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Case Report

Missed Renal Infarction Presenting as the Nephrotic Syndrome: a Case Report

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ABSTRACT. Aortic dissection may be associated with renal disease. The presentation, especially in the later stages of the process, includes proteinuria, hematuria and impairment of renal function. Thus the clinical picture may be confused with glomerulonephritis or hypertension. . We present a case of ischemic nephropathy resulting from involvement of the right kidney by an aortic dissection. The pateint presented with the nephrotic syndrome some two and a half months after the probable time when the aortic disection had occurred. At that time the initial back and flank pains had disappeared. Ultrasound examination revealed a smaller right kidney, compared to the left one. Imaging techneques, initaited for suspected renal artery stenosis, revealed aortic dissection involving the right renal artery starting from the descending aorta, distal to the origin of the left subclavian artery and extending down to the right common iliac artery; occluding the right renal artery. The medical literature is reviewed for patients presenting with ischemic nephropathy and the mechanisms of proteinuria discussed. We conclude that ischemic nephropathy can clinically mimic glomerulonephritis and can be missed if it is not included in the differential diagnosis of patients who present with heavy proteinuria and hypertension.

Key words: Ischemic nephropathy, Dissection, Hypertension, Proteinuria.

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Introduction

Occlusion of the renal artery leads to the development of hypertension and in many cases proteinuria.[1-4] This may confuse the attending physician as to the cause of

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hypertension. We here present a case of occlusion of the right renal artery, as a result of aortic dissection, presenting as hypertension, hematuria, nephrotic range proteinuria and renal impairment.

Case Report

41-year-old Sudanese male with no significant past medical history presented to a local hospital complaining of severe interscapular back pain along with right abdominal and left flank pain. He was found to be hypertensive, had right lower abdominal and bilateral flank tenderness.

The initial investigations revealed raised serum creatinine of 209 μmol/L (2.38 mg/dl) and mildly raised liver enzymes. Urinalysis showed RBCs 15-25/HPF, WBCs of 3-5/HPF and Protein dipstick +3. A 24-hour urine protein excretion was 1.84 grams. Ultrasound of the abdomen showed normal liver and kidney echotexture; however size of the right kidney was 9.2 x 4.4 cm and left 10.8 x 4.8 cm. Intravenous pyelogram was reported as normal.

The patient's blood pressure peaked initially to 210 systolic and diastolic of 110 mm Hg and was treated with a calcium channel blocker (Amlodipine). Two and half months after initial presentation, he was referred to our hospital for evaluation of glomerulonephritis and was seen in the primary care clinic.

Repeated laboratory work-up revealed serum creatinine of 206 μmol/L (2.34 mg/dl). 24-hour urine protein excretion was 4.0 gram and urinalysis showed significant hematuria with RBC of 70/HPF, and WBC 4-8/HPF. Investigations for secondary causes of glomerulonephritis were negative. The patient was referred to the nephrology clinic for hypertension, renal impairment and proteinuria. He did not have any abdominal pain or back pain and was admitted for renal biopsy. Ultrasound revealed a small right kidney that was 7.4 x 3.0 cm and left kidney 11.0 x 5.0 cm. Radionuclide renal scan showed non-functioning right kidney.

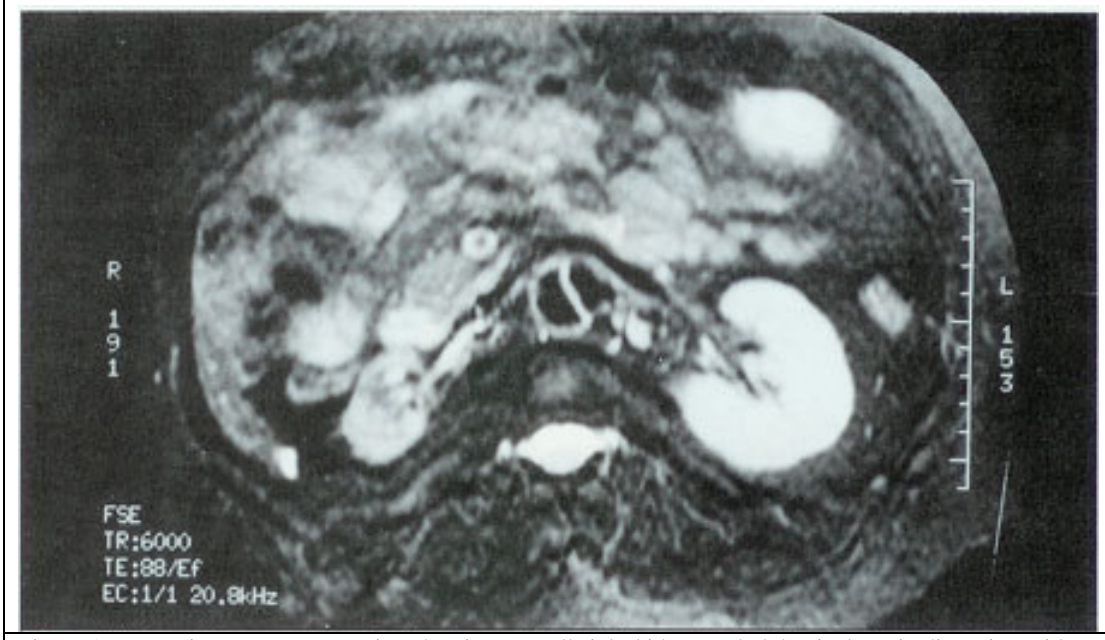

Figure 1. Magnetic Resonance Imaging showing a small right kidney and abdominal aortic dissection with medially

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located large false lumen occluding the right renal artery.

