Case Report

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A Pediatric Case of Kawasaki Disease with Gastrointestinal Involvement Mimicking Acute Abdomen

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Abstract

Background: Kawasaki Disease (KD) is a childhood acute vasculitis that predominantly affects medium-sized arteries, particularly the coronaries. While classical KD is diagnosed on the basis of defined clinical criteria, atypical presentations, such as with gastrointestinal (GI) involvement, can obscure timely recognition of this disease. GI symptoms, observed in subset of cases, may mimic acute surgical abdomen.

Objective: To highlight the diagnostic challenge of Kawasaki Disease with predominant gastrointestinal symptoms mimicking acute abdomen in a pediatric case, and to emphasize the importance of early recognition and timely initiation of intravenous immunoglobulin (IVIG) therapy to prevent potential cardiac complications.

Case: This is the case report of a 7-year-old male who was admitted with high-grade fever, severe abdominal pain, and signs suggestive of an acute abdomen. Initial clinical assessment and abdominal ultrasound indicated mesenteric lymphadenitis. Laboratory findings revealed marked leukocytosis, anemia, thrombocytosis, and elevated inflammatory markers (ESR, CRP) along with increased NT-proBNP suggestive of systemic inflammation. Further imaging ruled out surgical pathology. The persistence of fever, along with emerging mucocutaneous signs, led to the diagnosis of KD. The patient was treated with intravenous immunoglobulin (IVIG) and supportive therapy. Fever resolved within 24 hours, and abdominal symptoms improved markedly. Patient was then discharged afebrile with no evidence of coronary artery abnormalities on echocardiography.

Conclusion: This case illustrates that persistent fever and elevated inflammatory markers in the presence of GI symptoms should prompt consideration of KD, even in the absence of full diagnostic criteria. Early diagnosis and IVIG therapy are important as delayed diagnosis may lead to significant cardiac sequelae.

Keywords: Acute abdomen, intravenous immunoglobulin, kawasaki disease, mesenteric lymphadenitis.

Introduction

Kawasaki Disease (KD) is an acute febrile vasculitis of childhood first described by Dr. Tomisaku Kawasaki in 1967.[1] It predominantly affects children under five years of age and is characterized by systemic inflammation of medium-sized vessels with a particular predilection for the coronary arteries. Although the precise etiology remains unknown, current evidence suggests that an abnormal immune response to infectious triggers in genetically susceptible individuals contributes to its pathogenesis. KD has emerged as the most common cause of acquired heart disease in children in developed and developing nations alike. Without early diagnosis and timely treatment nearly 20–25% of patients may

develop coronary artery aneurysms.[2]

The diagnosis of KD is clinical (based on the American Heart Association (AHA) criteria) and include fever persisting for at least five days and at least four of the following five criteria: (1) bilateral non-exudative conjunctivitis, (2) oral mucosal changes (erythema, fissured lips, strawberry tongue), (3) peripheral extremity changes, (4) polymorphous rash, and (5) cervical lymphadenopathy..[3] However, incomplete or atypical KD poses diagnostic challenges particularly in children who do not initially meet the full criteria. Gastrointestinal (GI) manifestations are among the most misleading atypical presentations in cases of KD in paediatric age group.[4]

Gastrointestinal involvement in KD has been reported in approximately 2–35% of cases, ranging from mild symptoms such as diarrhea, vomiting, and abdominal pain to severe complications that may include gallbladder hydrops, pancreatitis, pseudo-obstruction, ischemic colitis and intestinal perforation.[5] These manifestations may mimic an acute abdomen like presentation and lead to unnecessary operative interventions. For example, cases of KD presenting as acute appendicitis or bowel obstruction have been reported, where the diagnosis of KD was only established after persistent fever and systemic signs continued despite surgery. Such clinical scenarios emphasize that abdominal symptoms can precede classical features of KD and have the potential to delay diagnosis and increasing the risk of cardiac complications. [6]

