Essential Hypertension in a 17 Years Old Girl: A Possible Correlate with Low Birth Weight and Growth Parameters

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ABSTRACT

We enclose a case of essential hypertension in a 17 years old girl presented for the first time with hypertensive encephalopathy. Various hematological, biochemicals, hormonal and imaging studies failed to explore underlying etiology. A possible correlate of low birth weight and growth parameters with early onset of essential hypertension was postulated.

Keywords: Hypertension, birth weight, growth

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Introduction

Long term hypertension contributes to significant cardiovascular and renal morbidity and mortality[1,2]. Although chronic hypertension is much rarer in the adolescent population than in adults, identifying the hypertensive adolescent and intervening with risk factors that my promote hypertension is important for the clinician treating adolescent. Moreover; since both primary and secondary hypertension may exist in the adolescent, a thorough sequential clinical and diagnostic evaluation must be undertaken including screening urinalysis, blood chemistries and renal imaging studies [3]

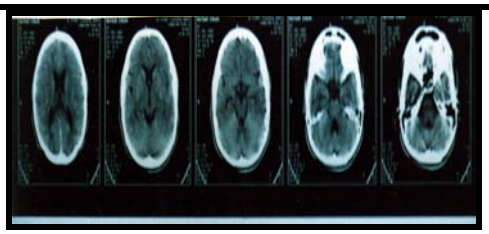
Cases of hypertension starts nowadays to surge in young age group [4] and this triggers a world – wide public deep concern.

Case Report

M.E.A, 17 years old student in the 5th secondary school in Baghdad with reasonable school achievements and obsessive personality, presented to the Al- Kindy Teaching Hospital /Emergency Department/ Baghdad in January 2004 with disturbed consciousness, vomiting and recurrent fits preceded by few days progressively increasing headache. She was irritable with blood pressure (BP) = 200/ 140 mmHg, pulse rate (PR) = 135/ minute, irregular respiration and temperature = 37.7 C^0. Systematic examination revealed normal double rhythm(NDR)heart without murmurs, clear chest and soft abdomen without organomegaly or lymphadenopathy and inaudible renal bruit. Central nervous system (CNS) examination was inconclusive apart from bilateral papilloedema. Urgent computerized tomography (CT) scan showed no space occupying lesion(SOL)with normal ventricles **Figure-1.**

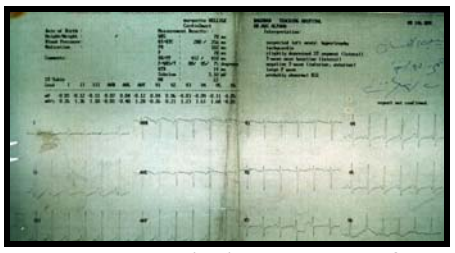
Cautious lumbar puncture was done and showed the following data: clear, specific gravity = 1.004, sugar = 100 mg %, protein = 105 mg %, RBC = 5 / mm^3, WBC = 3/ mm^3 (all lymphocytes), gram stain= negative, Zeihle Nelson stain for AFB = negative, Rose Bengal test = negative, Complement Fixation test for IgG = negative and VDRL = negative. Concomitant blood sugar was 120 mg %. Immediate resuscitation through multiple doses of I.V Mannitol, Frusemide (Lasix) and Diazepam (Valium) succeeded in aborting further fits and regressing BP. Oral hypotensive remedy as Captopril (Capoten) with Acetazolamide (Diamox) were initiated thereafter and sustained further stabilization of BP. Further evaluation of the patient revealed unremarkable history apart from recurrent attacks of headache since the age of 6 years with strong family history of migraine and ischemic heart disease. Furthermore, the patient seemed short and underweight [weight = 41 Kg, height = 144 Cm, all were below the 3rd percentile on the National Center for Health Statistics (NCHS) growth charts]. The family has been worried about her physique and convinced that she was tiny at birth. Both parents have a moderate frame. She has one brother aged 14 years and of reasonable height and weight for his age. Menstrual history showed regular cycles with Tanner stage 4 sexual maturity. Further laboratory investigations were arranged for including: Complete blood count (CBC) [Hb = 11.4 gm / dl, WBC= 7.2 x 10^9/ L with differential count, PLT= adequate and normochromic normocytic blood film without primitive cells], ESR= 27 mm/Ist hr, B. urea= 45 mg %, S. creatinine= 0.7 mg %, S. sodium= 139 meq/L, S. potassium= 4.2 meq/L, S. uric acid= 4.7 mg% , total serum protein= 7.2 gm /dL with normal albumin globulin ratio , S. cholesterol = 170 mg % . B. Rose Bengal test = negative in all titers with negative prozone phenomenon,

