CASE REPORT

RENAL ARTERY STENOSIS

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A Forty-eight years gentleman, well known social figure and the brother of our young colleague, presented with a history of hypertension for the last 17 years. During this protracted period he had been using various medications to combat hypertension. The spectrum stretched from diuretics to β-blockers, calcium channel blockers and ACE-inhibitors. His blood pressure (BP) had displayed a variable response to this wide range of drugs. The response had also been subject to fluctuations even to the individual drugs. According to the patient sometimes the response would be appreciable as evident from the scaling down of BP, while at times the BP would turn refractory and reluctant to show any signs of reasonable abatement. At presentation he was using Amlodipine 5mg and Metoprolol 50mg daily. On examination his BP was 160/100 mmHg. There was no radio-femoral delay or abdominal bruit. Cardiovascular examination was unremarkable. No other clinical clue could be found for the secondary cause of his hypertension. Laboratory investigations revealed normal renal function. A difference in the size of the two kidneys was noticed on ultrasonography. The size of the right kidney was 9.2 x 3.6 cm and that of the left 11.1 x 5.3 cm. The outline of the smaller right kidney was reported to be smooth. His relatively younger age, poor response to therapy and difference in the size of the two kidneys on ultrasonography prompted us to refer him to a tertiary care centre to dig out the possible renal cause for his hypertension. Investigations at referral centre revealed Haemoglobin 15 g/dl, fasting blood sugar 5.0 mmol/l, blood urea 4.4 mmol/l, serum creatinine 120 μmol /l, serum sodium 139 mmol/l, serum potassium 4.0 mmol /l and plasma rennin 4.29 ηg /ml (Reference range 0.31-3.95 ηg /ml). A renal angiogram was performed which revealed critical stenosis of the right renal artery. Percutaneous renal angioplasty and stenting was performed and it was successfully dilated. (Figures 1 & 2) After the procedure although the patient still has hypertension but the response to medications is favourable demonstrating signs of stability. We are planning to gradually taper off his antihypertensive medications.

While treating a patient with hypertension we must search for its cause in every patient, especially in those with younger age, negative family history and severe or poorly responsive hypertension.[1] Renal artery stenosis (RAS) has long been recognized as a cause of systemic hypertension. The diagnosis of renal artery stenosis is often overlooked because of the difficulty in establishing the diagnosis.[2] It is a progressive but potentially correctable problem.[3] It may be due to atherosclerosis or fibromuscular hyperplasia. Athermatous disease commonly affects the proximal portion of renal artery. Fibromuscular hyperplasia results in bands of renal artery stenosis separated by dilated segments giving rise to the classical string of beads (Tasbeeh) appearance on

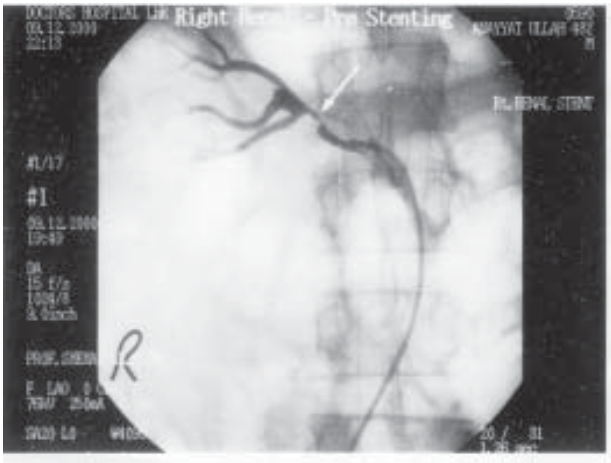

Fig. 1. Pre-Stenting

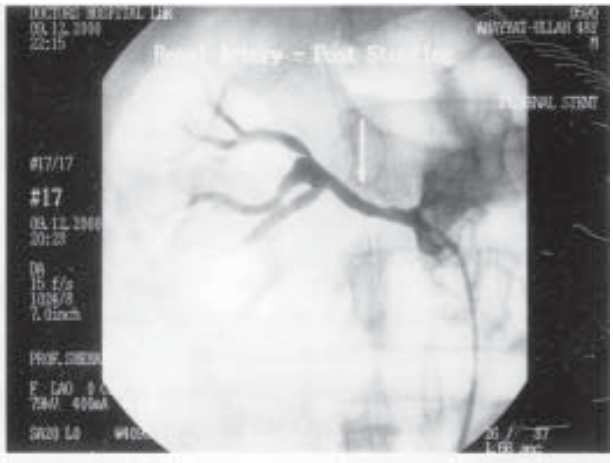

Fig. 2. Post-Stenting

angiography. It is noteworthy that RAS is not always functionally significant and is often found in normotensive patients being investigated for peripheral vascular disease. It may also be an incidental finding without functional significance in patients with hypertension. Hypertension is not an essential component of this disease.[4] The prevalence of incidental renal artery stenosis among hypertensive patients undergoing coronary catheterization is significant.[5] Renovascular hypertension is therefore defined as hypertension cured or improved by correction of RAS. GFR in the affected kidney is maintained by high Angiotensin II levels due to increased release of rennin from the juxtaglomerular cells of the ischemic kidney. Hypertension results from retention of salt and water by both the kidneys. The constricted kidney retains salt and water because of reduced renal arterial pressure in this kidney whereas retention by normal kidney is due to the production of increased amount of rennin by the ischemic kidney. Renal scan after captopril (ACE inhibitor) will show much lowered GFR. Dehydration, hypotension and ACE inhibitors may cause renal failure if RAS is bilateral. Diagnostic pointers to RAS are vascular disease elsewhere, severe or drug resistant hypertension, abdominal bruit, high blood urea or proteinuria. Duplex /Colour Doppler sonography serves a vital role in its diagnosis.[6] It represents a feasible and reliable technique in its detection.[7] Diagnosis is established by renal angiography. Treatment is percutaneous renal angioplasty or bypass surgery. Percutaneous renal angioplasty is the first choice because it is simpler than and as effective as surgical reconstruction.[8] It also has a beneficial effect on renal function.[8] Renal angioplasty can be done in selected patients with renal artery stenosis. The selection of patients for renal angioplasty is important in order to increase the clinical success rate. Clinical as well as angiographic follow-ups for the detection of the restenosis are mandatory.

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