

# Renal artery embolism on the contralateral kidney after a radical nephrectomy

## Radikal nefrektomi sonrası karşı taraf böbrekte renal arter embolisi

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### ABSTRACT

Renal artery occlusion results commonly from thromboembolism to renal artery from distant vascular system. Hyperhomocysteinemia is one of the hereditary hypercoagulability reasons. In order to elicit etiology in the cases of arterial thrombosis, necessity of hypercoagulopathy screening tests is still controversial. Diagnosis of renal artery thromboembolism should be remembered in the patients who develop postoperative anuria and renal angiographic intervention must be performed immediately for thrombolytic therapy after renovascular imaging. We report a case of a 54-year-old woman with renal artery embolism on the contralateral kidney due to hyperhomocysteinemia after an open left radical nephrectomy.

**Keywords:** Acute renal failure, hyperhomocysteinemia, renal artery embolism

### ÖZ

Renal arter tıkanıklığına, çoğunlukla uzak bölgelerdeki vasküler sistemden renal artere gelen tromboemboli neden olur. Hiperhomosisteinemi, kalıtsal hiperkoagülopati nedenlerinden birisidir. Arteriyel tromboz vakalarında, etiyolojisi ortaya çıkarmak için hiperkoagülopati tarama testlerinin gerekliliği hala tartışmalıdır. Ameliyat sonrası anüri gelişen hastalarda renal arteriyel tromboemboli akla gelmeli ve renovasküler görüntüleme sonrası anjiyografik trombolitik tedavi acilen uygulanmalıdır. Sol açık radikal nefrektomi sonrası karşı böbrekte hiperhomosisteinemiye bağlı renal arter embolisi gelişen 54 yaşında kadın hasta sunmaktayız.

**Anahtar kelimeler:** Akut böbrek yetmezliği, hiperhomosisteinemi, renal arter embolisi

### INTRODUCTION

Renal artery embolism is a rarely seen reason of renal failure and it's diagnosis is difficult. Renal function can be reversible after renal revascularization thanks to early diagnosis and treatment<sup>1</sup>.

There are two causes of acute renal artery occlusion; thrombus and renal artery embolism. Acute thrombosis is defined as a thrombus obstruction caused by renal vascular system. Trauma and instrumentation are the most important resources for the thrombosis. As for thromboembolism, it is known as the most common reason of the renal artery occlusion due to thrombosis originating from the distant vascular areas<sup>2</sup>.

The lack of Antithrombin III, Protein C and Protein S, the resistance of active protein C (the Factor V Leiden polymorphism), Prothrombin 20210A polymorphism, hyperhomocysteinemia, dysplasminogenemia, dysfibrinogenemia, high plasminogen activator inhibitor (PAI) levels (PAI-1 4G/5G polymorphism) and increased factor VIII levels are considered responsible for hereditary hypercoagulability while the presence of antiphospholipid, thrombocytopenia, dysproteinemia, heparin induced thrombocytopenia, estrogen treatment, malignancy, pregnancy, bed rest, operation and trauma result in acquired hypercoagulability<sup>3,4</sup>.

Recently, significant relationship between the development of venous thrombosis and hereditary hyper-

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coagulability has been demonstrated. Besides, local or diffuse atherosclerosis has been associated with arterial thrombosis<sup>5</sup>. Much as some studies have demonstrated a significant role of hypercoagulability in arterial thrombosis, others emphasize the less importance of hypercoagulopathy screening tests in the etiology of arterial thrombosis cases<sup>4,6-8</sup>.

In this paper we present hyperhomocysteinemia induced renal artery embolism case on the contralateral kidney following radical nephrectomy and aimed to discuss it in the light of the current literature.