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The blood pressure was difficult to control with the atenolol 100 mg/day, amlodipine 10 mg/day and the diueretic, furosemide. 24-hour urine protein excretion remained 4.5 grams even with the control of blood pressure to < 160/95 mm Hg.

Magnetic Resonance Imaging (MRI) was requested to evaluate the presence of renal artery stenosis as a cause of hypertension. MRI revealed aortic dissection involving the right renal artery (Figure 1). A computed tomography (CT) scan also corroborated the presence of aortic dissection extending from the descending aorta distal to the origin of the left subclavian artery and extending to the right common iliac artery; occluding the right renal artery, Figure 2. Angiogram confirmed the CT scan findings and showed leak in the thoracic part of the dissection (Figure 3 a, b).

The patient's blood pressure was eventually controlled with the vasodilator, minoxidil and atenolol. He underwent graft replacement of the thoracic part of the dissection at another center and tolerated the surgery well.

On follow-up two months later, the blood pressure was controlled with atenolol and amlodipine. We decided not to intervene to correct the right renal artery dissection, since blood pressure was well controlled and renal function was stable (serum creatinine of 208 μmol/L (2.36 mg/dl) and proteinuria was +1.

Discussion

Though it could be due to hypertension, the marked proteinuria in our patient was probably due to ischemic nephropathy. Poutasse in 1957 and others later, noted proteinuria with renal artery stenosis.[1-3]

Kumar and Shapiro[4] presented in 1980 three cases that had significant proteinuria of 1.0 to 12.9 grams per day secondary to renal artery stenosis. All three patients underwent nephrectomy of the stenosed side along with renal vein renin measurements. Proteinuria disappeared in two (follow-up one and 10 years) and in the third dropped from 9.0 grams to 1.0 gram after one month. They also documented drop in the level of renal vein renin levels after surgery.

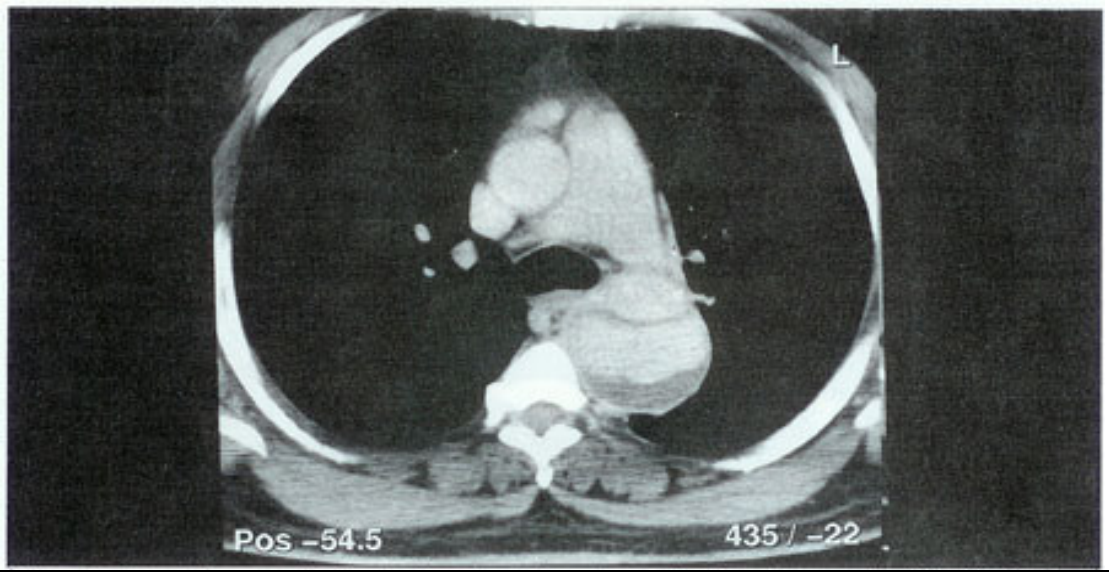

Figure 2. Computed tomography-showing dissection of thoracic aorta. True lumen is antero-laterally while the large false lumen is surrounding the inner 2/3rd residual hematoma also seen posterior to the false lumen.

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Zimbler et al[5] reported their findings in 46 patients with renal artery stenosis. Cure of hypertension was noted with remission of proteinuria post angioplasty. They also noted that proteinuria was massive if the renal artery was completely occluded.

