

Case Report

J Ped. Nephrology 2015;3(4):165-169
<http://journals.sbm.ac.ir/jpn>

A Rare Case of Childhood Hypertension and Hyponatremia

How to Cite This Article: Mazumdar J, Sarkar R, Pal S, Ghosh S, Kumar Das P. A Rare Case of Childhood Hypertension and Hyponatremia. J Ped. Nephrology 2015;3(4):165-169.

Jayitri Mazumdar,^{1*}
Rakesh Sarkar,²
Sumita Pal,³
Sukumar Ghosh,⁴
Pradip Kumar Das⁵

1 MBBS, MD Pediatrics, Senior Resident in Pediatrics, CNMC & H (Calcutta National Medical college & Hospital), Kolkata 700014

2 MBBS, MD Medicine, DM Cardiology (1st yr PDT), RGK Medical College & Hospital

3 MBBS, DGO, DCH, MD Pediatrics, Assistant professor, CNMC&H

4 MBBS, MD, Dip Card, DM Cardiology, Prof & Head, Dept of Cardiology, CNMC&H

5 MBBS, DCH, MD Pediatrics, Prof & Head, Dept of Pediatrics, CNMC&H

*Corresponding Author

Jayitri Mazumdar, MBBS
MD Pediatrics, Senior Resident in Pediatrics, CNMC&H
Email: jayidoc@gmail.com
Phone: 9230297392

Received: July-2015

Revised: Aug-2015

Accepted: Sep-2015

Hypertension and hyponatremia together, is an uncommon entity in children. We here described a 10-year-old boy presented with hypertensive emergency and altered sensorium with hyponatremia. After initial stabilization USG (ultrasonography) Doppler showed shrunken right kidney with absence of flow in the right renal artery. Right Renal Resistive Index was 0.9. Therefore, patient underwent right total nephrectomy and blood pressure ultimately came under control.

Keywords: Hypertension; Hyponatremia; Child.

Running Title: Childhood Hypertension and Hyponatremia

Introduction

Hypertension and hyponatremia together, is an uncommon entity in children. It occurs in renovascular hypertension due to increased activity of rennin angiotensin aldosterone axis [1]. In 1965, Brown et al. described HHS (hypertension hyponatremia sequence), a combination of severe resistant hypertension along with hyponatremia, polyuria, hypokalemia and proteinuria (sometimes in the nephrotic range) [2, 3].

Most patients reported were elderly, asthenic females, and in most patients the underlying renal artery pathology was atherosclerosis [2]. Malignant hypertension (HTN) as a presentation has been reported in adults with HHS, but is rare in children beyond infancy with few reports [3-5]. We here described a 10-year-old boy presented with hypertensive emergency and altered sensorium with hyponatremia.

Case Report

A 10-year-old boy was admitted in our hospital with headache since last 5 days, altered sensorium for last 2 days along with vomiting 3 times on the day of admission. Patient was apparently well 5 days before, thereafter he complained of headache, which was insidious in onset, initially moderate in intensity but assumed a peak within 3 days and persisted with the same intensity. Pain was throbbing and more in the occipital region. The headache was not relieved during sleep and hampered day to day activities. On the 4th day, patient had altered sensorium and only responded to painful stimuli. Patient was vomiting, which was projectile and non bilious. No history of fever, joint pain, rash, bleeding manifestation, oliguria, hematuria, generalized swelling, trauma or drug intake was present. No history of convulsion during this episode was noted. On general survey, GCS (Glasgow Coma Scale) was 9/15, patient responded only to painful stimulus and anthropometry measures were within the normal limits (height 135 cm, weight 25 kg). Pallor, icterus, clubbing, cyanosis and edema were not found, JVP (jugular venous pressure) was not raised, pulse 110/min, regular, normal volume, palpable in all four limbs, no radio-femoral delay, temperature normal, respiration normal pattern. BP: 190/120 measured in both upper limbs and 192/120 in lower limbs. (>5 plus 99th percentile for age & height percentile)

