CLINICAL VIGNETTE

Clinical Diagnosis Renal Artery Stenosis in a 65-Year-Old Male

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Case

A 65-year-old male was referred for evaluation of elevated creatinine and proteinuria. His past medical history included hypertension, atrial fibrillation, non-obstructive coronary artery disease, recurrent congestive heart failure. He first learned of proteinuria about 6 years prior. He also reported that his blood pressure was higher than usual the preceding couple of months. Physical exam included blood pressure of 154/78. BMI 35.4 kg/m^2 and trace pedal edema. There was no abdominal bruit and the remainder of the physical examination was unremarkable. His primary care physician started lisinopril 20 mg one week prior to his Nephrology appointment.

Lab data from one week prior serum creatinine of 1.36 mg/dl (reference range 0.60-1.30 mg /dl) and an estimate glomerular filtration rate (eGFR) of 55. Serum Potassium was normal at 4.2. Complete blood count was normal. Dipstick urine showed 2+ protein with clear urine microcopy. A spot urine protein-creatinine ratio was elevated at1.3g (reference range 0.0-0.4 g). Urine microalbumin-creatinine ratio was 895 mcg/mg, indicating that the proteinuria was predominantly albumin. Secondary proteinuria testing was negative, including Antinuclear antibodies, Anti double stranded DNA, and ANCA titers. Serum immunofixation and urine immunofixation did not reveal any monoclonal antibodies. Kidney ultrasound showed normal kidney sizes with Right kidney 11.8 cm and Left kidney 10.7 cm and no hydronephrosis.

At follow-up six weeks after starting the Lisinopril, serum creatinine increased to 1.8 mg/dL and eGFR had decreased to 40. Labs also noted for hyperkalemia with serum potassium 5.4 mmol/L (reference range 3.6-5.3 mmol/L). Urine analysis showed an improvement in Urine protein- creatinine ratio to 0.5 g (reference range 0.0-0.4) and presence of hyaline casts. The lisinopril dose was reduced to 10 mg daily. Amlodipine dose was increased to 10 mg from 5 mg. Labs six weeks later included marginally improved creatinine to 1.72 mg/dl (from 1.86) and eGFR marginally improved to 44. Urine analysis showed urine protein creatinine ratio of 0.6g but continued to show hyaline casts. His potassium remained elevated at 5.5 mmol/L. Lisinopril was discontinued and he was started on hydralazine, and amlodipine was continued.

Two months later labs were repeated and serum creatinine continued to improve to 1.4 mg/dl and eGFR had improved to 53. However, Urine protein-creatinine ratio had worsened to 1.4g. Serum Potassium was normal at 4.2 mmol/L. Urine did not show hyaline casts.

There was increased clinical suspicion for renal artery stenosis with:

a) Recent worsening hypertension
b) Change in creatinine much more than anticipated after the introduction of Angiotensin converting enzyme inhibitor (lisinopril) but improved significantly after stopping the lisinopril.
c) Hyperkalemia that resolved on stopping lisinopril
d) Hyaline casts that resolved on stopping lisinopril (Hyaline casts can appear in the urine due to many causes, decreased renal perfusion being one of them)

MR Angiogram of the renal blood vessels noted moderate (~50%) narrowing of the origin of the left renal artery which was otherwise patent.

He was referred to interventional radiology for further evaluation and management. Initial evaluation by interventional radiology found low suspicion of clinically significant renal artery stenosis. Ultrasound renal doppler done by interventional radiologist did not show any evidence of renal artery stenosis. The evaluation was limited due to patient's body habitus.

He continued to have worsening proteinuria when not on ACE inhibitors. Urine protein creatinine ratio increased to 2.1 gm. Lowest dose of Lisinopril 5 mg was introduced and Creatinine progressively worsened on serial lab checks to 2.4 mg/dl. Kidney biopsy showed segmentally thinning glomerular basement membranes, ischemic glomerular changes, moderate to severe arterial sclerosis, and no evidence of any immune complex mediated process.

