PEDIATRIC HYPERTENSION: CLINICAL PROFILE AND ETIOLOGY

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ABSTRACT

Thirty six symptomatic hospitalized hypertensive children were evaluated for clinical profile and etiology. They were divided into two groups of 23 and 13 patients, respectively of chronic persistent hypertension and acute transient hypertension. Headache, failure to thrive, dyspnea and edema were common clinical features. Renal parenchymal pathology was the commonest etiology in both groups with a tubulo-interstitial pathology being more common amongst chronic hypertensives and acute glomerulonephritis in acute transient hypertensives. Essential hypertension was uncommon, found in only two patients with chronic hypertension reflecting probably an asymptomatic status of most patients with essential hypertension.

Key words: *Hypertension, Acute glomerulonephritis.*

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Although not as frequently seen in children as in adults, hypertension is now a more commonly diagnosed entity in the pediatric age group. The second Task Force (1987) on blood pressure control in children has recommended a regular yearly check up of blood pressure after 3 years of age by primary care physician(1) but such a recommendation is questionable for children in developing countries with a heavy load of sick patients for the pediatrician.

It is now a recognised concept that roots of essential hypertension extend back into childhood with persistence of rank order with age, a concept known as "Tracking"(1). As opposed to earlier studies in which renal parenchymal pathology was the commonest cause of hypertension in children(2-6), some reports have inferred that primary hypertension is more common particularly following institution of blood pressure evaluation as a part of routine physical examination(1,7,8). In this study, we evaluated symptomatic hospitalized hypertensive children to find out the cause and also to know how they manifest clinically.

Material and Methods

Thirty six patients with elevated systemic blood pressure in the age group upto sixteen years were evaluated for the clinical profile and etiology. Three readings of blood pressure higher than values suggested by the Second Task Force (derived from normative data from 70,000 children) were considered as evidence of hypertension in the child. These patients were divided into two groups: Group A–23 patients with chronic persistent hypertension and Group B–13 patients with acute transient hypertension. Hypertension lasting more than three months and/or unlikely to

resolve spontaneously was defined as chronic persistent hypertension(4). A detailed history was taken and physical examination carried out. Investigations were done to find out the cause of hypertension.

Diagnostic Studies

The routine investigations which were carried out in all included hemogram; urine analysis for albumin by 20% sulfasalicylic acid and microscopy for RBCs, WBCs and casts; urine culture of midstream urine samples; blood urea; serum creatinine; serum electrolytes; *X*-ray chest and ECG.

If the cause of hypertension was detected after history, examination and basic diagnostic studies were carried out, *e.g.*, in patients of Group B with acute transient hypertension, no special studies except ASO and CRP were performed. Group A patients of chronic persistent hypertension were subjected to certain special investigations, *e.g*, urinary vanil mandelic acid estimation by chemical method, urinary catecholamines by bioassay and serum cortisol and plasma renin activity estimation by radio-immunoassay. The radiological investigations done on some selected patients of Group A included ultrasound examination of the abdomen, intravenous pyelography, micturating cysto urethrography (MCU) and digital subtraction angiography (DSA) for evaluation of aorta, inferior vena cava or renal arteries. Kidney biopsy was done with Vim Silverman needle and specimens stained with hematoxylin eosin stain. Two patients were operated upon and the tumor mass sent for histopathology.

Results

There were twenty three patients in the group with chronic persistent hypertension (Group A). Thirteen of these patients were male. There were three infants less than one year of age and all were males. The male : female ratio in the older age groups was, therefore, 1:1. All patients with acute transient hypertension (Group B) were less than twelve years of age. The age and sex distribution of these patients is shown in *Table I*.

The systolic blood pressure values at presentation in both these groups are depicted in *Fig. 1* and diastolic blood pressure values in *Fig. 2*. Whereas, 20 out of 23 patients (86.9%) in Group A had severe hypertension (>99th percentile), all but one patient in Group B was in this category.

