
CASE REPORT**Could Length and Reduced Diameter of Aberrant Renal Artery be Attributed to Significant Reno-vascular Hypertension in the Absence of Stenosis?**

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Abstract:

Hypertension depends upon various factors such as resistance, pressure difference viscosity, caliber and length of a vessel according to Hagen–Poiseuille law. Length of a vessel is one of the important causes of hypertension of reno–vascular origin in adolescence in the aberrant renal arteries. An adolescent girl with bilateral uveitis and hypertension was evaluated for the cause of hypertension. Serum rennin and aldosterone levels favoured hypertension of renovascular origin. Ultrasonography and C.T. scan angiography of the abdomen revealed multiple aberrant renal arteries without any stenosis. In the absence of renal artery stenosis length and caliber of the aberrant arteries are the parameters which contribute significantly to reno-vascular hypertension.

Keywords: Hypertension, Aberrant Artery, Stenosis

Introduction:

Any artery arising from abdominal aorta in addition to the main artery should be named as accessory artery and those arising from a source other than aorta should be called aberrant [1]. Main accessory and aberrant all are end or segmental arteries and hence the perfusion of the renal segment depends on the Hagen-Poiseuille law [2]. Various components of this law, either isolated or in combination may play a major role in affecting the resistance flow and blood pressure inside a vessel. There is a strong correlation between the incidence of aberrant renal arteries and the incidence of systemic hypertension [3].

Case Report:

A 13 year old female was referred from Department of ophthalmology with complaint of

diminished vision for 20 days and then with sudden loss of vision in both eyes, diagnosed as bilateral uveitis with exudative retinal detachment to the department of pediatrics on 6/8/15 for request of detail evaluation, appropriate drug dose of methyl prednisolone and with urine examination report of proteinuria, hematuria, few pus cells and passage of calcium oxalate crystals. She was evaluated in detail.

There was no significant pediatric history of headache, sudden pallor palpitation, sweating, vomiting or any signs of space occupying lesion, convulsions, no family history hypertension neither any intake of drugs leading to hypertension. There was no complaint of fever, increased frequency, urgency or burning of micturition, her general physical and systematic examination was normal, except eye changes as mentioned earlier. Her vital signs showed tachycardia 150/mt, blood pressure readings were 160/92, 160/90, 158/90, 160/94 in right and left upper limb, right and left lower limb respectively (which was above upper limits of normal). Being in early adolescence hypertension of renovascular origin was suspected as a common cause. Her blood pressure became normal with Furosemide and Nifedipine. She underwent following investigations C.B.C, serum electrolytes, calcium, renal function tests, liver function tests, HIV, HCV, Coagulation profile, urine examination which were normal. No growth on urine for c/s, sickling

test, HBsAg (Negative) X-ray chest, E.C.G, U.S.G. Abdomen and 2-D Echocardiography all were normal. Her plasma rennin activity was high 24.42 ng/ml/hr (normal values being 0.5 -3.3) and plasma aldosterone was also high 142ng/dl (normal value 2-22 in supine and 4-48 in upright). In repeat U.S.G abdomen left kidney was supplied by dual renal arteries with left lower pole having left renal artery. Cortico-medullary differentiation was well preserved bilaterally. A 16 slice C.T. scan with renal angiography revealed that the right kidney was supplied by two arteries and left kidney by three arteries. Right main renal artery arose at L₁-L₂ level, right accessory renal artery at L₃ level supplying upper pole of right kidney and another slender branch to diaphragm. Left main renal artery arose at L₁ inferior level, whereas one left accessory artery arises at L₁ -L₂ disc and another from left common iliac artery. There was no significant stenosis of the major vessels. Both kidneys demonstrated normal parenchymal enhancement and corticomedullary differentiation renal outlines were smooth. The details of renal vessels are in Table 1.

