LEARNING FROM IMAGES

Transplant Renal Artery Stenosis

Adrian Sequeira* and Kenneth Abreo

Interventional Nephrology Section, Division of Nephrology and Hypertension, Department of Medicine, Louisiana State University Health Sciences Center, Shreveport, LA, USA

Keywords: Transplant, renal, artery, stenosis.

CASE

A 42 y/o male, a recipient of a cadaveric renal transplant, was evaluated 3 years later for worsening blood pressure and lower extremity edema. He was on 7 anti-hypertensive medications. His serum creatinine had gradually increased from 2.5mg/dl to 3.1mg/dl over the last year. The initial renal Doppler ultrasound followed later by an angioimgram demonstrated the presence of a hemodynamically significant renal artery stenotic lesion (Fig. 1A). Following angioplasty and stent placement (Fig. 1B, C), his serum creatinine stabilized at 2.8mg/dl and his blood pressure was well controlled on 5 antihypertensives.

Fig. (1). (A) Arteriogram showing 75-80% anastomotic stenosis of the transplant renal artery at its junction with the external iliac artery (block arrow). (B) Fluoroscopic image following angioplasty and stent deployment at the anastomotic stenosis. (C) Arteriogram following stent deployment shows minimal residual stenosis.

The incidence of post transplant renal artery stenosis (TRAS) varies from 1-23% and it is responsible for upto 5% of post transplant hypertension [1]. The stenosis may occur at 3 locations: Proximal to the area of anastomosis (i.e., within the recipient’s artery), at the level of the anastomosis or distally within the donor renal artery. TRAS usually manifests 3 months to 2 years post transplantation. Anastomotic lesions occur early after transplantation, related to trauma during the surgical process [2]. Late onset TRAS is related to atherosclerotic disease and immune mediated endothelial damage [2]. Clinically, TRAS may be asymptomatic or may present as worsening hypertension with or without graft dysfunction. The latter two features may also result from the use of calcineurin inhibitors (CNI). On exam, an audible bruit over the site of the transplanted organ may be noted but this is not specific [1]. The traditional markers of renal artery stenosis (RAS) like elevated renin activity and hyperkalemia are also not specific in the transplant setting. The former may be elevated in the
presence of an acute rejection, with the concurrent use of diuretics for blood pressure control and may also arise from the native kidneys [1, 3]. Hyperkalemia may occur from the use of CNIs. Color Doppler ultrasonography (CDU) is used to screen patients. Magnetic resonance angiography (MRA) is superior to spiral CT [1] but angiogram is the gold standard. As CDU and MRA can produce false negative results, one may proceed with angiography if the clinical suspicion if high [2]. If the renal function is stable and there is no hemodynamically significant stenosis, then management is conservative with follow up CDU evaluation to assess progression. Progression of stenosis accompanied by worsening renal function and hard to control hypertension are suitable indications for percutaneous transluminal angioplasty (PTA) and stent placement. Clinical success rate as determined by an improvement in blood pressure and or creatinine is seen in 75% of cases [4]. PTA with stent placement has a recurrence rate of less than 10% [4]. Surgical revision is indicated if PTA is unsuccessful. In addition, anastomotic lesions do better surgically [1].

CONFLICT OF INTEREST

The authors confirm that this article content has no conflict of interest.

ACKNOWLEDGEMENTS

Declared none.

REFERENCES