TABLE I—*Age and Sex Distribution*

Age (yrs)	Chronic persistent hypertension				Acute transient hypertension			
	M	F	Total	Percentage	M	F	Total	Percentage
0-1	3	-	3	13.0	2	-	2	15.4
1.1-6	4	1	5	21.7	2	4	6	46.2
6.1-12	4	6	10	43.5	2	3	5	38.5
>12	2	3	5	21.7	-	-	-	-
Total	13	10	23	100.0	6	7	13	100.0

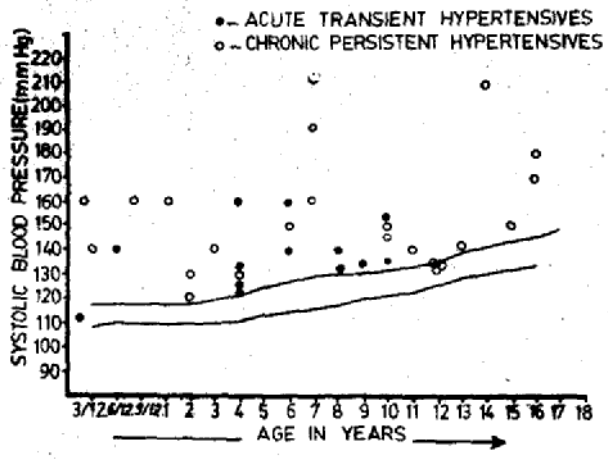

Fig. 1. Systolic blood pressure on admission.

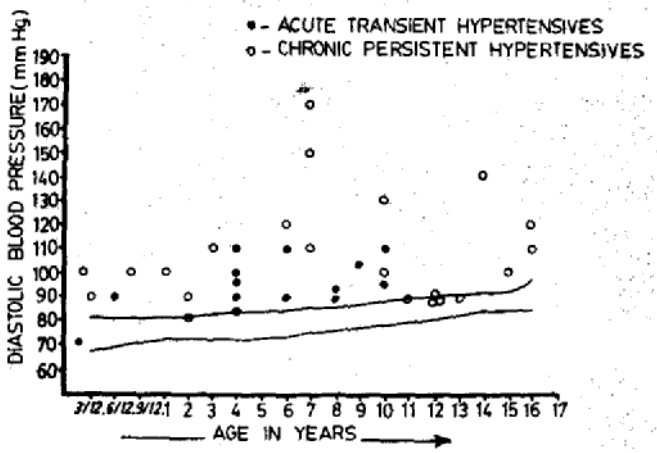

Fig. 2. Diastolic blood pressure on admission.

Clinical Features and Etiology

Chronic persistent hypertensives: Headache was the commonest clinical feature found in 13 patients (52.5%) followed by edema in 9 (39.1%), failure to thrive in 8 (34.8%), dyspnea in 7 (30.4%), convulsions in 6 (26.1%), palpitations in 5 (21.7%), repeated vomiting in 5 (21.7%), diminished vision in 4 (17.4%) and papilledema in 3 (13.0%).

The etiological distribution of chronic persistent hypertension is shown in *Table II* Renal etiology was responsible for 14 of the 23 patients (60.9%) (11 had renal parenchymal and 3 renovascular). Of these 11 patients with renal parenchymal pathology, 5 had chronic pyelonephritis (confirmed by kidney biopsy) and 2 had glomerulopathy- one of membranoproliferative glomerulonephritis with basement membrane thickening and mesangial proliferation and the other had crescentric glomerulonephritis. The remaining 3 had hydronephrosis as diagnosed on ultrasonography and intravenous pyelography (IVP)-2 had unilateral hydronephrosis due to congenital pelvi-ureteric junction obstruction (*Fig. 3*) and the third had bilateral hydronephrosis due to meatal stenosis. The last case was diagnosed to have congenitally dysplastic kidney on ultrasonography and IVP and which was surgically removed.

TABLE II —*Etiological Distribution of Chronic Persistent Hypertension*

Etiology	No.	Percentage
Renal	14	60.9
(*a*) Renal parenchymal	11	47.8
(*b*) Renovascular	3	13.0
Aortoarteritis	3	13.0
Coarctation of aorta	1	4.4
Neuroblastoma	2	8.7
Pheochromocytoma	1	4.4
Essential	2	8.7
Total	23	100.0

Renovascular hypertension in 3 patients included 2 patients with renal artery stenosis (*Fig. 4*) and one with renal vein thrombosis both being diagnosed on digital subtraction angiography (DSA). Aortoarteritis was a more common vascular cause than coarctation, their proportion being 3:1 again the mode of diagnosis being DSA.