Complement for IgG= negative, C- reactive protein = negative, ANF = negative. Echocardiography revealed neither congenital lesion nor pressure or volume overload pattern. Abdominal ultrasound showed no congenital or acquired lesions and without suprarenal mass. Bone age was consistent with chronological age in skeletal survey. Seemed to be invasive and risky as patient and family thought, jejunal biopsy was not accomplished. Meanwhile, serum anti-gliadin antibodies were not feasible because of technical difficulties. Hormonal studies depicted normal serum T_3 ,T_4 , TSH, Prolactin, Estrogen, Progesterone, and Growth Hormone (both basal and after stimulation). Twenty four hrs urine collection for Vanyl Mandellic Acid (VMA) [repeated twice at 3 weeks interval] was normal, the same was true for urine 17 Ketosteriod. Brain magnetic resonance imaging (MRI) showed no SOL with normal ventricles **Figure-4 .**Electrocardiography (ECG) illustrated suspected Left ventricular hypertrophy**Figure-2.** Chest radiography demonstrated normal heart contour with no parenchymal or bony lesion **Figure-3.** Abdominal MRI illustrated normal visceral texture and density with no suprarenal lesion **Figure-5**.Abdominal MRI angiography demonstrated normal calibers of renal vessels **Figure-6.** Oral Captopril and Acetazolamide were continued for the next one month and made stable BP curve with resolution of papilloedema. However, BP started to rise once again and Captopril was unable to stabilize BP despite reaching maximum dose. Additional hypotensive drugs either alone or in combination like diuretics (Frusemide,Spironolactone,Hydrochlorthiazide+ Amiloride) ,Methyl dopa,β blockers (Atenolol),ACEinhibitors(Enalapril,Lisinopril),Angiotensin-2-antagonist as Valsartan (Diovan) and vasodilators (Nifidipine, Diltiazem, Amlodipine) were unsuccessful in targeting normotensive state either because of intolerance to their side effects or reaching maximum dose. Finally, combined Valsartan 160 mg(Diovan) and Atenolol 50 mg (Tenormin) were able to normalize BP with combined diuretic therapy as Chlorthiazide 50 mg + Amiloride 5 mg (Moduretic) added occasionally to cover stressful conditions particularly during menses. She made clinically well over the last one and a half year follow- up with reasonable BP range of 120 – 130/ 70 – 85 mmHg. Apart from Right oculomotor palsy, she had intact CNS with no more papillodema or retinal arteriolar changes.

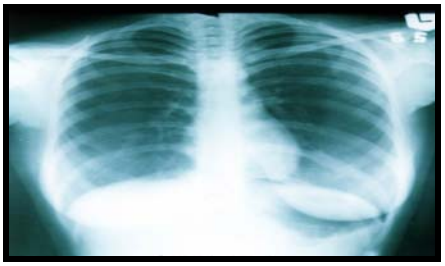


(Figure-1) *Normal CTscan of studied patient*

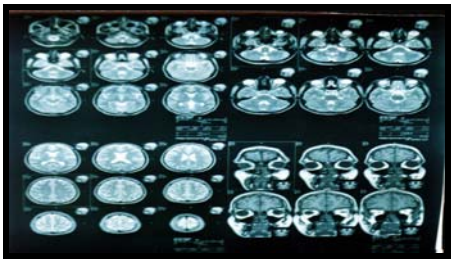
(Figure-2) *Suspected L.Ventricular Hypertrophy in ECG of Studied Patient*



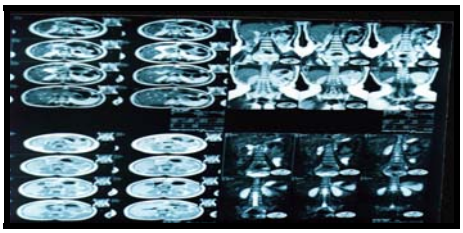
(Figure-3) *Normal Chest X-Ray of Studied Patient*



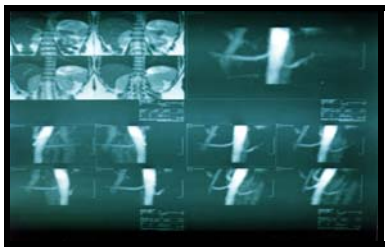
(Figure-4) *Normal MRI of Brain of Studied Patient*



(Figure-5) *Normal Abdominal Mri of Studied Patient*



(Figure-6) *Normal Abdominal Mri Angio of Studied Patient*



Discussion

It was well- known that the younger the hypertensive patient, the greater the chance the hypertension will be secondary to another disease rather than being essential [3,5,6]. However, this concept has been revised with eruption of cases of essential hypertension in younger age group. In one study, 92.7% of studied hypertensive patients aged 13 – 40 years tended to be essential rather than secondary to other diseases [7]. In the current study, the sustained hypertension over one and a half yr. follow- up despite negative urinalysis, biochemical, hematological, hormonal and imaging studies preclude the existing renal, cardiovascular, endocrine, neurological, autoimmune and metabolic etiology for this hypertension. Moreover; the striking observation in the current case is the convinced history of low birth weight and the current growth faltering documented by height and weight being below 3rd percentiles on corresponding NCHS growth charts that can't be attributed to secondary effects of hypertension. Recent studies stressed that arterial BP records is elevated among malnourished adolescents. The mechanism seems to be related to malnutrition during early phase of life suggesting the programming theory which proposes that intrauterine undernutrition continuing during the Ist years of life causes permanent metabolic and vascular disorders. These alterations amplified with time depending on the quality of diet and environmental factors [8,9,10,]. Furthermore; accelerated neonatal growth was found to enhance later propensity to long-term cardiovascular diseases [11,12].

In conclusion, inquiry about birth weight and ascertaining growth profiles seem to represent additional steps to be considered during challenging cases of hypertension particularly in young age group.

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