3] Atrial fibrillation was detected in 11 of 17 patients infarction.[2] In another report presenting the data of 44 patients with AF and kidney thrombosis; it was observed that 40 of the patients were previously diagnosed with AF, and 4 of them were diagnosed for the first time concurrently with the diagnosis of kidney infarction.[3] When the literature is examined, arterial thromboembolism in the renal allograft is very limited.[7,8] When our case was considered, AF was newly diagnosed as the etiology for renal artery thromboembolism.

The gold standard diagnostic method for renal artery thromboembolism is renal angiography. However, a carefully performed renal Doppler ultrasonography by an experienced physician can aid in diagnosis as seen in our patient. Serum LDH elevation is almost accepted as the rule in kidney infarction.[9] It can be used as a surrogate marker in embolism cases.[10] In our patient, 5-fold increased LDH values were detected at the time of admission.

In the treatment, heparin, local thrombolytic agents (t-PA, streptokinase, and urokinase), iloprost, and surgical embolectomy

can be applied. Generally, renal ischemia for more than 3 hours initiates irreversible damage to the kidney.[3] However, rare data show that renal injury can be completely or partially reversed by thrombolytic therapy administered up to 72 hours.[11] In our case, intraarterial local thrombolytic therapy and heparin infusion were started after 4 hours of admission. However, recanalization was not achieved. We consider that the admission of the patient 3 days after the onset of symptoms prohibited the success of treatment.

This case was aimed to emphasize that AF can lead to renal artery thrombosis in patients with kidney transplantation. Irreversible parenchymal damage may result in end-stage renal failure if the blood flow to the allograft cannot be restored. Early diagnosis and interventional approaches might influence the clinical scenario.

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## REFERENCES

1. Cheng KL, Tseng SS, Tarng DC. Acute renal failure caused by unilateral renal artery thromboembolism. *Nephrol Dial Transplant*. 2003;18(4):833-835. [CrossRef]
2. Domanovits H, Paulis M, Nikfardjam M, et al. Acute renal infarction. Clinical characteristics of 17 patients. *Medicine*. 1999;78(6):386-394. [CrossRef]
3. Hazanov N, Somin M, Attali M, et al. Acute renal embolism. Forty-four cases of renal infarction in patients with atrial fibrillation. *Medicine*. 2004;83(5):292-299. [CrossRef]
4. Akmeşe A, Atabay Y, Ersan S, Taşkıran E, Akar H. Farklı etyolojideki iki renal enfarktüs olgusu. *Istanb Bilim Univ Florence Nightingale Tıp Derg*. 2018;4:22-26.
5. Ponticelli C, Moia M, Montagnino G. Renal allograft thrombosis. *Nephrol Dial Transplant*. 2009;24(5):1388-1393. [CrossRef]
6. Frost L, Engholm G, Johnsen S, Møller H, Henneberg EW, Husted S. Incident thromboembolism in the aorta and the renal, mesenteric, pelvic, and extremity arteries after discharge from the hospital with a diagnosis of atrialfibrillation. *Arch Intern Med*. 2001;161(2):272-276. [CrossRef]
7. Müller-Deile J, Schwarz A, Menne J. Thromboembolism in renal transplant artery due to atrial fibrillation. *Clin Nephrol Case Stud*. 2013;1:10-13. [CrossRef]
8. Akagün T, Artan A, Yazıcı H, Kılıçaslan İ, Bakir B. An acute renal infarction due to atrial fibrillation in a transplant patient: a case report. *Istanb Bilim Univ Florence Nightingale Tıp Derg*. 2015;1(2).
9. Antopolsky M, Simanovsky N, Stalnikowicz R, Salameh S, Hiller N. Renal infarction in the ED: 10-year experience and review of the literature. *Am J Emerg Med*. 2012;30(7):1055-1060. [CrossRef]
10. Winzelberg GG, Hull JD, Agar JW, Rose BD, Pletka PG. Elevation of serum lactatedehydrogenase levels in renal infarction. *JAMA*. 1979;242(3):268-269.
11. Eurvilaichit C, Tirapanich W, Thongborisute E. Renal artery embolism: therapy with intra-arterial streptokinase infusion. *J Med Assoc Thai*. 1999;82(10):978-983.