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# An Unusual Complication in 3 Cases: Renal Subcapsular Hematoma Following Percutaneous Angioplasty

## Üç Olguda Nadir Bir Komplikasyon: Perkütan Anjioplasti Sonrası Renal Subkapsüler Hematom

**ABSTRACT**

Renal artery stenosis is the leading cause of secondary hypertension. Percutaneous treatment options are safe and effective but can, in rare cases, entail possible complications such as renal subcapsular hematoma. Awareness of such complications will enable better management. Although post-intervention subcapsular hematomas are believed to occur secondary to wire perforation, in this report, we present 3 cases demonstrating reperfusion injury findings rather than wire perforation.

**Keywords:** Balloon angioplasty, percutaneous stenting, renal artery stenosis, reperfusion injury, subcapsular hematoma

**ÖZET**

Renal arter darlığı, sekonder hipertansiyonun önde gelen nedenidir. Perkütan tedavi seçenekleri güvenli ve etkilidir ancak nadiren renal subkapsüler hematom gibi komplikasyonlara neden olabilir. Bu tür komplikasyonların farkındalığı daha iyi bir hasta yönetimi sağlayacaktır. Girişim sonrası subkapsüler hematomların tel perforasyonuna sekonder olarak oluştuğuna inanılsa da bu sunumda tel perforasyonundan ziyade reperfüzyon hasarı bulgusu gösteren üç olgu prezente ediyoruz.

**Anahtar Kelimeler:** Balon anjiyoplasti, perkütan stentleme, renal arter stenozu, reperfüzyon hasarı, subkapsüler hematom.

**CASE REPORT**
OLGU SUNUMU

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Renal artery stenosis (RAS) is the most common cause of secondary hypertension and may be caused by several pathological processes such as atherosclerosis, fibromuscular dysplasia, and vasculitides.[1] Diagnosis of RAS relies on a variety of radiological imaging techniques.[2] Renal artery stenosis can be treated by percutaneous transluminal balloon angioplasty or renal artery stenting.[3] Complications of these procedures include the formation of renal artery thrombus/embolus, dissection or rupture, and aortic dissection at the renal artery level.[3] A rare complication of renal artery intervention is bleeding in the perirenal, renal subcapsular, or retroperitoneal spaces.[4] Among these, subcapsular hematoma is an even rarer complication. Herein, we present 3 cases of subcapsular hematoma, a highly rare complication that followed percutaneous transluminal renal artery interventions including balloon and stenting. The first impression regarding the etiology of the complication was wire perforation; however, immediate flat detector CT findings revealed the possibility of reperfusion injury. Multisite self-limiting extravasations in digital subtraction angiography (DSA) and CT imaging were the most characteristic findings supporting the diagnosis.

### Case 1

A 42-year-old woman was referred for endovascular treatment because of RAS. The patient had hypertension that could not be controlled with medications for 2 years. The blood pressure was measured to be 160/95 mmHg despite multidrug therapy. Computed tomography angiography (CTA) revealed left RAS. The hemoglobin level was 11.2 g/dL before the intervention. For access to the arterial system, a puncture of

the right common femoral artery was performed and a 6F 45 cm long introducer sheath was placed at the origin of the left renal artery. The left renal artery was catheterized with a 5F Cobra catheter through the long sheath. Digital subtraction angiography demonstrated severe narrowing of the left renal artery at the ostial level with post-stenotic dilation (Figure 1A). The narrow segment of the left renal artery was passed through with a 0.018-inch wire. Next, primary stenting was performed over the wire with a 6 × 15 mm balloon-expandable stent. Follow-up angiography showed proper stent positioning and complete expansion without any sign of bleeding or peripheral embolization (Figure 1B). Approximately 20 minutes after the procedure, the patient suddenly complained of left flank pain. An abdominal ultrasound showed a 5 × 8 centimeter-sized hypoechoic collection compatible with subcapsular hematoma. Complete blood count data revealed a decreased hemoglobin level of 10.4 g/dL. Digital subtraction angiography revealed multiple peripheral microaneurysms without any observable active extravasation (Figure 1C). A cone-beam CT was performed at the angiography table to understand the precise etiology of the extravasation. Computed tomography showed a sizable left subcapsular hematoma displacing the renal cortex from the capsule (Figure 1D). The final diagnosis was reperfusion injury due to the existence of multiple tiny extravasation sites. There was no active bleeding that required intervention, only the need for follow-up. The patient was discharged from the hospital after 2 days with decreased blood pressure levels. A triphasic CT scan was obtained on the seventh day after the procedure, which demonstrated a minimal decrease in a subcapsular hematoma. A blood sample was taken, showing a stable hemoglobin level (11 g/dL). The patient had blood pressure within normal limits and no further complaints of pain in the flank.