### CASE PRESENTATION

A 54-year-old white female patient with a left kidney mass found incidentally via ultrasonography was referred to our clinic. Physical examination, serum biochemistry and urine analysis were normal while contrast-enhanced computed tomography scan revealed a 85x63 mm, contrast enhanced, solid mass located on the left kidney upper pole and several lymph nodes with the largest one measured as 11,7 mm in the left paraaortic area (Figure 1). Paraneoplastic syndrome wasn't detected. On the preopera-

tive renal doppler ultrasonography thrombosis was seen only on the left renal vein, which didn't extend to the vena cava inferior.

A left open radical nephrectomy was planned for the patient. Twelve hours before the surgery, 6000 IU low molecular weight heparin was administered subcutaneously to the patient. The left radical nephrectomy with left paraaortic lymph node dissection was performed and the operation took about 110 minutes. While it was detected HBG:11.7 g/dl, HTC:36 and serum creatinine: 1.61 mg/dl on the first postoperatively blood count, creatinine level was 2.79 mg/dl after four hours of surgery. It was initially thought to be analgesic-associated nephropathy. Then, the patient fell into anuria and serum BUN and creatinine levels increased to 5.22 mg/dl and 110 mg/dl after twelve hours of operation, respectively. Since it was thought as acute renal failure, ultrasound scan of abdomen including renal doppler was planned. Doppler examination revealed an embolism in the right

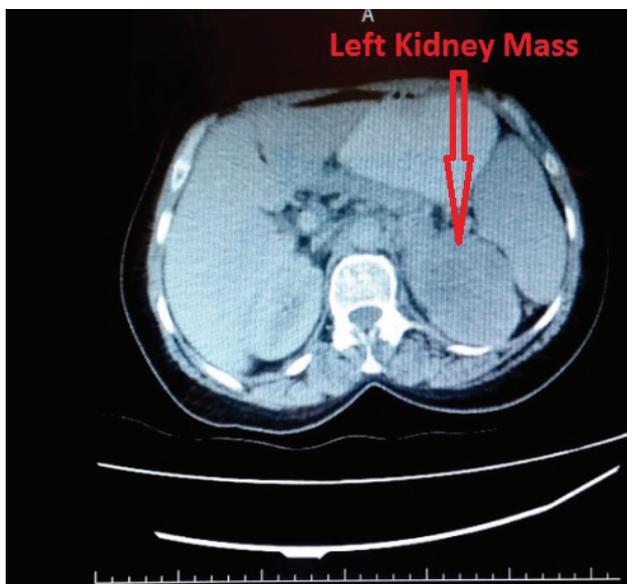


Figure 1. Preoperatively CT image of the patient. CT of the abdomen with intravenous contrast demonstrated a 85x63 mm, well-circumscribed, solid mass in the upper pole region of the left kidney (arrow).

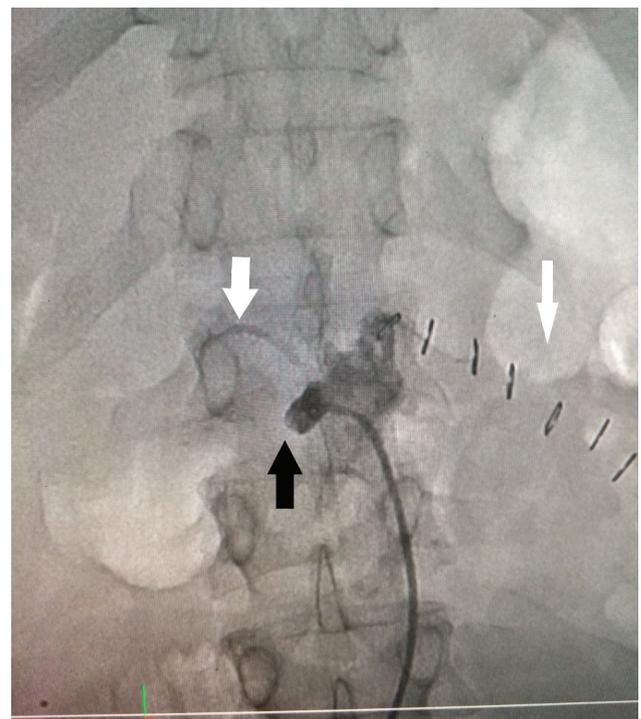


Figure 2. An angiographic image of the abdominal aorta. Angiography revealed total occlusion in the right main renal artery (black arrow). Contrast flow to a branch of abdominal aorta (thick white arrow) and skin staples on the area of the skin incision of left nephrectomy (thin white arrow) can be seen.

