A Case Report of Kawasaki Disease with Manifestations of Acute Renal Failure

Seyed Mohammad Reza Razavi1, Mohammad Azadegan1, Sajjad Rezvan3, Enayatollah Noori4*

1 Department of Pediatrics, Faculty of Medicine, Qom University of Medical Sciences, Qom, Iran
2 Larestan University of Medical Sciences, Larestan, Iran
3 Department of Radiology, Rafsanjan University of Medical Sciences, Rafsanjan, Iran
4 Research Committee of Qom University of Medical Sciences, Qom, Iran

* Corresponding author: Enayatollah Noori, Research Committee of Qom University of Medical Sciences, Qom, Iran. Tel: 09195952857; Email: enoori@muq.ac.ir

Abstract

Background: Kawasaki disease is an acute febrile multisystem vasculitis that affects medium- or small-sized arteries in patients, especially children under 5 years of age. Kawasaki disease is a systemic vasculitis that can affect several organs. Renal manifestations of this disease are rarely reported in the form of sterile pyorrhea and proteinuria trace in articles. In the current article.

Case Report: we report a case of Kawasaki disease in patient 18-month-old boy with no history of specific illness with manifestations of acute renal failure.

Conclusion: Kawasaki’s diagnosis has been challenging for physicians due to the atypical manifestations of the disease. A definitive diagnosis may not be possible with the initial manifestations. Renal disease as a primary manifestation of Kawasaki is not common and has only been reported in a previous case with a different etiology.

Keywords:
Acute kidney failure
Demonstration
Kawasaki

Introduction

Kawasaki disease is an acute febrile multisystem vasculitis that affects medium- or small-sized arteries in patients, especially children under 5 years of age (1). This disease presents with fever and subsequent vasodilation and inflammation of the mucous membranes (2). The course of this disease can be divided into three clinical phases: acute, subacute, and convalescent (3). The acute phase characterized by fever lasts 1-2 weeks and is accompanied by signs and symptoms of fever, conjunctivitis, erythema of the oropharyngeal mucosa, erythema and indurative edema of the hands and feet, rash, cervical lymphadenopathy, aseptic meningitis, diarrhea, and renal dysfunction (4). Myocarditis, pericardial effusion, and coronary arthritis may also occur at this stage, while echocardiography usually does not show any aneurysms (4).

The acute phase of the disease begins 2 weeks after the onset of fever, with a decrease in lymphadenopathy and rash. In this stage, irritability, anorexia, and conjunctivitis persist. Moreover, the skin peels on fingers and toes and thrombocytopenia occur: The risk of sudden death at this stage is high due to coronary artery aneurysms. The acute phase usually lasts two weeks. The convalescent phase begins when all clinical signs of the disease have resolved and persists until the erythrocyte sedimentation rate (ESR) returns to normal 6-8 weeks after the onset of the disease (3). Classic Kawasaki disease is identified by the following diagnostic criteria.

One criterion is a high fever which lasts for 5 days or more with at least four symptoms (bilateral...
conjunctival hyperemia, lip ulcers and inflammation in the oral cavity, polymorphic rash, edema and scaling of the limbs, and neck lymphadenopathy). Another criterion is when the fever is accompanied by less than the four above-mentioned; nonetheless, patient echocardiography indicates coronary artery disorders. Kawasaki disease is a systemic vasculitis that can affect several organs. Renal manifestations are rarely reported in the form of sterile pyorrhea and proteinuria trace in articles (3, 5-8). In this article, we report a case of Kawasaki disease with the manifestations of acute renal failure.

**Case Report**

The patient was an 18-month-old boy with no history of specific illness who had developed fever five days earlier, vomited two days later, and had visited a doctor. The patient had undergone initial treatment with androsterone and dexamethasone. He had recovered from the vomiting; however, he later developed edema around the eyes, sensitivity to light, and discharge from both eyes. The patient also developed oliguria 2 days before hospitalization. On examination, the patient appeared to be unwell but non-toxic with a high fever. The patient’s respiratory rate was more than usual. Signs of dehydration were detected. The patient’s axillary temperature was reported as 37.5°C.

Other vital signs were as follows: respiration (breathing rate)=30, pulse rate=128, and blood pressure=140.70 mm Hg. No skin rash was observed. The swelling was detected around his eyes. The patient had pharyngeal erythematic and tonsillar hypertrophy. The left ear canal and tympanic membrane were edematous. Head and Neck Lymphadenopathy was not reported. During lung auscultation, wheeze or crackles (rales) were not heard. The patient's heart rate was normal, with a sharp S1 and S2 and no other significant sounds and heart murmur. The patient's abdomen was soft and did not have an organomegaly. The patient's lower limbs had non-pitting edema++. The patient's height and weight were normal in the range of 150.

The patient’s initial tests are demonstrated in Table 1:

<table>
<thead>
<tr>
<th>Table 1. Patient's initial tests</th>
<th>CBC</th>
<th>Biochemistry</th>
<th>Urine Analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBC: 13100/mm³</td>
<td>Na: 130 mEq/L</td>
<td>Blood: 3+</td>
<td></td>
</tr>
<tr>
<td>PMN: 71.5%</td>
<td>K: 5.6 mEq</td>
<td>Pr: 2+</td>
<td></td>
</tr>
<tr>
<td>LYM: 15.9%</td>
<td>Urea: 255 mg/dl</td>
<td>Glu: 1+</td>
<td></td>
</tr>
<tr>
<td>PLT: 120000/mm³</td>
<td>Cr: 5.4 mg/Dl</td>
<td>WBC: Many</td>
<td></td>
</tr>
<tr>
<td>HB: 11.5 g/dl</td>
<td>ESR: 117 mm</td>
<td>RBC: Many</td>
<td></td>
</tr>
<tr>
<td>CRP: 51.2 mg/L</td>
<td>CRP: 117 mm</td>
<td>epithelial Cell</td>
<td></td>
</tr>
</tbody>
</table>