Based on the biopsy findings and worsening creatinine, Interventional radiology performed bilateral Renal angiogram which demonstrated greater than 90 percent left renal artery stenosis. He underwent a successful stent placement into the left renal artery.[1]

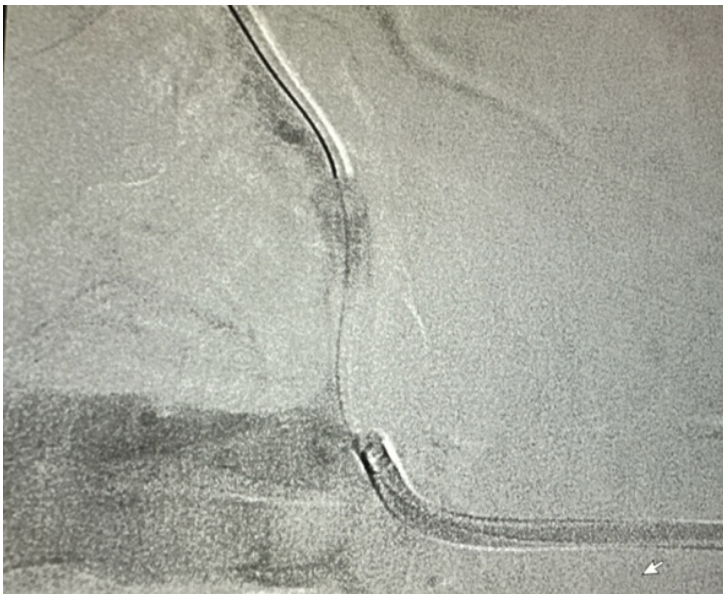

Figure 1 – before Intervention

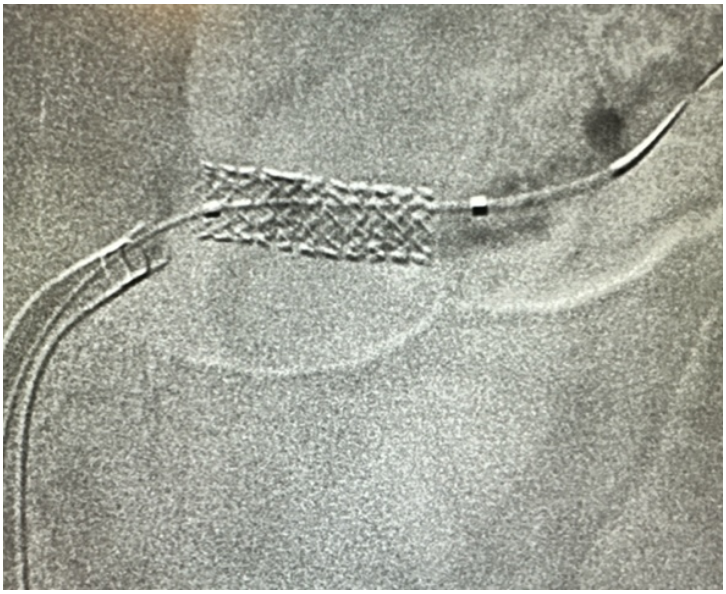

Figure 2 – after Intervention

Discussion

In renal artery stenosis, invasive therapy is recommended if there is luminal occlusion of at least 60-75%[2] and rapidly progressive disease/worsening creatinine, failure to use appropriate medical therapy, difficult to control hypertension, unexplained heart failure.

This patient's initial imaging underestimated the severity of the occlusion. Strong clinical suspicion led to further diagnostic procedures and interventions.

After stent placement his creatinine improved to 1.8 mg/dl and is being closely followed. Thin basement membrane was probably responsible for the proteinuria. The clinical scenario was not suggestive of Alport's Syndrome, although genetic evaluation is being considered. This case illustrates that high clinical suspicion warrants aggressive evaluation despite of initially insignificant ultrasound doppler and MR Angiogram.

REFERENCES

1. **Cooper CJ, Murphy TP, Cutlip DE, Jamerson K, Henrich W, Reid DM, Cohen DJ, Matsumoto AH, Steffes M, Jaff MR, Prince MR, Lewis EF, Tuttle KR, Shapiro JI, Rundback JH, Massaro JM, D'Agostino RB Sr, Dworkin LD; CORAL Investigators**. Stenting and medical therapy for atherosclerotic renal-artery stenosis. *N Engl J Med*. 2014 Jan 2;370(1):13-22. doi: 10.1056/NEJMoa1310753. Epub 2013 Nov 18. PMID: 24245566; PMCID: PMC4815927.
2. **Drieghe B, Madaric J, Sarno G, Manoharan G, Bartunek J, Heyndrickx GR, Pijls NH, De Bruyne B**. Assessment of renal artery stenosis: side-by-side comparison of angiography and duplex ultrasound with pressure gradient measurements. *Eur Heart J*. 2008 Feb;29(4):517-24. doi: 10.1093/eurheartj/ehm631. PMID: 18276621.