On examination, patient had altered sensorium, GCS 9/15 and cranial nerves, motor and sensory system were normal. Reflexes were normal, planter bilateral flexor, meningeal signs absent, pupil bilaterally equal and reacting, ophthalmoscopy examination normal and neurocutaneous markers absent. Cardiovascular system revealed apex at 5th space on the left mid clavicular line, ejection systolic murmur at pulmonary area, no thrill, S1 S2 audible, GI (Gastrointestinal system) system abdomen soft, no organomegaly, no ballotable mass. No bruit on abdominal auscultation, respiratory system within normal limit. Patient was initially treated with infusion labetalol (1mg/kg/hr) along with intravenous normal saline. Ondansetron was administered 0.2 mg/kg IV TDS, within first 12 hours and BP reduced to 160/90; within two days BP came down to 140/90 mmHg (>99th percentile for age & height percentile) and patient became conscious. Oral labetalol was started and other supportive managements performed. Hemoglobin level was 12.2 g/dL, total leukocyte count

7900/per microliter (mCL), platelet 170000/mCL, Urea: 24 ,Cr: 0.7 mg/dL, Na:125, K:3.5 mEq/L, Lipid profile normal, Urine analysis and culture all within normal limit.

Arterial Blood Gas (ABG) showed pH :7.405, pCo₂: 40, pO₂: 85, HCO₃:24, Spo₂: 97%, Anion gap:12, Chest X ray: normal, ECG: no abnormality, Echocardiography showed Grade III diastolic dysfunction with LVEF 48%, increased interventricular septal thickness (12 mm) without any anterior movement of septal cusp of mitral valve; all suggestive of hypertensive changes, serum C3, C4 normal, ANA (anti nuclear factor), CRP (C-reactive protein) negative. Ultrasonography (USG) showed right kidney shrinkage with a size 6.61 cm, left kidney was 9.91 cm with normal corticomedullary differentiation, not suggestive of renal parenchymal disease (Fig 1). USG Doppler showed critical occlusion of right renal artery with reduced systolic flow (Fig 2& 3) and renal resistive index (RI) was 0.9 and normal flow in the left renal artery with RI = 0.25

$$RI = \frac{\text{Peak Systolic Velocity} - \text{End Diastolic Velocity}}{\text{Peak Systolic Velocity}}$$

RI more than 0.8 indicates irreversible microvascular change in the kidneys. Renal angiography showed complete occlusion of the right renal artery and normal flow in all other branches of aorta (Fig 4, 5 & 6).



