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CASE REPORT

CRS-IV (Chronic Renocardiac syndrome) in a female child. Heart and Kidney Dysfunction intertwined.

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ABSTRACT… Renal artery stenosis is the major cause of renovascular hypertension, can be found isolated or have association with syndromes or autoimmune conditions. It can be due to atherosclerotic deposition in vessels, autoimmune etiology or fibromuscular dysplasia, the latter is the commonest cause, however, needs biopsy for diagnosis and exclusion of other possible causes. Patients can have diverse presentation, disease may be discovered as incidental finding, with hypertensive crises, Nephropathy or cardiac manifestations. We report a case of 7 years female patient presented with severe respiratory distress, hypertension, Grade 1 BL pitting edema, hepatomegaly, basal crackles in chest and gallop. He had no history of any diagnosed medical condition prior. However, symptoms were evident for last 2 months with Headache, fatigue, cough, exertional dyspnea, edema and decreased urine output. She had no history of arthralgia, frothy urine, hematuria or any skin rashes. Evaluation for Hypertension revealed small sized right kidney with decreased perfusion in segmental arteries. Echocardiography showed Biventricular dilation, dysfunction with Ejection fraction of 40%. She initially managed with iv labetalol infusion later started enalapril and amlodipine then referred to specialty where percutaneous transangioplasty planned abut deferred by pediatrics nephrology as patient responded with medical management and it is considered for resistant hypertension or persistent high renin and aldosterone. The case concluded with diagnosis of Reno cardiac type-iv syndrome, as manifestations are concordant with chronic kidney disease – right SSK due to hypoperfusion that resulted in renovascular hypertension and Cardiac failure.

Key words: Hypertension, Renal Artery Stenosis, Congestive Cardiac Failure.

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INTRODUCTION

Hypertension in children is secondary most commonly linked to renal then endocrine disorders.[1] Renal artery stenosis however rare, can progress to Cardiorenal Syndrome (CRS) causing cardiac distress and has a significant mortality risk. Majority of these lesions are fibromuscular in nature arising from second and third renal artery branches.[1] Other causes of Renal Artery Stenosis reported include Atherosclerosis and Vasculitis.[2] Although, its mostly asymptomatic, the most common manifestation is severe hypertension.[2,3] Cardiorenal syndrome are the co-existence of cardiac and renal pathophysiology and can present with either acute or chronic failure of either cardiac or renal functions.[4] In 2008, five different manifestations of CRS were classified by Acute Disease Quality Initiative (ADQI).[5] Type I and II describes Renal dysfunction originating from cardiac failure, where type I refers to Acute heart failure resulting in Acute Kidney Injury (AKI) while Type II includes chronic heart failure leading to renal failure. Type III, also referred as Acute Reno-Cardiac Syndrome, refers to AKI causing acute heart failure. On the other hand, Type IV is referred as Chronic Reno-Cardiac Syndrome, where a chronic kidney disease results in heart failure. Type V is acute failure of both cardiac and renal functioning secondary to a systemic cause.[4,5] In this study we present a case of CRS Type IV in an 7-year-old female.

CASE PRESENTATION

We report a case of 7 years female patient weighing 16 kg, height of 116 cm at 10th centile presented in emergency with severe respiratory distress getting worsen with lying supine. She was Hypertensive BP 170/110 mm Hg.

Her symptoms became significantly evident for last 2 months with headache that significantly impaired her school learning temporarily relived with

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analgesics however she remained fatigue most of time and had persistent dry cough throughout day and night aggravating during sleeping and getting her awake from the sleep as well, she experienced exertional dyspnea and was not able to walk longer, play with kids, developed edema for last 1 month and decreased urine output was noted for last 2 days before presenting in our emergency.

At Presentation she was tachycardiac, tachypneic, severely anemic, with grade 1 pitting edema, hepatomegaly, basal crackles, hyper precordium, displaced apex beat at 6th intercostal space lateral to midclavicular line, there was gallop but no murmur was audible.

She stabilized with keeping her elevated at 45 degrees to decrease cardiac load, IV labetalol infusion started with intermittent Lasix doses (0.5mg/kg/dose x 6 hourly), switched to oral enalapril, amlodipine, Lasix and spironolactone after 36 hours as her condition stabilized and she weaned off from oxygen as well.

It was noted as her blood pressure improved her urine output improved as well.

She was investigated for hypertension, 4 limb BP does not show any change, urine DR did not show any sediments, US KUB with Doppler showed Right small sized kidney with size 3.4 x 4.1 and reduced perfusion in segmental arteries left sided kidney was normal in size with normal Doppler flow.

Her echocardiography done that was suggestive of biventricular dilation, dysfunction with poor contractility and ejection fraction of 40%, no structural deformity noted in valve or cardiac structure.

She had no history of arthralgia, frothy urine, hematuria or any skin rashes and clinically there were no evidence of any xanthomas. Her Baselines, including lipid profile, autoimmune markers were normal except severe anemia that stabilized with PCV transfusion, reports are summarized in Table-I.

Among possible causes of RAS, she was suspected case of fibromuscular dysplasia as autoimmune workup was also negative, but patient need Renal biopsy for the confirmation of diagnosis of FMD, it was not performed as intervention will not change the management, histopathology is done if nephrectomy is done or there is conflict in some mixed disorder.

Patient's CT angiogram findings were consistent with Doppler ultrasonography. Her percutaneous transangioplasty dilation is deferred as Blood pressure normalized with enalapril and amlodipine, renin and aldosterone were also decreased in follow up as mentioned in Table-II.

She is kept in follow up. Her blood pressure is normalized and anti-failure medicines for cardiac care are continued by cardiologist with follow up with echocardiography imaging is advised after 3 months.