renal artery. The patient was referred immediately to another hospital for renal angiographic intervention for thrombolysis however that procedure failed as shown in figure 2. Antithrombin III, antiphospholipid antibody, factor V Leiden polymorphism, protein C, protein S, homocysteine levels were evaluated to determine prone to the thrombosis. The total plasma homocysteine level was high as 128.2  $\mu\text{mol/L}$  (the reference range for the female patient: 3.7-10.4  $\mu\text{mol/L}$ ). Hemodialysis treatment was performed until the kidney transplantation, which was performed 6 months later.

## DISCUSSION

Since acute renal embolism is very rare and highly difficult to diagnose, it can only be detected if there is a clinical suspicion. It may occur in patients with atrial fibrillation, cardiac valve diseases, endocarditis or any embolism history. Clinical presentation may vary from asymptomatic to sudden severe abdominal or lumbar pain<sup>9,10</sup>.

In our case, atrial fibrillation was detected neither during the operation nor in intensive care unit after the early hours of operation. The patient had no history of embolism or cardiovascular diseases. There was no sign of right renal artery infarct symptom and complaint of specific pain. The only factor that led us to make diagnosis was the ratio of BUN/Crea which was compatible with the prerenal acute renal failure. Because of the high serum creatinine level, contrast-enhanced computed tomography scanning could not be carried out and renal artery embolism was revealed by renal doppler ultrasonography.

Although there is no standard treatment for the renal artery embolism, angiographic intervention using either with the thrombolytics alone or with anticoagulant agents can be preferred primarily<sup>11-13</sup>. However, thrombolytic therapy should be started within the first 90-180 minutes when vitality of the kidney tissue is considered to be alive. If the ischemia time takes more than 180 minutes, it will cause irreversible renal parenchymal damage<sup>11</sup>. In contrast, some

studies reported successful revascularization results even after 72 hours and recovery of renal function after some obstructive cases<sup>14,15</sup>.

Blum et al.<sup>11</sup> reported that loss of renal tissue was observed in 30 of 32 surgically treated patients (27 embolectomy, 3 nephrectomy, 2 bypass surgery), however no renal tissue loss was seen in two patients with less than 3 hours of occlusion. Today the use of surgical treatment of renal embolism is very limited because of the high morbidity and limited kidney conservation rates. Under these conditions we chose renal angiographic thrombolytic therapy in this case.

Hyperhomocysteinemia was found the main reason of embolism in our case. Homocysteinemia-disorder of methionin metabolism- is a rare, autosomal recessively inherited disease and linked to an increased incidence of arterial and venous renal thrombosis<sup>17,18</sup>.

Several hypotheses have been suggested about how the high serum homocysteine levels cause thrombosis and atherosclerosis. One of these hypotheses, which is also supported by several experimental studies is that; homocysteine is toxic to the vascular endothelial structure and coagulation mechanisms<sup>19</sup>. Another hypothesis suggests that hyperhomocysteinemia is a sign of abnormal methionin metabolism that effects DNA and cell membrane<sup>20</sup>.

It has been shown that there is a proportional relationship between the high serum homocysteine levels and atherosclerotic coronary heart disease. When the concentration of total homocysteine blood level increases by 5  $\mu\text{mol/L}$ , the cardiovascular disease risk increases about 1.6 to 1.8 fold<sup>21,22</sup>.

As a result, renal artery thromboembolism diagnosis should be remembered in patients who develop postoperative anuria, and it should not be forgotten that post-surgical pain may be clinically misleading in these patients. It is also important to perform the renovascular imaging as early as possible.

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