CASE REPORT

A rare and overlooked mechanical complication of partial nephrectomy: Accelerated hypertension due to renal artery stenosis

Parsiyel nefrektominin gözden kaçan ve nadir görülen mekanik komplikasyonu: Renal arter darlığına bağlı akselere hipertansiyon

Yusuf Ziya Şener, M.D.,[1] Uğur Canpolat, M.D.,[1]
Mustafa Sertaç Yazıcı, M.D.,[2] Enver Atalar, M.D.[1]

[1]Department of Cardiology, Hacettepe University Faculty of Medicine, Ankara, Turkey
[2]Department of Urology, Hacettepe University Faculty of Medicine, Ankara, Turkey

Summary– Secondary hypertension accounts for 5% to 10% of all hypertensive cases, and renal artery stenosis is one of the most common causes of secondary hypertension. Although atherosclerotic vascular disease and fibromuscular dysplasia are the leading causes of renal artery stenosis, there are other, rare etiologies, such as vasculitis and trauma. A partial nephrectomy is the standard of care treatment option for early stage renal carcinoma patients. Traumatic renal artery stenosis can occur during this surgical intervention, though it is a very rare adverse event, and only a few case reports have been reported in the literature. This report is the description of successful percutaneous treatment of accelerated hypertension secondary to traumatic renal artery stenosis after a partial nephrectomy.

Özet– Sekonder hipertansiyon tüm hipertansiyon olgularının %5–10'unu oluşturmaktadır ve renal arter darlığı sekonder hipertansiyonun en sık görülen nedenlerinden biridir. Her ne kadar aterosklerotik damar hastalığı ve fibromüsküler displazi renal arter darlığının en sık nedenleri olsa da, vaskülit ve travma gibi diğer nadir etiyolojik nedenler rol oynayabilir. Erken evre renal karsinom tedavisinde parsiyel nefrektomi standart tedavi yaklaşımıdır, bu cerrahi girişim esnasında travmatik renal arter darlığı gelişebilir. Çok nadir görülen istenmeyen yan etki olsa da, literatürde sadece birkaç olgu sunumu mevcuttur. Bu nedenle, bu yazıda parsiyel nefrektomi sonrası travmatik renal arter darlığına ikincil akselere hipertansiyon gelişen bir hastanın başarılı perkütan tedavisi sunulmuştur.

Although hypertension is called essential or primary in most patients without any identifiable reason, secondary causes should be suspected in cases of new-onset advanced stage (≥stage 2) hypertension, resistant hypertension, unexpected age of the patient (<30 or >50 years old), paroxysmal episodic high blood pressure, or the presence of concomitant hypokalemia or abnormal renal function test results. Renal parenchymal and renovascular etiologies are known as the most common causes of secondary hypertension.[1]

Renal cell carcinoma (RCC) is a prevalent malignancy among adults. Smoking, a positive family history, hypertension, and obesity are well known risk factors for RCC. Patients may present with local symptoms or paraneoplastic syndromes, like hypertension. Surgery is the preferred therapeutic option in early stages. A partial nephrectomy is the standard of care for T1 tumors (tumor size of <7 cm and tumor is localized to kidney).[2] Although there are several potential complications as a result of a partial nephrectomy, traumatic renal artery stenosis (RAS) has only been reported in 1 patient in the literature.[3]

Abbreviations:

CT	*Computed tomography*
DSA	*Digital subtraction angiography*
MR	*Magnetic resonance*
RAS	*Renal artery stenosis*
RCC	*Renal cell carcinoma*
RDS	*Renal artery Doppler ultrasound*

Presently described is the case of a patient in whom secondary hypertension was diagnosed due to traumatic RAS just after a partial nephrectomy performed for stage 1 RCC.

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Correspondence: Dr. Uğur Canpolat. Hacettepe Üniversitesi Tıp Fakültesi, Kardiyoloji Anabilim Dalı, 06100 Ankara, Turkey.
Tel: +90 312 - 305 17 80 e-mail: dru_canpolat@yahoo.com

