AN INTERESTING CASE OF SECONDARY HYPERTENSION-A CASE REPORT

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Abstract:
INTRODUCTION: Renovascular hypertension is an important cause of secondary hypertension. Renal artery stenosis is commonly caused by atherosclerosis (in 90 percentage of cases) and fibromuscular dysplasia (in 10 percentage of cases). CASE PRESENTATION: A young female aged 20 years was admitted with giddiness and hypertension. Clinically she had right renal artery bruit. On evaluation found to have unilateral contracted right kidney in ultrasound. Doppler study showed dampened flow and increased acceleration time in right renal artery. Subsequent CT angiogram revealed right renal artery stenosis in the proximal segment. Diuretic renal study showed reduced size, perfusion and function of right kidney. Hypertension was easily controlled with antihypertensive drugs and she was referred to higher centre for surgical correction. CONCLUSION: Renovascular hypertension is the most common cause of renovascular stenosis in patients less than 40 years especially in females, typically presenting as hypertension. In contrast to atherosclerotic renal artery stenosis, fibromuscular dysplasia is often cured by angioplasty. Keyword: Renovascular hypertension, Renal artery stenosis, Fibromuscular dysplasia

INTRODUCTION: More than 80–95% of the cases of hypertension have no single and reversible cause detectable and termed the essential or primary hypertension. In remaining 5–20% of cases a definable cause of hypertension can be made out, hence termed secondary hypertension (chronic kidney disease, renal artery stenosis, coarctation of aorta, cushings syndrome, pheochromocytoma etc). The most common curable form of hypertension is renal artery stenosis which has two principle causes – fibromuscular dysplasia in children and young adults (accounts 10% cases) and atherosclerosis in older patients (90% cases). CASE PRESENTATION: A 20 years old female from Ottapelaram IP NO 36828 was admitted with history of giddiness for 1 week. She is a known hypertensive for past 4 years. History of giddiness intermittently for 1 week. No
history of syncope, vertigo or swaying while walking. No history of seizures or vomiting or headache. No history of blurring of vision. No history of chest pain, palpitation or breathlessness. No history of decreased urine output or leg swelling. Past medical history revealed lower motor neuron type of facial nerve palsy 4 years back, treated in a private hospital and recovered in 1 month. During that treatment she was informed as having high blood pressure, but not prescribed drugs due to fluctuating BP. Patient is on regular treatment with antihypertensives for past 6 months. She gave history of her BP being 160/100 mm Hg persistently despite regular medications for 6 months. Not a known diabetic / ischemic heart disease / stroke/ epileptic / renal disease. No history of hypertension among her parents or siblings. Attained menarche 6 years back, regular menstrual cycles with normal flow.

On examination: She was comfortable, moderately built, pale, no icterus or pedal oedema. Pulse rate 90 / min, regular, normal volume, no specific character, felt equally in all peripheral vessels, no radiofemoral delay. No carotid bruit. BP on admission was right upper limb 180/100 mm Hg, left upper limb 180/100 mm, right lower limb 190/100 mm and left lower limb 190/100 mm. No postural variation of BP. Cardiovascular system examination – apical impulse normal position and character, S1 normal, S2 heard with A2 louder. No murmur. Abdomen examination – right renal artery bruit heard. No organomegaly. No pulsations. Respiratory system and neurological examination were normal. Fundus examination normal, no evidence of hypertensive retinopathy.

On investigations: urine-albumin, sugar, deposits-nil; Blood sugar 86 mg/dl, urea 18 mg/dl, creatinine 0.8 mg/dl; serum electrolytes Na 135 meq, K 4.8 meq/l, Ca 8.1 mg%; Body weight 49 kg; By applying Cockcroft Gault formula for renal function (140-age) x body weight x 0.85 / 72 x serum creatinine) her creatinine clearance is 87 ml/min.

Complete blood count total count 7400, differential count – P70%L27% E3%, Hb 7.6 g%, RBC 4.40 million, PCV 36.4%, platelet 4.62 lakhs, ESR 40mm/hr. Liver function tests normal, Lipid profile – cholesterol 102 mg, TGL 72 mg/dl. ECG – 100/min, sinus rhythm, normal axis, left ventricular hypertrophy by voltage criteria, few ventricular premature complexes present. Chest XRay – heart and lungs normal. Ultrasound abdomen and pelvis – right kidney 8.6x2.9 cm, left kidney 10.2 x 4.6 cm cortical medullary distinction maintained, cortical echoes normal. No obvious adrenal mass. Echocardiogram normal study, ejection fraction 74%. Peripher al smear cytology-microcytic hypochromic anemia; Serum uric acid 4.9 mg%; Rheumatoid factor negative, thyroid profile normal. 24 hour urine estimation volume 1300 ml, protein-0.2 g 24 hour urine VMA- 4 mg (normal 2-7 mg/24 hr) Plasma renin level (after withholding antihypertensives)- 4.5 ng/ml/hr (normal 1.9 – 3.7 ng/ml/hr). Stimulated plasma renin level 21 ng/ml/hr. Renal artery Doppler study – right kidney small in size, flow dampened, increased acceleration time in the hilar, interlobar and arcuate arteries in right kidney signifying proximal narrowing. However renal artery orifice could not be traced. Left kidney normal Doppler study. CT abdomen – right kidney smaller 7.7 x 3 cm. No renal calculus.
Left kidney 9.8 x 3.5 cm, normal. CT renal angiogram – abdominal aorta normal. Single renal artery on both sides. Critical stenosis of proximal segment of right renal artery (about 10 mm). Multiple tiny perinephric and periureteric collaterals on right side, reforming mid and distal segments of right renal artery. Small size of right kidney. Left renal artery normal. No evidence of aneurysm / AV malformation or stenosis in segmental, interlobar and lobar branches in both sides. Single renal vein in both sides.