The pathophysiology of gastrointestinal involvement in KD is thought to be related to systemic vasculitis which affects the submucosal vessels of the bowel. This leads to edema, ischemia, or even hemorrhage in the gastrointestinal tract (GIT).[7] Clinically atypical presentations such as acute abdominal pain create a diagnostic dilemma. In Pediatric age group when abdominal pain, tenderness, or peritoneal signs dominate the presentation, pediatricians may initially suspect appendicitis, mesenteric adenitis, or enteritis rather than KD.[8] Radiological findings such as mesenteric lymphadenitis, bowel wall thickening or colonic edema may add to the confusion but should raise suspicion in the right clinical context. Importantly, the persistence of fever despite antibiotics or surgery together with evolving mucocutaneous features should prompt treating pediatrician to consider KD as an underlying etiology.[9] Early recognition of KD in children presenting with acute abdomen is critical. IVIG administered in early phase of illness significantly reduces the risk of complications such as development of coronary artery aneurysms.[10]

This case specifically underscores how mesenteric lymphadenitis and acute abdominal pain can precede the classic mucocutaneous signs of KD. These non-specific GI symptoms can create a diagnostic challenge that clinicians must actively recognize to avoid delayed treatment. Early recognition of these presentations can prevent not only unnecessary surgical interventions but also enables timely initiation of IVIG and ultimately reduce the risk of serious cardiac sequelae.

Case Report

A 7-year-old male child was brought to ED with complaints of persistent high-grade fever for three days, severe abdominal pain, cough and reduced oral intake. On examination he appeared acutely ill and was febrile with a temperature of 39.2°C. His lips were cracked, and there was mild peeling of the skin over his hands. Respiratory examination revealed tachypnea with scattered wheezes and crepitations. Abdominal examination demonstrated diffuse tenderness. The total leukocyte count was elevated at 21.9×10^3/µL, hemoglobin was reduced at 9.5 g/dL, and platelet count was significantly raised at $733x10^3$/µL. In view of presence of diffuse abdominal tenderness an ultrasound of the abdomen was done which showed enlarged mesenteric lymph nodes suggestive of mesenteric lymphadenitis. Given presence of diffuse tenderness and no significant findings on USG other than enlarged mesenteric lymph nodes a computed tomography (CT) of abdomen was advised. CT abdomen also showed presence of enlarged mesenteric lymph nodes without any other significant intraabdominal pathology.

Presence of significant abdominal pain and the mesenteric lymphadenitis initially favored the diagnosis of acute abdomen. However, absence of a definitive intraabdominal surgical pathology on imaging created uncertainty. The persistence of fever despite supportive management and emerging systemic features prompted reconsideration of the diagnosis of acute abdomen and further investigations were ordered. Inflammatory markers were ordered which showed significantly raised erythrocyte sedimentation rate (80 mm/hr) and C-reactive protein levels (91.7 mg/L). The coagulation profile showed a mildly elevated D-dimer (0.51 µg/mL) whereas PT

(12 seconds) and INR (1.1) values were found to be within normal limits. Additional testing revealed elevated NT-proBNP (293.5 pg/mL). Procalcitonin levels were normal (0.04 ng/mL).

Elevated inflammatory markers and presence of mucocutaneous changes including (cracked lips and peeling of the skin on the hands) supported the suspicion of Kawasaki Disease. An echocardiography was done which showed trace tricuspid regurgitation and minimal pericardial effusion. These systemic and cardiac manifestations when considered alongside the persistent fever caused the transition from an initial diagnosis of acute abdomen to Kawasaki Disease.

The patient was managed with intravenous fluids, broad-spectrum antibiotics and antipyretics. Additionally intravenous immunoglobulin (IVIG) was started. Following administration of IVIG the fever started subsiding and abdominal pain diminished. Additionally oral intake started improving. By the time of discharge (6th day since admission) the child had remained afebrile for more than 48 hours and demonstrated marked clinical recovery. At discharge parents were advised close outpatient follow-up with ongoing care and paediatric cardiology consultation for repeat echocardiography to monitor for possible late-onset coronary artery complications.

The child's gastrointestinal presentation, in the form of mesenteric lymphadenitis and severe abdominal pain initially overshadowed the classical features of Kawasaki Disease and created the risk of delayed diagnosis. However, persistence of fever despite supportive therapy raised the suspicion of Kawasaki Disease. Timely administration of IVIG led to prompt resolution of signs and symptoms.