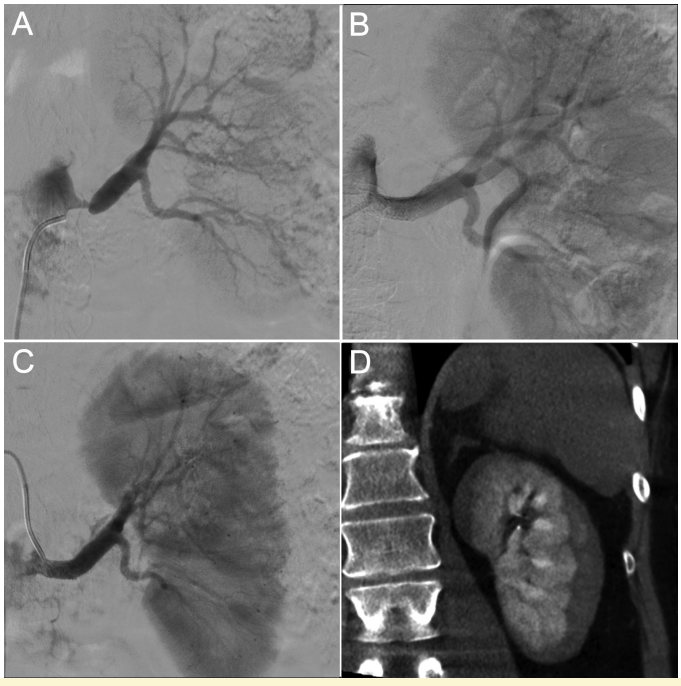


**Figure 1. (A) Digital subtraction angiographic image of left renal artery before the procedure showed severe ostial narrowing that required stenting. The branches of the left renal artery and contrast enhancement of the parenchyma were normal. (B) Digital subtraction angiographic image of the left renal artery after transluminal angioplasty revealed normal renal artery caliber. (C) Digital subtraction angiographic image of the left renal artery was taken 40 minutes after the procedure upon the patient's complaint of flank pain. The angiography showed multiple microaneurysms especially at the distal renal artery branches and the presence of a slightly compressed renal parenchyma along the long axis on the coronal view. (D) Cone-beam tomography, carried out to reveal the cause of parenchymal compression, showed the presence of subcapsular hematoma with compressed and displaced left renal parenchyma.**

**Case 2**

A 68-year-old female patient presented with chronic superior mesenteric artery (SMA) occlusion and severe left RAS detected with CTA. The patient was admitted to the angiography unit for SMA recanalization and renal artery stenting. The left brachial artery was accessed using a 5F vascular sheath to be able to access the occluded SMA in an anterograde manner. Digital subtraction angiography of the left subclavian artery revealed total occlusion of the proximal segment that did not allow the passage of the catheter and wire in a retrograde manner. Next, a right femoral puncture was carried out and the subclavian artery was revascularized with a balloon-expandable stent by a bilateral approach. Next, a left brachial 6F 90 cm long introducer sheath was inserted. Arteriography of the abdominal aorta showed total occlusion of the SMA and stenosis of the left proximal renal artery. Following the passage of the occluded segment of SMA with the diagnostic catheter and wire, angioplasty and implantation of a 6 × 40 mm self-expandable stent was performed. After catheterizing the stenosed left renal artery (Figure 2A), a 6 × 17 mm stent was placed over a 0.018-inch wire. Left renal artery angiography revealed normal findings (Figure 2B). Forty minutes after the procedure, the patient complained of left plank pain. Angiography was performed immediately to evaluate any probable vascular complications. Selective left renal arteriography showed no extravasation of the contrast media from the main renal artery or branches. Microaneurysms and tiny areas of contrast extravasations were observed. The final diagnosis was reperfusion injury rather than guide-wire perforation owing to the presence of multiple foci of extravasation of contrast media, irrelevant to distal migration of the guide-wire. A decision for follow-up was made as the multiple tiny extravasation sites required no further intervention. Cone-beam CT revealed similar findings (Figure 2C). The hemoglobin level was 8.3 g/dL which was 2 units lower than the level at admission; therefore, one pint of packed red blood cell (RBC) was transfused. After 2 days of hospitalization, the patient was discharged without any further complaints. Ten days later, a follow-up contrast-enhanced CT scan demonstrated that the subcapsular hematoma was still present, but was decreased in size (Figure 2D). The patient was asymptomatic.