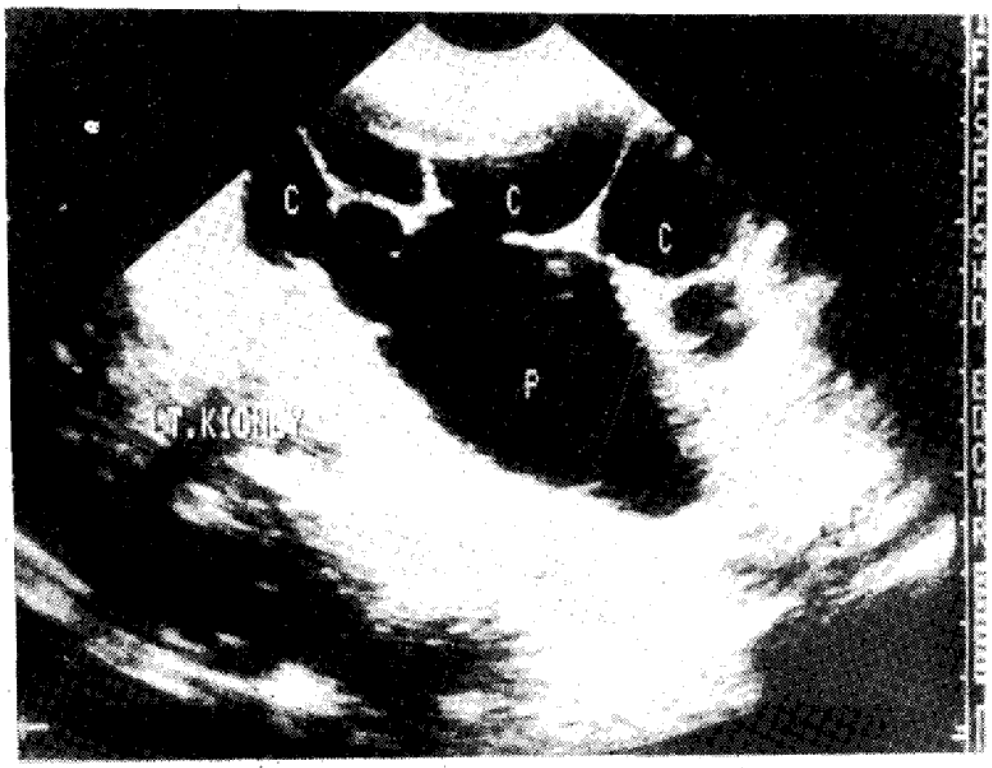

Fig. 3. Oblique Scan showing grossly dilated pelvicalyceal system with narrowed PU junction.