CT RENAL ANGIOGRAM

ANTERIOR VIEW-CONTRACTED RIGHT KIDNEY WITH STENOSED PROXIMAL SEGMENT OF RIGHT RENAL ARTERY

POSTERIOR VIEW

TINY COLLATERALS REFORMING MID AND DISTAL SEGMENT OF RIGHT RENAL ARTERY

DIURETIC RENAL STUDY:- Tc -99 EC with lasix renal dynamic study showed reduced perfusion of right kidney. The right kidney is small and showed reduced tracer concentration. Left kidney showed normal perfusion and normal tracer concentration. The intrarenal transit is normal on both sides-4 min(normal 2-4 min). There is excretion into the pelvis, ureters and into the bladder. No obvious obstruction to drainage. Excretory T₁/₂ Left kidney=06 min, Right kidney=11 min. Total Glomerular filtration rate=101 ml/min(normal lower limit of GFR for age = 90 ml/min). Relative renal function- Left Kidney=70% (71ml/min), Right kidney=30% (30 ml/min).Impression of diuretic renal study: reduced size, reduced perfusion and reduced function of right kidney with no obstruction to drainage. Normal function of left kidney with no obstruction to drainage.

DISCUSSION:- Renal artery stenosis may be due to atherosclerosis or fibromuscular dysplasia. Other causes include arteritis, renal artery aneurysm, extrinsic compression etc. Renal artery stenosis can be unilateral or bilateral.
Pathophysiology:
Renal artery stenosis is suggested to cause 2 types of hypertension. With unilateral Renal artery stenosis and normal functioning and perfused contralateral kidney, BP elevation is renin dependent and characterised by increased peripheral resistance. Normal kidney maintains natriuresis. When stenosis is bilateral or when the other kidney is absent or dysfunctional because of the absence of natriuretic effect intravascular volume increases and renin secretion decreases over a period of 5 – 10 days.

Fibromuscular dysplasia is the usual cause of renal artery stenosis in young age group especially in females. Fibromuscular dysplasia of the renal artery is commonly unilateral, 35% of cases are bilateral. Typically presents as hypertension and rarely causes renal impairment. Usually involves the middle segment or distal segment of renal artery. In contrast atherosclerotic renal artery stenosis affects older age group, more common in men, especially smokers, stenosis involving proximal segment of renal artery near ostia, with evidence of atherosclerosis elsewhere, causes hypertension often treatment resistant, often associated with renal impairment.

Fibromuscular dysplasia pathologically classified depending on the arterial wall involved. Medial fibroplasia is the most common characterized by classic angiographic string of beads appearance. Intimal fibroplasia occurs in less than 10%, angiographically may appear as focal, concentric stenosis and is often bilateral. Perimedial dysplasia (about 10%) causes multiple highgrade stenosis of the main renal artery without aneurysmal dilatation. Adventitial hyperplasia is the rarest type with limited angiographic information. Sharply localized tubular areas of stenosis have been observed. Noninvasive imaging techniques-Doppler ultrasound, captopril renography, CT and MR angiography, digital subtraction angiography. Angiography is the standard test for diagnosing.

Treatment:
Treatment of unilateral renal artery stenosis responds well to renin angiotensin system blockade such as ACE inhibitors and beta blockers. Renin angiotensin system blockade is contraindicated in bilateral renal artery stenosis. Those resistant to drug treatment with antihypertensives, those who have lost the renal volume because of ischaemic nephropathy should be considered percutaneous transluminal renal angioplasty with or without stenting, especially in fibromuscular dysplasia. Complications of PTCA include Renal artery dissection and perforation, contrast nephropathy, hematoma and pseudoaneurysm at access site. Conclusion:
In this case report, we presented a case of unilateral renal artery stenosis causing hypertension in a young female due to fibromuscular dysplasia involving the proximal segment of the main renal artery. Her BP was controlled with antihypertensives and she has been planned for PTCA at higher centre.

REFERENCES:
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