Successful Treatment of Resistant Hypertension Associated Ascites in a Renal Transplant Patient

Özür

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Renovasküler Hipertansiyon; Renal Arter Darlığı; Balon Dilatasyonu

Abstract
Transplante renal artery stenosis (TRAS) is defined as renal artery diameter reduction of more than 50%, which causes a reduction in glomerular filtration rate and a rise in plasma creatinine concentration. Current treatments are endovascular-balloon-angioplasty and stent implantation. We present the case with renal artery stenosis and ascites treated with endovascular-balloon-dilatation. A 52-year-old female patient who was admitted to the nephrology clinic five months after the transplantation with blood pressure: 180/110 mmHg, ascites and chest radiograph did not show a pulmonary oedema. An occlusion of 80 % in a segment and the stenosis was reduced to 10 % by endovascular-balloon dilatation. Following endovascular-balloon dilatation, arterial blood pressure were normal and no ascites. Endovascular balloon dilatation is minimally invasive method that is also successful, contemporary and valid procedures with easy applicability for the management of TRAS.

Keywords
Renovascular Hypertension; Renal Artery Stenosis; Balloon Dilation
Introduction
Renal artery stenosis (RAS) is defined as renal artery diameter reduction of more than 50%, and accounts for 10% of all cases of hypertension [6]. RAS leads to a critical decline in renal perfusion pressure and results in hyperperfusion in renal parenchyma, and activation of the renin-angiotensin-aldosterone system (RAAS). It causes a reduction in glomerular filtration rate (GFR) and a rise in plasma creatinine concentration (>1.5 g/dl) [1]. The prevalence of transplant renal artery stenosis (TRAS) currently increases along with the raise in rate of renal transplantations. TRAS is a vascular complication that most frequently occurs within the first 6 months after kidney transplantation. Stenoses most commonly occur at the anastomotic site, proximal or distal to the anastomosis, and may happen due to twisting. It is among the significant causes of graft dysfunction or loss in transplant recipients [2]. TRAS may also lead to hypervolemia (oedema, ascites), refractory hypertension, and Pickering syndrome known as flash pulmonary oedema [9]. Although conventional angiography is the gold standard, doppler ultrasound is a commonly preferred tool for early diagnosis [3, 4, 5]. Early diagnosis and treatment of TRAS is important for prevention of graft damage and the systemic effects of hypertension. As a consequence of the developments in interventional radiology, operations including endovascular-balloon-angioplasty and stent implantation are more commonly performed treatment modalities [4,5,6,7,8].

Herein, we report an interesting case with transplant renovascular hypertension and ascites, both problems of whom resolved with a successful intervention of renal angioplasty.

Case Report
A 52-year-old female patient receiving hemodialysis three times a week for 7 years due to polycystic kidney disease was performed a cadaveric renal transplantation on August 23, 2015. The patient was admitted to the nephrology clinic five months after the transplantation with complaints of headache and abdominal distention. Physical examination revealed blood pressure: 180/110 mmHg, heart rate: 92 /min, body temperature: 36.7oC, ascites in abdomen, and bilateral 1+ pretibial oedema. Pulmonary examination was normal, and the chest radiograph did not show signs of pulmonary oedema. Antihypertensive therapy was gradually regulated as carvedilol 2x12.5 mg/d, furosemide 80 mg/d, indapamide 2.5 mg/d and lercanidipine 20mg/d. Immunosuppressive therapy included mycophenolate mofetil (2 gr/d), tacrolimus (8 mg/d), and prednisolone (5 mg/d). Laboratory results were as follows; total blood count: normal, BUN: 29.3 mg/dL, creatinine: 1.69 mg/dL, Na: 137 mmol/L, K: 3.7 mmol/L, urinalysis: urine density: 1.019, protein: -, leukocyte: 2, erythrocyte: 9, C-reactive protein: 25.2mg/dL, erythrocyte sedimentation rate: 8 mm/h. Other laboratory tests were within normal limits. Portal doppler ultrasound performed in order to enlighten ascites aetiology revealed cysts in the spleen and liver, and normal diameter (1.8 cm) and flow of the portal vein. Blood and urine cultures and the culture from peritoneal fluid were negative. The acid-fast stain of the ascites fluid with transudate features was negative, and the cytologic examination of the fluid was benign. PCR for BK virus was negative. Decoy cells were negative in urine cytology. The patient was anergic for PPD.

The ultrasound revealed multiple septated cysts of 15x18 mm in the right and 31x35mm in the left native kidneys. In the right iliac fossa, infero-anterior to the transplant kidney, an anechoic image of 96x45x40 mm in size with irregular margins was observed. There was no hydro nephrosis, a urine leak or renal rupture. Creatinine in ascites fluid was normaly. Also the ultrasound revealed free fluid with a maximum depth of about 7 cm (ascites ?) in the perihepatic region, around intestinal loops, the pelvic area and prominent stenosis in the anastomotic site of the renal artery. (Image1)

Contrast-enhanced MR angiography showed high degree narrowing at the anastomotic site of the iliac branch of renal artery. An occlusion of 80 % in a segment of 1 cm at the end to side anastomosis area of the right iliac artery was detected by renal angiography performed by interventional radiology. The stenosis was reduced to 10 % with endovascular-balloon dilation. (Image 2)

Following the balloon dilation arterial blood pressure turned to normal limits, and antihypertensive treatment was discontinued. In patient’s follow up two weeks after the operation, complaint of abdominal distention had dissolved, blood pressure was within normal limits, and no ascites was observed on ultrasound.

Discussion
While RAS is defined as the narrowing of the renal artery of more than 50%, even stenosis greater than 50% may occasionally remain asymptomatic. However, the narrowing of more than 70% is generally symptomatic, and leads to refractory hypertension [7]. Our patient had resistant hypertension and ascites...
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How can we explain the existence of severe ascites without pulmonary oedema? We considered that ascites formation in the abdomen may have been facilitated by supervening RAS on grounds of pre-existing compensated portal hypertension due to liver cysts. Consequently, endovascular balloon dilation and stent placement are minimally invasive methods that are also successful, contemporary and valid procedures with easy applicability for the management of TRAS.

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Competing interests
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References

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