

Acute renal failure in a child with Kawasaki disease

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Abstract. Renal manifestation of Kawasaki disease is not frequently described. A four-year-old boy was referred to our institute with acute renal failure for dialysis. Detailed history coupled with physical examination revealed telltale symptom complex of Kawasaki Disease. We diagnosed the case to be Kawasaki Disease complicated by Acute Renal Failure at presentation. The patient recovered with supportive measures alone without requiring renal replacement therapy. Acute renal failure is a rare accompaniment of Kawasaki Disease as presenting manifestation.

Key words: Kawasaki disease, acute renal failure

1. Introduction

Kawasaki Disease (KD) is an acute vasculitic syndrome of childhood characterized by fever, rash, conjunctival infection, erythema and swelling of hands and feet and cervical lymphadenopathy. It was first described by Tomisaku Kawasaki in 1967 (1-3). Apart from these, certain unusual manifestations have also been described in literature (4,5). But, renal manifestations of KD has been rarely reported in literature (6-8). We report one such case of KD presenting with acute renal failure.

2. Case report

A four years old boy was referred to our hospital which is a tertiary care referral hospital in eastern India for management of acute renal failure. History revealed that the boy was suffering from high-grade spiky fever for last seven days with associated irritability. He developed oliguria for the last two days. There was no history of intake of any medication likely to cause renal failure. The child did not have any history of abdominal pain and diarrhea.

On examination, the child was found to have extreme irritability with bilateral conjunctival infection, swollen and red lips and tongue, postauricular lymphadenopathy and subcutaneous edema of dorsum of hand. His anthropometry was within normal limits.

The blood pressure was elevated to 102/68 mm of Hg (> the 99th percentile). The child was diagnosed as suffering from Kawasaki disease with renal failure. He developed Beau's line over the nails and desquamation of hands and feet during hospital stay.

Complete hemogram showed hemoglobin of 10.0 gm/dL, total leukocyte count of 14800/mm³ with polymorph 84% and lymphocyte 14%, ESR 96 mm/1st hr and platelet count of 240.000/mm³. Repeat platelet count after two days was 600.000/mm³. His chest X-ray was normal.

Electrocardiogram demonstrated sinus tachycardia. Echocardiography revealed normal cardiac function and there was no evidence of coronary aneurysm. His initial renal biochemistry was deranged with serum urea 128 mg/dL, BUN 58 mg/dL and creatinine 3.2 mg/dL. His BUN/creatinine ratio was 18.1. He had a normal serum electrolyte study (sodium 133 meq/L and potassium 3.5 meq/L). Urinalysis showed 5-18 white blood cells/HPF and a few cellular casts. Urine culture was sterile. Urinary sodium was 36 meq/L, urinary creatinine 56 mg/day, urinary specific gravity was 1.010 and urine osmolality was 294 mos/L. Calculated fractional excretion of sodium (Fe Na) was 1.54 suggestive of intrinsic renal failure. Bilateral acute renal parenchymal diseases with increased cortical echogenicity with loss of cortico-medullary differentiation were documented in ultrasound scan without any enlargement of mesenteric lymph node. His ASO titer, C-reactive protein, C3, C4 and ANA were within normal limits.

The patient was diagnosed as Kawasaki disease with acute renal failure. He was put on empirical parenteral antibiotic, aspirin in anti-inflammatory dosage and supportive management including maintenance of fluid and electrolyte homeostasis.

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Financial reasons precluded use of intravenous immunoglobulin. Renal failure was managed conservatively. The patient showed improvement in renal function with urine output increasing within two days and azotemia receded gradually. There was no need for renal replacement therapy. He also became afebrile subsequently and discharged on anti-platelet dose of aspirin. Repeat echocardiography after 6 weeks was also normal. However, with the child improving, the family did not give consent for kidney biopsy.

3. Discussion

Since it was described for the first time (1) several complications have been recognized in context to Kawasaki Disease. The main disease burden, indeed, is due to the coronary sequelae of this condition (2-4). However, several extracardiac complications may worth attention. Renal disease is one of them.

Available literature (5) described meatitis, dysuria and sterile pyuria as the main complications relating to the genitourinary system. A few cases of acute renal insufficiency, however, have been reported (6-12) from all over the world. One case even masqueraded as acute pyelonephritis (13). The precise mechanism of renal damage remains unknown because in most cases histopathological alteration could not be explored with renal biopsy. Second, and perhaps more important in our context, no such complication has been reported in cases reported from India till date (14-16).

Several hypotheses were put forward for explaining this renal insufficiency. It has been said in one case that loss of fluid to the extravascular compartment and consequent drop in renal perfusion was responsible for the kidney injury (17). Histopathological examinations in some cases reveal an association of interstitial nephritis with KD (18). Drug induced interstitial nephropathy could also be the cause, as demonstrated by histopathological examination in one case (19). A British group of investigators, earlier, suspected that their patient with KD had passed into renal failure due to vasculitis but histological evidences were not available (20). One report from New Jersey suggested immune complex damage to the kidney on biopsy as possible mechanism of nephrotoxicity (21). With increased use of IVIG, there remains a thin but definite risk of acute kidney injury (22). In Kawasaki disease, the renal disease characteristically runs a benign course. Barring a few cases (11), most of these children completely recovered, often without needing renal

replacement therapy (8). A Turkish study group has held Kawasaki disease responsible for renovascular hypertension in 2 % of cases in a nationwide survey (23). An old report of renal involvement with increased cortical echogenicity and enhanced cortico-medullary differentiation on sonographic evaluation was described in 1985 (24). Kawasaki syndrome associated with *Yersinia* infection is a close mimicker of Kawasaki disease and are sometime associated with renal failure (25). Our case did not have the classical symptomatology and mesenteric lymphadenopathy characteristic of *Yersinia* infection. In our case, we could exclude pre-renal cause of renal failure by urinary studies and USG showing bilateral renal parenchymal disease. As C3 and C4 were normal, immune complex mediated disease was not a possibility. Our case had renal failure due to tubulo-interstitial nephritis following Kawasaki disease.

4. Conclusion

To the best of our knowledge, this is probably the first case of Kawasaki disease described from India with acute renal insufficiency at presentation.

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