**Case 3**

A 25-year-old female patient with a diagnosis of Takayasu arteritis was admitted to our hospital for endovascular treatment

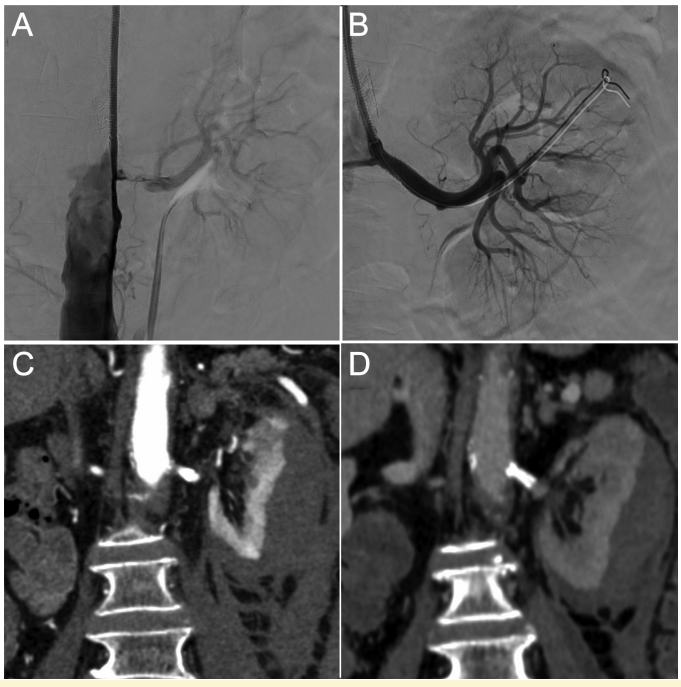


**Figure 2. (A) Digital subtraction angiographic image of left renal artery reached in an anterograde manner showed the presence of ostial narrowing. (B) Digital subtraction angiographic image of the left renal artery after transluminal angioplasty revealed normal renal artery caliber. The branches of the left renal artery and contrast enhancement of the parenchyma were normal. (C) Computed tomography of the patient was taken 7 hours after the procedure due to a complaint of flank pain. Computed tomography showed left renal subcapsular hematoma compressing the renal parenchyma along the long axis on the coronal view. (D) A computed tomography image obtained 10 days after the procedure showed minimal regression of the left renal subcapsular hematoma.**

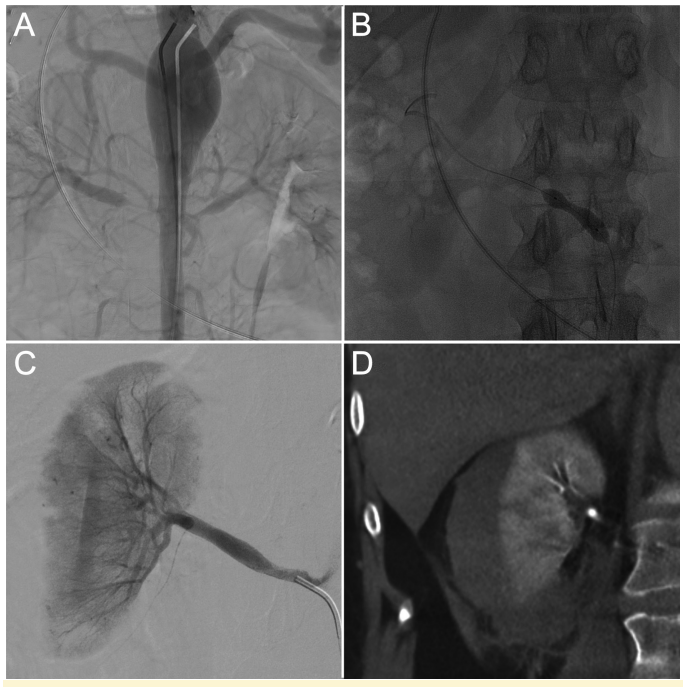


**Figure 3. (A) Digital subtraction angiographic image of the aorta and bilateral renal artery showed the presence of bilateral renal artery stenosis, aortic narrowing, and dilation. (B) Angiographic image of the right renal artery during the balloon angioplasty procedure. (C) Digital subtraction angiographic image of the right renal artery after transluminal balloon angioplasty revealed normal renal artery caliber. Multisite microextravasations were also noted. (D) A computed tomography image obtained after 1.5 hours after the procedure showed the presence of extensive right renal subcapsular hematoma, compressing and displacing the renal parenchyma.**