The pathophysiology of proteinuria induced by ischemia has been elucidated in animal models and humans.

Eisenbach et al[6] and Bohrer et al[7] infused renin and angiotensin II (AgII), respectively, and induced reversible proteinuria in the experimental animals. More recently, Miller et al[8] and Johnson et al[9] illustrated the development of focal segmental glomerulosclerosis and changes in the podocytes in rats after the infusion of AgII for 8 and 2 weeks, respectively.

Kimura et al[10] studied patients with unilateral renal artery stenosis by catheterizing the ureters. They calculated glomerular capillary pressure and glomerular filtration rate using Para-amino hippurate and inulin infusions. Glomerular hypertension and hyperfiltration were noted in the non-stenosed kidney.

Alkhunaizi and Chapman,[11] recently reported a patient with unilateral renal artery stenosis who underwent renal biopsy of both kidneys. The ischemic kidney had normal glomeruli with hyperplasia of the juxtaglomerular apparatus and the contralateral kidney revealed focal segmental glomerulosclerosis (FSGS). Thus, the evidence suggests that it is probably the high AgII level that leads to glomerular proteinuria. The surge of AgII from the ischemic kidney may cause traction and detachment of podocytes from the basement membrane in the contralateral kidney forming a nidus of sclerosis that results in FSGS.[12] The high AgII level also affects efferent arteriole in the ischemic kidney that may exert physical pressure on the podocytes and may lead to sclerosis causing proteinuria.[13]

We believe that our patient had significant proteinuria due to ischemic nephropathy although renal biopsy was not done. Post operation follow-up of the patient showed stable mild impairment of renal function, easily controlled blood pressure with only two antihypertensive drugs, and diminution of proteinuria.

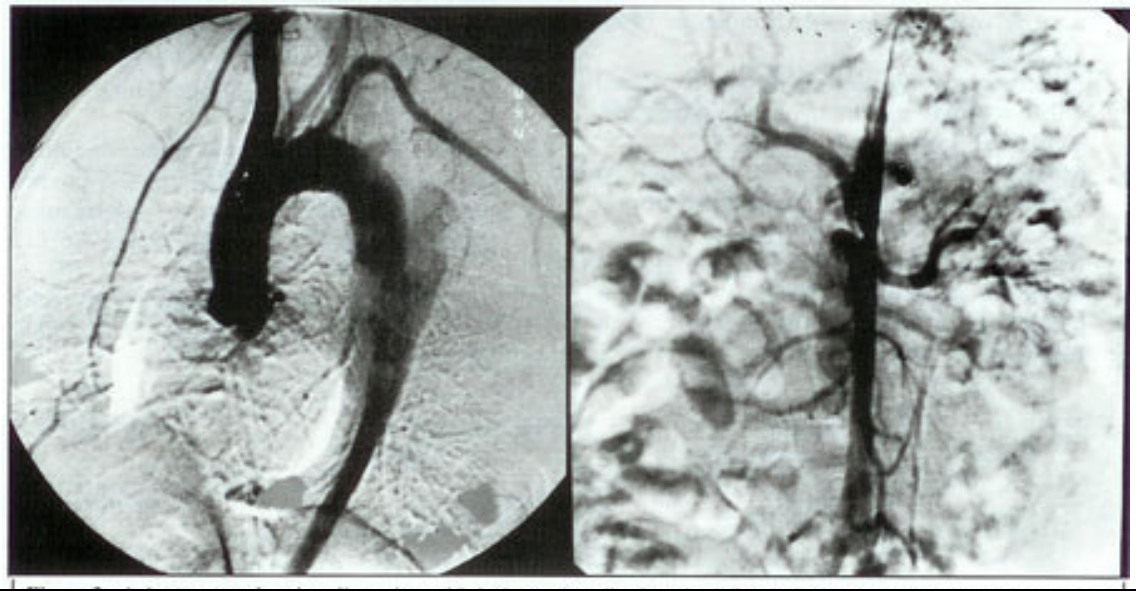

Figure 3. a) Aortogram showing dissection with leak starting distal to the left subclavian artery origin, b) Right renal artery is not visualized and dissection extends to the right common iliac artery.

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Another interesting observation in our patient was the decrease in renal size from 9.2 cm to 7.4 cm in a mater of two and a half months. Strandness et al[14] have shown, in their prospective follow-up of 54 patients with documented renal artery stenosis, a decrease in renal size by more than one centimeter in three (5%) patients by six months and in 14 (27%) patients by two years. They also found that in the ischemic kidney the size decreased more quickly with complete occlusion, severely elevated blood pressure and in the presence of bilateral ischemia. Our patient had complete occlusion of the right renal artery and his blood pressure was not well controlled initially, which most likely lead to the rapid decrease in the renal size. We conclude that ischemic nephropathy can clinically mimic glomerulonephritis and it can be missed if it is not included in the differential diagnosis of patients who present with heavy proteinuria.

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