Figure 1. Shrunken Right Kidney in Ultrasound

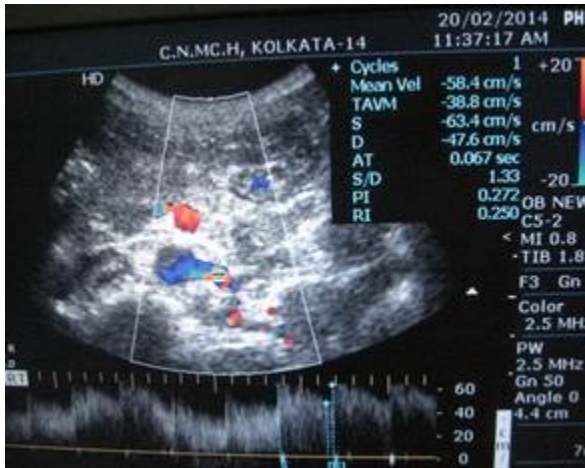


Figure 2. Normal Flow Pattern in the Left Renal Artery With RI 0.25

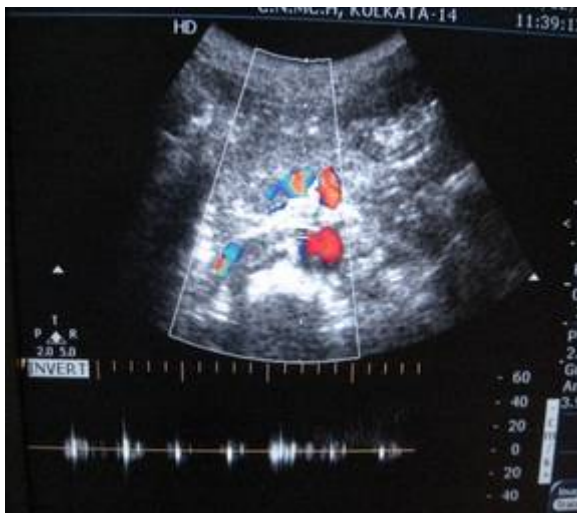


Figure 3. Reduced Flow in the Right Renal Artery With RI 0.9

BP was not controlled with labetalol tablet alone. Nifedipine was added orally. With these two drugs BP was 160/100. Patient was discharged initially with four antihypertensives; oral labetalol 100 mg BD, oral olmesartan 20 mg OD, oral chlorthalidone 6.25 mg OD and oral nicardia retard 10 mg BD. However, BP remained around 160/100. We initially consulted with vascular surgeon regarding reconstructive surgery but the most unique finding in our case was the presence of complete septa totally occluding the lumen of the right renal artery creating total cut-off in the

angiogram. Initial attempt for stenting was unsuccessful, as guide wire could not be negotiated through the septal occlusion.



Figure 4. Angiography Showing Complete Occlusion of the Right Renal Artery



Figure 5. Normal Aorta With its Branches

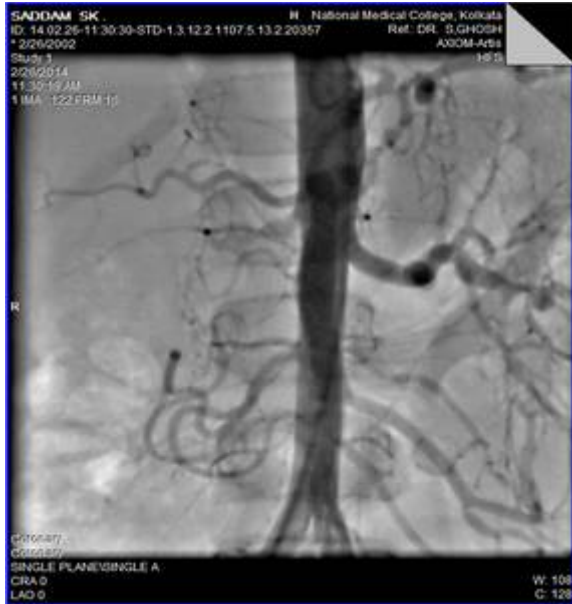


Figure 6. Complete Occlusion of the Right Renal Artery With no Abnormality in Other Branches of the Aorta

Furthermore, RI was more than 0.8, which indicated permanent microvascular damage difficult to be salvaged by vascular reconstruction. Hence urosurgeon planned for right total nephrectomy. Patient underwent right sided total nephrectomy and discharged on oral anti-hypertensive therapy (tab Labetalol 100 mg bd, tab olmesartan 20 mg od, tab chlorthalidone 6.25 mg od). After six months of follow-up, patient was only receiving oral labetalol and chlorthalidone and BP was 110/70 (<90th percentile for age & height).

Discussion

In 1965, Brown et al. described HHS (hypertension hyponatremia sequence), a combination of severe resistant hypertension along with hyponatremia, polyuria, hypokalemia and proteinuria (sometimes in the nephrotic range) [2, 3]. Most patients reported were elderly, asthenic females, and in most patients the underlying renal artery pathology was atherosclerosis [2]. Malignant hypertension (HTN) as a presentation has been reported in adults with HHS, but is rare in children beyond infancy with few reports [3-5]. Table 1 represents cases with HHS beyond infancy reported till date. In addition, HHS can be present as a paraneoplastic

