Case Report

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Hyponatremic Hypokalemic Natriuresis in a Child with Malignant Hypertension and Abdominal Tuberculosis

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Abstract

Pediatric hypertension is not uncommon in children; yet, it is very commonly missed by primary physicians and end organ damage has already started by the time it is diagnosed. Hypertension is sometimes considered a consequence of an illness when it actually is the etiology of that illness, for example, intracerebral hemorrhage with raised intracranial pressure.

Keywords: Hypertension; Hyponatremia; Hypokalemia.

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Introduction

Hypertension is seen in 2-5% of pediatric patients (1). It is very commonly missed by primary physicians and therefore organ damage has already started by the time it is diagnosed. The recently reviewed clinical practice guidelines by the American Academy of Pediatrics have defined normal, elevated, stage 1 and 2 hypertension as follows (2). The most common cause of hypertension in children is secondary hypertension, among which renal parenchymal disease and renal artery stenosis are particularly important. Other important causes include coarctation of the aorta, endocrine causes like renin secreting tumors, etc (3) Renal artery stenosis is an important secondary cause of hypertension in children. We present a complex case of abdominal tuberculosis with congenital vascular anomalies of the aortic arch and double left renal artery with stenosis at ostium.

Case Report

A 4-year-old child was admitted to our ward with a history of polydipsia, polyuria, constipation, failure to gain weight, and abdominal distension. He had persistent vomiting for the last three days prior to admission. His father was a known case of pulmonary tuberculosis and had completed his anti-TB treatment three months ago. He was the only child of his parents. On examination, the child had pallor, severe acute malnourishment (weight and height below the 3rd percentile) and abdominal distension with no organomegaly. He had tachycardia, tachypnea and stage 2 hypertension. His blood pressure was 144/112 mmHg in the right upper limb, 108/70 mm Hg in the left upper limb, 148/100 mmHg in the right lower limb, and 142/94 mmHg in the left lower limb. On cardiovascular examination, a grade III pansystolic murmur was auscultated in the parasternal region. All peripheral pulses were palpable. Other systemic examinations were

unremarkable. He had a hemoglobin level of 9.6 g/dl and total leucocyte count of 11.15×10^3/microL (P-58%, L -40%, M-2%). His general hematological profile was suggestive of microcytic hypochromic anemia. The renal function tests were essentially normal (serum urea= 26 mg/dl, serum creatinine=0.28 mg/dl). Serum sodium was 130 mmol/L, serum potassium was 2.90 mmol/L, and serum calcium was 9.10 mg/dL. His random blood sugar was 88mg/dl and liver function tests were normal. CRP was negative, and blood and urine cultures were sterile. ABGA was advised in view of polyuria, polydipsia and hypokalemia. Blood pH was 7.48 and HCO3−was 38.5 mEq/L. Urine routine microscopy showed trace proteinuria with an acidic pH of 6. No WBC, RBC, or casts were seen in urine routine microscopy. Abdominal ultrasound showed a small left kidney (38x20 mm) with raised echotexture. Tuberculin test with 2 TU was positive (20x10mm). His gastric aspirate samples were negative for acid fast bacilli. Two-dimensional echocardiography was suggestive of dilated cardiomyopathy with a poor ejection fraction (30%). Endocrine workup was done in view of stage 2 hypertension, hyponatremia and hypokalemia,. Thyroid function tests were normal. Plasma renin activity was >24 ng/ml/hr and serum aldosterone was > 200ng/dl. Stage 2 hypertension, hypokalemia, a small left kidney, and elevated levels of plasma renin and aldosterone pointed towards a renovascular cause of hypertension. Hence, an abdominal CT scan with contrast enhancement was done, which revealed a small left kidney (38X20mm) with two left renal arteries that were both severely attenuated at the ostium. In addition, there were multiple necrotic and calcified abdominal lymph nodes, some of which showed calcification. There were few subcentric hypodense lesions (probably hemangioma) in the splenic parenchyma. A CT angiography of the thorax and abdomen was done (Figure 1), which revealed a bovine arch characterized by the left common carotid artery arising from the brachiocephalic trunk with a severely attenuated left renal artery. 99mTc- DTPA renal scan showed a non-functioning left kidney and a normal functioning right kidney. Hence, the patient was a malnourished pale child with congenital vascular anomalies of the aortic arch and renal arteries with left renal artery stenosis and a non-functioning left kidney. Secondary to renal artery stenosis, he developed stage 2 hypertension and dilated cardiomegaly. The central mechanism for hypertension was the stimulation of the renin-angiotensin-aldosterone axis, which triggered vasoconstriction as well as fluid and salt retention. In addition, he had abdominal tuberculosis. He was managed with antihypertensive agents, potassium supplementation, digoxin, and nutritional supplementation. Anti-TB drugs also started in view of abdominal Koch's. His blood pressure was gradually controlled left nephrectomy was advised.

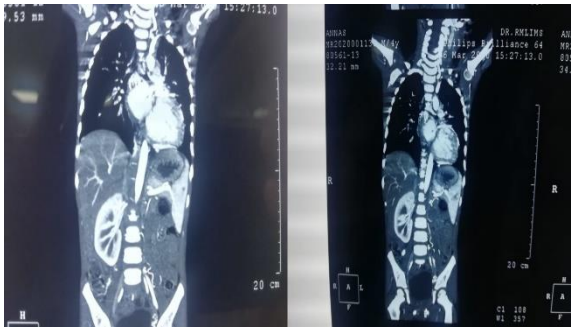

Figure1. CT angiography of thorax and abdomen

Discussion

Our case had hypertension and hypokalemia in presence of metabolic alkalosis. There have been occasional case reports of renal artery stenosis associated with hypertension, and the majority present with hyponatremic hypertensive syndrome (4-7) Unilateral renal artery stenosis results in natriuresis of the non-stenotic kidney; hence, the child will present with polyuria, polydipsia, failure to thrive, hypertension, hypokalemia, and proteinuria. In fact, some authors recommend that if hyponatremia and hypertension are found in any child, renal artery stenosis should be suspected (7). Kovalski Y et al (4) reported four cases similar to our case who presented with polyuria, polydipsia, metabolic alkalosis, and nephrotic range proteinuria. All of them had underlying renal ischemia, which was corrected by percutaneous angioplasty. Dixit et al (5) reported hyponatremic hypertensive syndrome in an 18-month-old child who had three left renal arteries but all of them stenosed.

Similarly, Seracini D et al reported a 15-month-old girl with hyponatremic hypertensive syndrome caused by stenosis of the left renal artery. She also improved after percutaneous angioplasty. Our patient had hyponatremic hypokalemic natriuresis with malignant hypertension. In addition to renal artery stenosis, the child had other congenital vascular malformations as well. His abdominal Koch's further complicated his clinical course. In conclusion, if metabolic and electrolyte abnormalities like hyponatremia and hypertension are found in a child, a renovascular cause of hypertension should be suspected.

Conclusion

We presented a 4-year-old boy with polyuria, polydipsia, abdominal distension, and vomiting. He was diagnosed with congenital bovine variant of aortic arch and stenosis of double left renal arteries leading to malignant hypertension. In the majority of pediatric hypertension cases, early detection and management are key to survival. A renovascular cause of hypertension must be suspected in any child with hyponatremia hypokalemia and hypertension.

Conflict of interest

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