of severe stenoses of the descending aorta and bilateral renal arteries. The patient was under anti-inflammatory treatment and acute phase reactants were within normal limits, suggesting a non-active phase of the disease. Digital subtraction angiography revealed severe bilateral renal stenosis and descending aortic stenosis with post-stenotic dilation (Figure 3A). A 6F 45 cm long introducer sheath was passed through the right femoral artery. Balloon angioplasty of the aorta with a 16 mm × 40 mm balloon was carried out, followed by balloon dilation of both renal arteries. Stenotic segments were dilated with 5 × 40 mm balloon catheters over a 0.018-inch wire (Figure 3B). One hour later, the patient complained of sudden onset right flank pain. Transabdominal ultrasound revealed the presence of a 6 × 3 cm renal subcapsular hematoma. Angiography revealed multiple microaneurysms and cone-beam CT showed that the subcapsular hematoma was extended and was compressing the renal parenchyma. However, no specific vascular injury could be identified (Figure 3C, D). We were aware that the microwire was sent deeply into the capsule in the previous angiographic procedure; however, the location of the extravasation areas appeared to be unrelated to the localization of the wire. This suggested the presence of reperfusion injury, rather than wire perforation. The general condition of the patient stabilized on the day following the intervention, and the patient was discharged without any symptom.

## Discussion

Percutaneous transluminal renal artery angioplasty is regarded as a safe and effective procedure for the treatment of RAS.[5] Among the complications of the procedure, subcapsular hematoma is extremely rare but should be considered in the appropriate clinical context. Subcapsular hematoma was reported to be secondary to reperfusion injury, similar to cerebral hyperperfusion syndrome.[6] Cerebral hyperperfusion syndrome is caused by impaired automatic regulation of cerebral blood flow. Severe carotid stenosis can cause a chronic low-flow state that may result in a compensatory dilation of cerebral vessels as part of the normal auto-regulatory response to maintain sufficient cerebral blood flow. Thus, the vessels lose their ability to regulate vascular resistance in response to changes in blood pressure, ending up with increased cerebral blood flow after recanalization.[7] The mechanism of subcapsular hematoma is similar and has been explained as a transient hyperperfusion of the kidney tissue after

revascularization. Similar to cerebral loss of autoregulation, the long-lasting RAS and hypoperfusion cause compensatory vascular dilation resulting in impaired auto-regulation.[4] Hyperperfused distal renal artery vessels may rupture and result in hemorrhage.[4] Although it is not possible to exclude iatrogenic microvessel perforation, the lack of active extravasation, a single pseudoaneurysm or arteriovenous (AV) fistula, along with the presence of multisite microextravasations support the idea of reperfusion injury. A previous study has reported a decrease in renal flow following the correction of a dissecting aortic aneurysm, leading to renal subcapsular hematoma with no renal intervention. This report also supports the theory of reperfusion injury.[8] Kyung et al[6] have reported a similar case of renal subcapsular hematoma following renal artery stenting that was secondary to reperfusion injury and regressed conservatively. Also corroborating the current study, Pastroma et al[9] reported a case of renal subcapsular hematoma due to the reperfusion injury, as shown by the multiple foci of extravasation of contrast media irrelevant to guidewire distal migration. The hematoma was observed during renal artery balloon angioplasty performed to provide revascularization of an occluded renal artery stent.

Multiple microaneurysms developed under hyperperfused conditions can contribute to further hemorrhage, as seen in the current cases. Previous studies have reported renal subcapsular hematoma after percutaneous renal artery stenting; however, the current study is the first to report reperfusion injury following sole renal artery balloon angioplasty for RAS.

In conclusion, the cases described in the current study highlight the possibility of reperfusion injury to the kidney after percutaneous transluminal renal artery angioplasty. Sudden onset flank pain and decreased hemoglobin levels following the intervention, combined with supportive DSA findings showing the presence of multiple tiny microaneurysms should raise the suspicion for subcapsular hematoma resulting from reperfusion injury. Multiple bleeding sites noted at the upper pole, mid zone and lower pole of renal parenchyma may be attributed to reperfusion injury as the guide wire did not approach some of these areas. Long-standing severe RAS, as in our cases, may form a basis for the development of subcapsular hematoma.

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