manifestation of tumors such as rennin producing leiomyosarcomas [5]. The classic presentation of HHS includes hyponatremia associated with hypertension (systolic blood pressure more than 165 mmHg and diastolic more than 95 mmHg with or without anti-hypertensive medication) with evidence of renal ischemia [2]. Pathognomonic laboratory finding is an elevated rennin level, which is secondary to renal ischemia. As in our patient, if the renal ischemia is unilateral, elevation in systemic blood pressure can induce pressure natriuresis via the normal kidney leading to hyponatremia and volume depletion. The presence of volume depletion, in turn leads to a further increase in renin production. Similarly, increased renin would increase production of angiotensin II and thus aldosterone leading to persistent hypokalemia. A persistently elevated level of angiotensin II can directly stimulate the thirst center in the brain leading to increased production of antidiuretic hormone (ADH), which explains resistant dilutional hyponatremia [6]. Fibromuscular dysplasia is one of the most common pathological lesions in (renal artery stenosis) RAS in children [3], but the unique finding in our case was total occlusion of the right renal artery lumen by a septa. The patient developed cough after initial therapy with ACE inhibitor, hence we prescribed olmesartan orally along with other drugs and as the Right renal resistive index was >0.8, we decided to do right total nephrectomy.

Table 1. Cases With HHS Beyond Infancy Reported Till Date

Authors	year	No. of cases	Etiology	Outcome
Mazumdar et al.	Current study 2015	one	Complete occlusion by septum	Nephrectomy
Dixit et al.	2004	one	Left renal arterial (3) stenoses	Successful renal auto-graft
Dahlem et al.	2000	one	Fibro-muscular dysplasia of the left renal artery	Nephrectomy
Kaneko et al.	1994	one	Left renal artery stenosis	Percutaneous transluminal angioplasty
Misiami et al.	1994	one	Renin-producing LMS*	Removal of the LMS*; relapse

LMS: leiomyosarcoma

After nephrectomy, rennin and aldosterone levels decreased and BP became within normal limit. Similar encouraging results after nephrectomy reported before in four similar patients [3, 6]. Though uncommon in children, combination of hyperreninemic malignant HTN with hyponatremia (HHS) does occur and excluding RAS is strongly recommended. The purpose of this case report was to build awareness to not miss RAS in children.

Conflict of Interest

None declared

Financial Support

None declared

References

1. Tasian GE, Copelovitch L. Evaluation and medical management of kidney stones in children. *J Urol.* 2014 Nov; 192(5):1329-36.
2. Balestracci A, Battaglia LM, Toledo I, Idiopathic hypercalciuria in children with urinary tract infection. *Arch Argent Pediatr.* 2014 Oct; 112(5):428-33..
3. DeBerardinis RJ, Coughlin CR 2nd, Kaplan P. Penicillamine therapy for pediatric cystinuria: experience from a cohort of American children. *J Urol.* 2008 Dec; 180(6):2620-3.
4. Aydogdu SD, Kirel B, Coskun T, Kose S. Prevalence of cystinuria among elementary schoolchildren in Eskisehir, Turkey. *Scand J Urol Nephrol.* 2009; 43(2):138-41.
5. Halperin EC, Thier SO, Rosenberg LE. The use of D-penicillamine in cystinuria: efficacy and untoward reactions. *Yale J Biol Med.* 1981 Nov-Dec; 54(6): 439-446.
6. Seyedzadeh A, Momtaz HE, Moradi MR, Moradi A. Pediatric cystine calculi in west of Iran: a study of 22 cases. *Urol J.* 2006 Summer;3(3):134-7; discussion 138..
7. Asanuma H, Nakai H, Takeda M, et al. [Clinical study on cystinuria in children--the stone management and the prevention of calculi recurrence]. *Nihon Hinyokika Gakkai Zasshi.* 1998 Sep;89(9):758-65..
8. Tangnararatchakit K, Ariyaprakai W, Tapaneya-Olarn W, et al. Cystinuria: cause of recurrent renal stones in a 4-year-old girl. *J Med Assoc Thai.* 2002 Nov;85 Suppl 4:S1281-6.