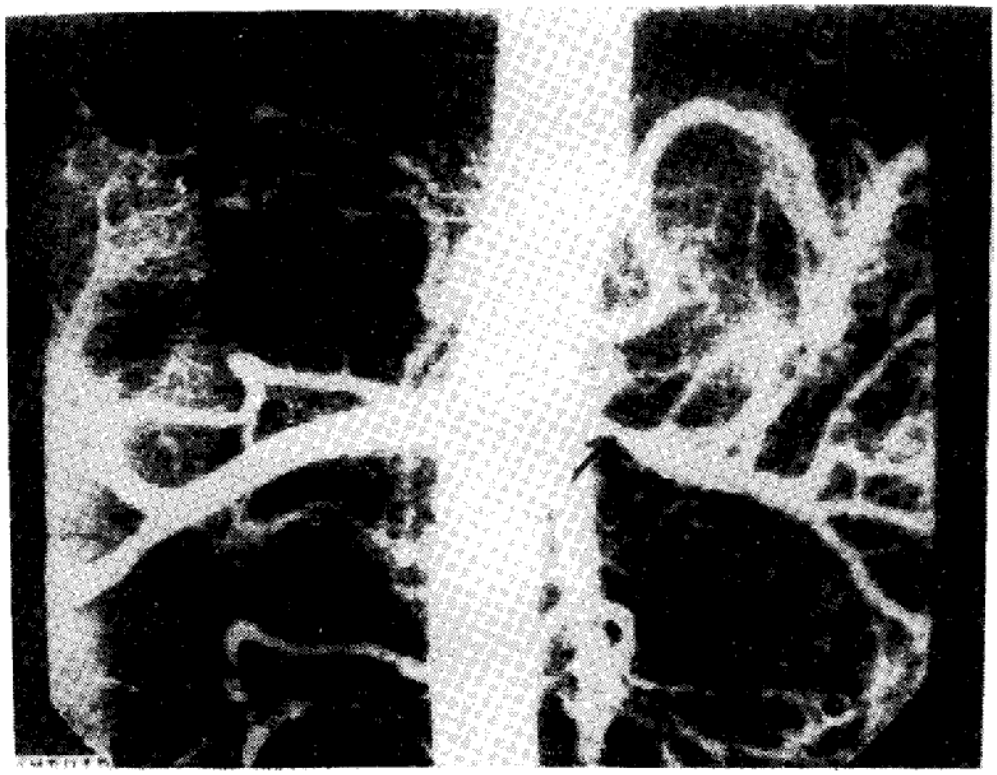

Fig. 4. DSA showing left renal artery stenosis.

There were three patients, who had masses in the right suprarenal region and another had a well defined encapsulated mass in the region of pancreatic tail and they were operated upon.

On histopathological examination, the masses removed from the former patients showed pheochromocytoma and neuroblastoma and in the latter case as neuroblastoma. The remaining patients were diagnosed as having essential hypertension as no cause of hypertension, after their investigative work up, was known.

Acute *transient hypertensives*: Among 13 patients with acute transient hypertension, 9 (69.2%) had typical presentation of acute glomerulonephritis. Edema and oliguria were common clinical features mostly accompanied by microscopic hematuria. The ASO and CRP did not reveal any abnormality. There were 2 patients with iatrogenic Cushing's syndrome, one with hemolytic uremic syndrome and one with Landry Guillaine Barre' syndrome.

Discussion

The selection of cases has been stated to be the most important single factor affecting the etiological distribution of pediatric hypertension in any study(7,8). Earlier studies on symptomatic sick children with severe hypertension have found renal parenchymal pathology to be the commonest cause of hypertension(2-6,9-11). On the other hand, Loggie reported an increase in the percentage of essential hypertension in her practice from 20% in 1969 to 55% in 1975 as picked up on routine blood pressure measurement in her juvenile patients(7). Londe found that 95% of his pediatric patients detected to be hypertensive during routine physical examination had essential hypertension. We, in this study investigated symptomatic hospitalized hypertensive children. No screening methods were employed to detect hypertension in asymptomatic children.

In our study, 65% of 20 patients (excluding 3 infants) of Group A had headache as one of their major complaints. This finding is not in agreement with the reported figures 36, 16 and 4.5% by earlier workers(4,10,11). Excessive crying and failure to thrive were the two most important presenting features in infants with hypertension. Renal parenchymal pathology was the commonest etiology found in 11 (47.8%) patients with chronic persistent hypertension. This is consistent with most of the hospital-based early studies (4-6,9-12). A tubulointerstitial pathology and more specifically chronic pyelonephritis was the commonest etiology in our study. This is consistent with the results of some studies(2,3,10,12) but at variance from others who have found chronic glomerulopathies to be more common(4,6,9) etiology of pediatric hypertension.

Four patients (17.3%) had a vascular pathology, three of them having aortoarteritis while one had coarctation of aorta. Earlier studies from India(5,9,13) have also found aortoarteritis to be a more common entity than coarctation.

Only two patients (8.7%) were diagnosed as having essential hypertension. This low percentage of essential hypertension is in accordance with the earlier studies(2,4-6,9,10).

Our study shows that amongst hospital admitted symptomatic hypertensives, secondary hypertension is much more common than essential hypertension, renal parenchymal hypertension being the commonest form. A low incidence of essential hypertension is probably due to most such patients remaining asymptomatic in the

community with mild hypertension and likely to be detected only on mass screening or regular blood pressure measurement of healthy asymptomatic children.

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