Table 1: Details of Length and Caliber of Renal Arteries

Artery	Length of Vessel	Caliber
Right Renal Artery	7.8 mm	6.1 mm
Right Renal Vein	27.9 mm	15.8 mm
Left Main Renal Artery	51.6 mm	4.9 mm
Accessory Left Renal Artery	51.8 mm	2.9 mm
Left Renal Vein	34.9 mm	17.5 mm

On the basis of available history, examination and investigations, she was diagnosed as a case of reno-vascular hypertension.

Discussion:

Variations of renal arteries are recorded from mid nineteenth century and frequency varies from 25% to 61% of cadavers [4-6]. A persuasive literature supports a dominant role for activation of the rennin-angiotensin system in maintaining the elevated systemic pressure in patients with unilateral artery stenosis [7-9]. In our case also, we tried to assess the cause of reno-vascular hypertension (as her plasma rennin activity and plasma aldosterone levels were very high), that which factor was responsible for hypertension, particularly in the absence of any stenosis in both major renal arteries as well as aberrant renal arteries. Studies [10-12] that have attempted to correlate the etiological significance to the simultaneous presence of nonstenotic multiple renal arteries and a modest elevation of systemic renin activity discovered during evaluation of hypertensive subjects.

In a study by Kem *et al.* [12] of two cases similar to renovascular hypertension with aberrant arteries, without stenosis, a high rennin activity on serial measurement of rennin from renal vein was reported. Hypertension subsided on partial nephrectomy of this segment which had a supply of aberrant artery, and provided evidence for explanation for the presence of hypertension. The pattern of blood supply to the kidney is segmental and main renal arteries as well as aberrant arteries are end arteries without inter segmental anastomosis. According to authors, it was a possibility that there could be a relative ischemia due to various abnormal components of Hagen-Poiseuille's law acting on aberrant arteries resulting in increase of resistance leading to under

perfusion, release of angiotensin II and resultant hypertension.

Hagen Poiseuille law is $F(\text{flow}) \propto \delta P \pi / R$,

Where, $R \propto 8 \mu L / r^4$,

$F(\text{flow}) \propto \delta P \pi r^4 / 8 \mu L$, where viscosity (μ), resistance (R), length of the vessel (L), radius of the vessel (r), (δP) the pressure difference. However as, $R \propto 8 \mu L / r^4$, the greater the length or tortuosity of the artery the greater will be resistance, but a small decrease in radius will increase resistance (R) four times as r^4 (fourth power) is the denominator. Gyori [10] postulated that supernumerary renal arteries were more prone to develop stenosing lesions and more importantly, he also speculated that they are longer and their caliber is smaller than main trunk, thus raising resistance and potentially predisposing to under perfusion according to Hagen-Poiseuille law. Glodny *et al.* [11] found a higher peripheral plasma rennin activity in a group of hypertensive patients with aberrant renal arteries as compared to control group of hypertensive patients without aberrant renal arteries. A study by Kem *et al.* [12] has estimated the critical actual length of an aberrant artery required that would be capable of increasing the resistance to the degree that is at least equal to a > 50% reduction in the diameter of the main trunk and also the length of the aberrant vessel that is capable of contributing to the increased vascular resistance and reduced vascular flow, with all other components remaining unchanged.

They also noted before and normalization of blood pressure after resection of the non stenosed but narrow elongated accessory artery with partial nephrectomy. In our case the left aberrant renal artery was an end artery and its length was equivalent to main left renal artery but the caliber was narrower than main renal artery (without stenosis), therefore the radius (r) and length (L) might have contributed significantly to hypertension, the probable explanation. However, in our case the patient was not operated for partial nephrectomy and hence the resultant correction of the hypertension could not be confirmed.

Conclusion:

Presence of long tortuous, narrow multiple accessory /aberrant arteries in the absence of significant stenosis may contribute to hypertension and hence should be considered in the differential diagnosis of a cause of renovascular hypertension in children. As there are not many studies with significant statistical data available, it is difficult to comment on percentage caliber difference for such aberrant renal arteries.

Limitation:

In this study, there were bilateral aberrant arteries, so the overall rise in renin level might have been contributed by both sides, though selective renal vein rennin levels would have helped to confirm the diagnosis but due to lack of investigation facility it was not performed.

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