DISCUSSION

Coexisting cardiac and kidney failure is referred to as cardiorenal syndrome (CRS), which affects up to 50% of persons overall.[6,7] The idea of CRS was developed to span a range of illnesses in which primary acute or chronic malfunction in one organ causes acute or chronic dysfunction in the other, emphasizing "organ crosstalk" and the special interaction between the heart and kidneys. The current conceptual description of CRS was provided by the Acute Disease Quality Initiative (ADQI) in 2008[8,9] and was divided into five subtypes: acute cardiorenal, chronic cardiorenal, acute Reno cardiac, chronic Reno cardiac and secondary to systemic disorders.

According to Pradhan SK et al.'s systemic review, type 4 CRS is the least frequently reported in children, with just 7% of papers describing combined type 4 and 5 CRS. Chronic renal dysfunction is the primary pathophysiology of type 4 CRS (CKD), resulting in ventricular hypertrophy, cardiac dysfunction, and a higher chance of cardiovascular events. The kidneys and the heart interplay are bidirectional; chronic kidney disease (CKD) causes a series of pathological changes that eventually affect cardiac function.[10] Renal artery stenosis (RAS) is a significant contributor to chronic kidney disease (CKD), with various studies highlighting

its prevalence and impact. Research indicates that About 18% of patients with chronic kidney disease (CKD) have RAS, compared to 6% of people without CKD, and 22.9 percent of patients had RAS more than 50%. Although RAS is a known cause of chronic kidney disease (CKD), it is important to remember that other variables, such as diabetes and hypertension, also significantly contribute to the progression of kidney disease.[11-13] Unilateral RAS causes activation of renin-angiotensin-aldosterone system (RAAS), natriuresis from opposite kidney and suppression of its renin secretion. Angiotensin II thus formed, also results in secondary hypertension due to vasoconstriction. Up to 30% reduction in renal blood flow can be adapted by renal cortex and medulla without development of severe hypoxia. 70 to 80% stenosis of vascular lumen causes marked cortical hypoxia leading to activation of inflammatory cascades that eventually lead to chronic ischemic injury and interstitial fibrosis.[2,3]

Doppler ultrasound (US KUB) remains non-invasive and valuable initial modality for detecting RAS, as was utilized in our case.[15] Initial management essentially involves antihypertensives to stabilize blood pressure and reduce cardiovascular complications. PTRA with stenting has been stabilized as an effective therapy in children restoring perfusion and improving hypertension control, in refractory cases.[2]

In our patient renovascular hypertension (RVH) and ischemic nephropathy due to renal artery stenosis, interact intricately leading to Bi-ventricular hypertrophy and a reduced ejection fraction. While RAS is a well-documented cause of secondary hypertension in pediatric population, its presentation with left ventricular hypertrophy, symptomatic heart failure having respiratory distress at such a young age is relatively uncommon. Our case underscores the need for early cardiovascular assessment in hypertensive children to prevent irreversible myocardial damage. Renal artery stenosis should be considered in children with this clinical presentation.

CONCLUSION

CKD has diverse presentation, stage-1 may be diagnosed incidentally with renal structural malformation or SSK or patient may land with stage-5 complications and need urgent dialysis. This patient labelled stage 1 CKD with RSSK, normal RFTs is good prognosis at time of presentation. However RAS presentation with cardiac failure is rare which was due to long standing chronic hypertension. Multimodal management, including medical stabilization and interventional therapy, plays a crucial role in improving prognosis. Regular follow-up is necessary to assess long-term renal and cardiac outcomes and need of surgical intervention.

Legends

FIGURE-1

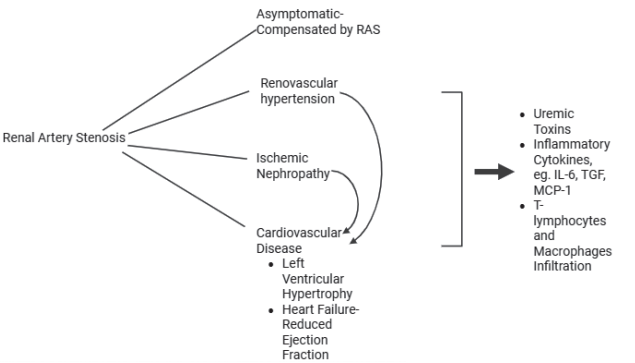

TABLE-I

Hemoglobin (g/dL)	6	9	BUN (mg/dL)	12	Total (mg/dL)	125
Mean Corpuscular Volume (fL)	69	72	Cr (mg/dL)	0.2	Triglyceride (mg/dL)	135
Total Leukocyte Count (L)	4.9 x 10^{-6}	5.2 x 10^{-6}	Na (mEq/L)	136	Serum HDL	50 mg/dl
Neutrophils (%)	56	50	K (mEq/L)	3.4	Serum LDL	110 mg/dl
Lymphocytes (%)	36	40	Cl (mEq/L)	102	triglyceride	135 mg/dl
Platelets (x 10^9)	162	182	HCO_3 (mEq/L)	22		

TABLE-II			
-	At Admission	Follow-up	Normal Range
Serum Renin (ng/L)	41	32	1.9-3.7
Serum Aldosterone (ng/L)	149	137	<37

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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AUTHORSHIP AND CONTRIBUTION DECLARATION	
1	**Aisha Haleem:** Conceptualization.
2	**Amanullah Lail:** Manuscript editing.
3	**Aamina Shaikh:** Data analysis.
4	**Noor Fatima Suri:** data collection.
5	**Laraib Ghangro:** literature review.