Discussion

Gastrointestinal involvement in Kawasaki disease (KD), though uncommon, can pose a significant diagnostic challenge. This case involving a 7-year-old child who was admitted with history of fever and acute abdominal pain and was later diagnosed to be having KD. This transition of diagnosis from acute abdomen to KD highlights critical diagnostic challenge in cases of KD. While the classic features of KD are well known atypical presentations such as gastrointestinal symptoms require a high index of suspicion. Colomba *et al* conducted a retrospective analysis of KD patients with gastrointestinal manifestations and reported that in many cases, GI symptoms like abdominal pain, vomiting, and diarrhea may precede the appearance of typical mucocutaneous signs.[11] The authors further concluded that the diagnosis and treatment of Kawasaki disease might be delayed if intestinal symptoms appear before the characteristic clinical features of Kawasaki disease. In this case significant abdominal pain, diffuse abdominal tenderness and mesenteric lymphadenitis on ultrasound initially pointed toward acute abdomen however subsequent clinical picture and investigations shifted the focus from acute abdomen to KD. Similarly, Nasri *et al*. also reported several GI signs and symptoms such as pseudo-obstruction and colitis changes in cases of KD all of which initially can divert attention away from KD.[12]

Delayed diagnosis of KD has been directly associated with an increased risk of later complication coronary artery aneurysms. Fabi *et al*. in a multicenter prospective study, demonstrated that children with gastrointestinal involvement had significantly higher rates of delayed treatment, intravenous immunoglobulin (IVIG) resistance and coronary artery abnormalities compared to those without GI symptoms.[13] Many studies have reported that GI presentations may be associated with a more severe inflammatory type of KD. Moreover, the elevated inflammatory markers seen in this patient, including raised CRP and ESR are consistent with the findings as reported by Fabi *et al* in their cohort of patients with GI-predominant KD.[13] Therefore, GI signs and symptoms in febrile children particularly when persistent and accompanied by laboratory signs of systemic inflammation should raise suspicion of KD.

Imaging studies in KD with gastrointestinal involvement can further complicate the diagnostic picture. Mesenteric lymphadenitis, as seen in this patient has been reported in several cases as an early radiologic finding. Blevrakis *et al* described a case in which KD presented with acute abdomen and prominent mesenteric lymphadenopathy on ultrasound initially leading to a provisional diagnosis of acute abdomen and an exploratory laparotomy was done.[14] However, the patient later developed classic KD features. This case is similar in that abdominal ultrasound findings contributed to the initial diagnostic dilemma. Importantly the authors emphasized that sterile mesenteric lymphadenitis in a febrile child should raise suspicion for KD. This is more so if no definite surgical

cause is identified. In this case serial clinical observation and persistence of fever helped avoid unnecessary surgical exploration. The resolution of abdominal pain after IVIG administration confirmed the inflammatory rather than surgical nature of the pathology in this case.

Cardiac involvement remains one of most feared complication of in KD. The most crucial strategy in mitigating this risk is timely IVIG therapy. Eladawy *et al* studied the diagnostic pathways in patients with KD who initially presented to gastroenterology services due to abdominal complaints.[15] The authors suggested that gastroenterologists should be aware of gastrointestinal presentations of KD and Unexplained gastrointestinal symptoms in the presence of fever, should prompt consideration of KD in the differential diagnosis so that IVIG administration is not delayed. In this case IVIG was administered within the first week of illness which resulted in rapid clinical improvement and no coronary artery abnormalities were found on initial echocardiography. However, the literature consistently emphasizes the need for serial echocardiographic follow-up as coronary changes usually evolve over time and generally manifest after clinical resolution.

The limitation of this case report is absence of serial echocardiographic follow-up which limits the evaluation of long-term cardiac outcomes as cardiac manifestations in Kawasaki Disease may appear after a delayed period.

In conclusion this case of KD presenting with clinical features mimicking an acute abdomen underscores the importance of recognizing gastrointestinal manifestations as part of the disease spectrum of KD. Recognizing GI-dominant KD at an early stage is crucial because not only these cases are more prone to diagnostic delay but may also reflect a more severe inflammatory burden and a higher risk of coronary complications as compared to KD cases without GI symptoms. High index of suspicion, appropriate work up and prompt IVIG therapy are essential to prevent adverse outcomes.

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