Ultrasound showed normal kidneys, increased parenchymal echo, and diminished corticomedullary differentiation. Considering patient sign, including high creatinine, oliguria, edema, and vomiting, our initial approach was acute renal failure, including liquid therapy and antibiotic therapy (ceftriaxone and vancomycin). Moreover, the patient received Foley catheter and LASIX injection for oliguria. The child’s fever decreased after the initial procedure, the edema of the limbs decreased, and the patient’s blood pressure was reported to be within 100-120 mmHg. The child’s urine output during the first night of hospitalization was 65 cc (more than the minimum required). Sulfacetamide drops of 20% and ocular erythromycin ointment were prescribed for exudative conjunctivitis.

After about 9 days of treatment, the patient’s creatinine was reduced to 2.2; nevertheless, he had leukocytosis (WBC: 23400) and fever in complete blood count (CBC). It was initially thought that endocarditis may be the cause; on the other hand, due to the history of high fevers and high ESR, Kawasaki disease was suspected. On suspicion of Kawasaki, the patient received 22 grams of IVIG, 3 drops per minute, and 1-drop increase every 15 minutes. After 3 days of treatment, the patient’s general condition improved and the patient’s fevers stopped. The patient underwent echocardiography which demonstrated good heart function and good coronary artery size. A Control echocardiography was requested for the patient 1 month later. The child was discharged with aspirin antiplatelet therapy and Cardiology Department outpatient follow-up. An echocardiogram of the patient a year later showed normal heart function.

**Discussion**

Kawasaki disease is a multi-system disease with unknown etiology. It is assumed that this disease may be a reaction of the immune system to infection with genetic predisposition as the atopic disease is (9). Recent studies have attempted to identify a number of inflammatory genes that are responsible for driving the development of this disease (10). Therefore, since it is a multi-system ailment, Kawasaki disease may mimic the symptoms of other diseases. Several complications, such as coronary artery aneurysm involvement, are identified for this disease which is the most common cause of acquired pediatric heart disease in children in developed countries, such as Japan and the United States (11, 12). However, many adverse Noncardiac complications have been reported for this disease, including kidney disease. According to studies, kidney involvement in Kawasaki is rare except for sterile pyorrhea and proteinuria trace.

acute renal impairment due to tubulointerstitial nephritis, nephropathy mediated by immune complex, acute nephritic syndrome, renal tubular

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dysfunction, renal impairment on imaging, and renal arterial disease are among the recognized disorders in Kawasaki (13). Acute renal failure has been reported as a common complication in hospitalized children and as an uncommon complication in patients with Kawasaki (13). The development of this complication in the course of Kawasaki disease has been ascribed to some reasons, namely Acute Kidney Injury (AKI), prerenal associated with acute heart failure, intrinsic AKI due to tubulointerstitial nephritis, acute nephritic syndrome, Hemolytic uremic syndrome, immune complex-mediated nephropathy, Rhabdomyolysis, and Kawasaki disease shock syndrome (13).

AKI is defined as an increase in serum creatinine to 1.5 times baseline according to guidelines (14-16). This manifestation as the initial manifestation of Kawasaki disease is very rare. Only one case of a 5-year-old child with bloody diarrhea and acute renal failure has been reported with a final diagnosis of Kawasaki (3). In this patient, hemolytic-uremic syndrome was suggested as the cause of acute renal failure. A number of 39 patients with Kawasaki disease have been diagnosed with acute renal failure. The etiology of this complication was unknown in 25% of patients. Oliguria, edema, hypotension, and hypertension were symptoms of this complication. 68.4% of patients had multiple organ dysfunction, with an average of two organs involved (13). Involved organs include the respiratory, nervous, hemolytic, and hepatobiliary systems. The treatment of patients with Kawasaki disease with prenatal AKI and heart failure includes restoration of normal adequate intravascular volume, cardiac support, and special treatment for Kawasaki (17).

In patients with intrinsic AKI, renal damage is caused by renal artery vasculitis, T or B-cell-mediated autoimmune disorders of B or T cells. Therefore, therapies prescribed based on the severity of Kawasaki disease also treat AKI, of course, with regard to water, electrolyte, and diuretic treatments (17, 18). Our patient had a decrease in fever after receiving the initial antibiotic, the initial symptoms of renal failure, namely edema, oliguria, creatinine more than 1.5 times normal, showed dehydration. He underwent diuretic, water, and electrolyte treatments, and developed fever and leukocytosis after 9 days. On suspicion of Kawasaki disease, IVIG was prescribed and he gave a dramatic response to treatment. The importance of this case report lies in the fact that in the early manifestation of a sick child with high creatinine, edema, fever, and vomiting, Kawasaki disease can be considered for the patient.

Conclusion
Kawasaki's diagnosis has been challenging for physicians due to the atypical manifestations of the disease. A definitive diagnosis may not be possible with the initial manifestations. Renal disease as a primary manifestation of Kawasaki is not common and has only been reported in a previous case with a different etiology. We hope that this report will help pediatricians with the diagnosis and timely treatment of Kawasaki in patients with rare clinical demonstrations.

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Conflict of interest
This article did not use any funding to conduct this research and have no conflict of interest.

References
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both the urethra and the kidney. Pediatr Nephrol 2007;22(7):987-91. PMID: 17323086