CASE REPORT

A 52-year-old man who had been in a follow-up for a simple cyst on his left kidney for 6 years was referred to the urology outpatient clinic due to a marked increment in the size of the cyst and the suspicion of malignancy. He had no chronic disease other than medically controlled primary hypertension, for which he had been taking amlodipine 5 mg/day for 2 years. A physical examination was unremarkable, with an office blood pressure of 130/70 mm Hg. Normocytic normochromic anemia was established in a hemogram analysis, and all other laboratory test findings were in the normal reference limits. An abdominal computerized tomography (CT) was performed and a mass lesion 44x40 mm in size was detected at the upper pole of the left kidney. There was no local or distal metastasis observed on abdominal and thoracic CT scans. The patient was diagnosed as stage 1 (T1bN0M0) RCC based on the radiodiagnostic findings. Nephron-sparing surgery was performed, and a pathological examination of the specimen revealed International Society of Urologic Pathology grade I-II clear cell RCC with no capsula invasion and intact surgical margins. The patient was taken into follow-up without further therapy for the RCC. At the post-surgical fifth month, he presented at another hospital with the complaint of headache. His blood pressure was measured as 160/95 mm Hg during the physical examination and ramipril 5 mg/day was added to the daily amlodipine. Transthoracic echocardiography revealed a left ventricular ejection fraction of 60%, interventricular septal thickness of 11 mm, and no valvular pathology. Despite the ramipril + amlodipine combination therapy, his blood pressure level remained high (up to 200/110 mm Hg) and acute kidney injury was observed after 1 week of ramipril administration (serum creatinine level increased to 1.5 mg/dL from a baseline level of 0.9 mg/dL). A consultation with our cardiology outpatient clinic was performed for uncontrolled, accelerated hypertension and acute kidney injury following ramipril therapy. His physical examination was unremarkable, including the absence of a differential blood pressure level between arms and no bruits over the renal artery traces. After the initial assessment, ramipril treatment was discontinued and the antihypertensive therapy was revised to carvedilol 12.5 mg/day + doxazosin 4 mg/day and amlodipine 10 mg/day. At about 10 days after the last medical management strategy, his blood pressure level was 140 to 150/90 mm Hg. Due to the dysregulation of his blood pressure level just after the partial nephrectomy and impaired renal function test results after angiotensin-converting enzyme inhibitor initiation, renal artery Doppler ultrasound (RDUS) was performed. The findings were consistent with reduced left renal perfusion. Renal artery CT angiography confirmed the diagnosis of concentric stenosis at the mid-to-distal part of the left renal artery (Fig. 1a and b, arrow). Percutaneous intervention for left RAS was performed as a result of uncontrolled hypertension and impaired renal function. The left renal artery

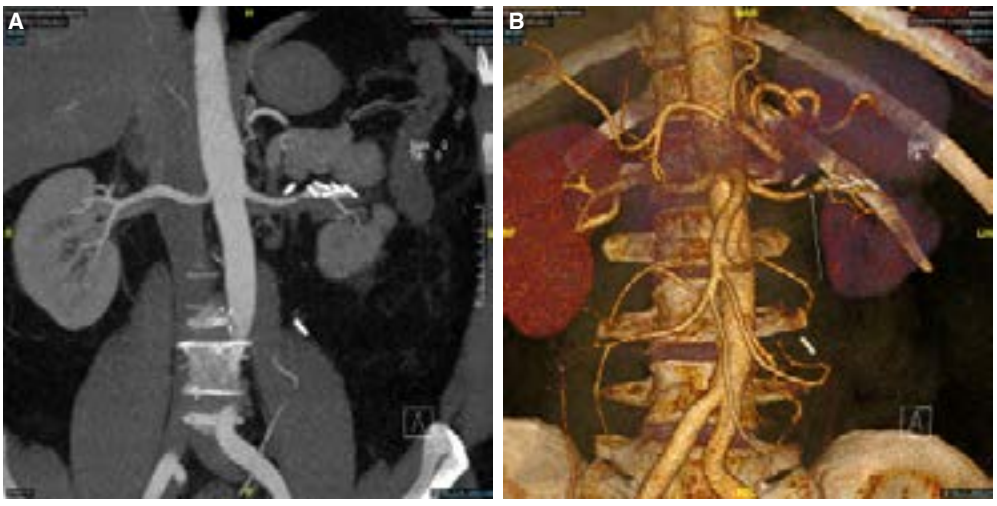

Figure 1. (A, B) Computed tomography angiography showing the stenosis in the mid-to-distal part of the left renal artery.

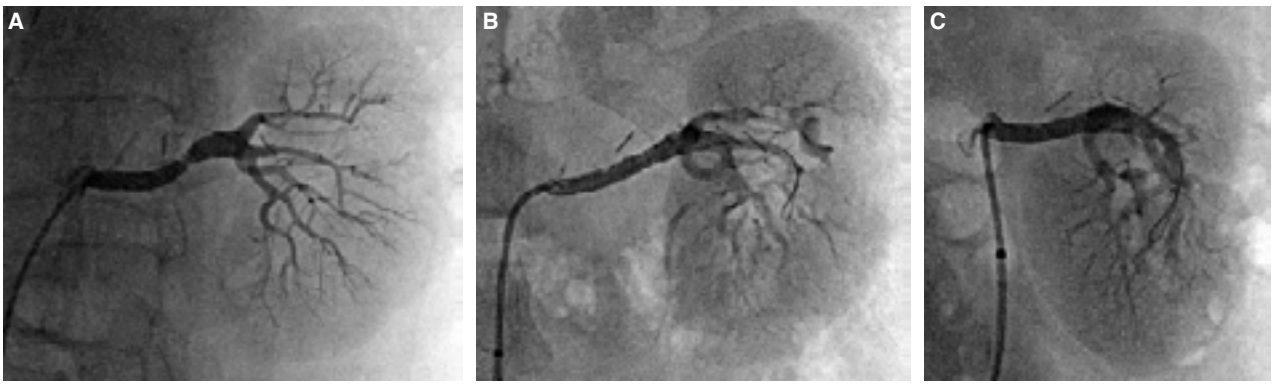

Figure 2. (A) Selective angiography confirmed the diagnosis of critical left renal artery stenosis. **(B, C)** Successful revascularization of the lesion with balloon angioplasty and stent implantation.

was cannulated selectively with a guided catheter and initial angiography revealed 95% stenosis at the mid-to-distal part of the left renal artery (Fig. 2a). The lesion was easily passed using a 0.014-inch floppy guidewire. After predilatation with 4.0x15-mm balloon, a 4.5x15-mm drug-eluting stent was implanted without any residual lesion remaining (Fig. 2b and c). His blood pressure level returned to pre-surgical levels a week after the renal artery intervention. During a 2-week follow-up period, the use of both carvedilol and doxazosin were discontinued and his blood pressure was well controlled with amlodipine (10 mg/day) monotherapy.

DISCUSSION

RAS is known as one of the most common causes of secondary hypertension. Even though atherosclerosis is the leading cause of RAS (90% of cases), other rare conditions, such as fibromuscular dysplasia, vasculitis, and trauma may also be present. RAS should be excluded in patients with flash pulmonary edema, patients with hypokalemia, patients with uremia and unexplained atrophic kidney, resistant hypertension, malignant or accelerated hypertension, and patients with an unusual age at diagnosis of primary hypertension (<30 years or >50 years). RDUS, renal artery CT angiography, magnetic resonance (MR) angiography, and digital subtraction angiography (DSA) are diagnostic options to evaluate both the presence and severity of RAS. Although RDUS is an initial screening test, it has a low sensitivity in obese subjects with lower image quality. Operator dependency is another disadvantage. CT and MR angiography are more sensitive diagnostic tests, however, they should be ordered cautiously in patients with a reduced glomerular filtration rate because of the nephrotoxic contrast media risk with CT angiography and gadolinium-related nephrogenic systemic fibrosis in MR angiography (particularly in patients with a glomerular filtration rate of less than 30 mL/min).[4] DSA is the gold standard diagnostic approach, with the advantage of a potential subsequent percutaneous intervention in the same session in the event of RAS. It should be performed cautiously n patients with a reduced glomerular filtration rate. Although there has been debate regarding the percutaneous treatment of RAS, renal artery revascularization should be preferred in patients with RAS of more than 80%, patients with a high translesional gradient, blood pressure that remains difficult to control with more than 3 antihypertensive drugs, patients with flash pulmonary edema and rapidly deteriorating kidney function, patients with truncal stenosis rather than ostial stenosis, and post-transplant RAS cases.[5]

RCC is the third most common genitourinary cancer and surgery is the main therapeutic option in the early stages of the disease. In a meta-analysis reported by Kim et al.,[6] they found no conclusive difference between early stage RCC patients treated with radical nephrectomy or partial nephrectomy in terms of all-cause mortality or cancer-specific mortality. Urinary fistula, hematoma, acute and chronic kidney disease, renal artery pseudoaneurysm and arteriovenous fistula are well known potential complications of a partial nephrectomy.[7] Although there are no data about the frequency of traumatic RAS after a partial nephrectomy, it may occur as a result of clamping the renal artery during surgery. Goel et al.[3] reported the case

of a 48-year-old woman with new-onset hypertension and impaired renal function tests secondary to RAS just after partial nephrectomy due to early stage RCC. Percutaneous intervention for RAS was successful in restoring kidney function and achieving blood pressure level normalization. A clamp time of more than 15 minutes, clamp force of more than 200 g, and the clamp type, such as the bulldog and Satinsky clamps, have been described as risk factors for endothelium injury and subsequent RAS. In our case, possible risk factors for traumatic RAS occurrence included a clamp time of 15 minutes and a Satinsky clamp used during surgery.

This case has some important clinical implications. New-onset or accelerated hypertension and acute kidney injury in a patient following a partial nephrectomy should bring to mind the possibility of traumatic RAS. Thus, renal artery imaging should be performed to exclude RAS in such patients.

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