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Exacerbation of Atopic Dermatitis Secondary to Kawasaki Disease

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ABSTRACT

Kawasaki disease (KD) is a systemic vasculitis that affects small and medium-sized vessels, most observed in infants and young children. Although its etiology remains unclear, recent studies suggest that infectious agents and immune dysfunction play a role in its pathogenesis. On the other hand, the atopic triad is associated with immune dysregulation and a hyperreactive immune system. This study aims to investigate the potential relationship between KD and the atopic triad, as children with a history of combined allergic diseases appear to have a higher risk of developing KD.

We present the case of a 2-year-old male patient recently diagnosed with atopic dermatitis (AD), who presented with periorbital edema, periungual desquamation of the hands and feet, fever, rhinorrhea, strawberry tongue, abdominal pain, and bilateral conjunctival injection. A presumptive diagnosis of incomplete KD was made, accompanied by a concomitant exacerbation of his AD during the same episode. Treatment was initiated with intravenous immunoglobulin (IVIG), acetylsalicylic acid, syndet cleansers, and emollients.

A genetic predisposition to the atopic triad may be associated with increased immunological susceptibility to KD, as these patients are more prone to hyperreactivity in response to infections or antigens that come into contact with the skin and mucous membranes. Studies have shown that patients with KD exhibit higher levels of eosinophils, Th2 cytokines (such as IL-4), and eosinophilic cationic protein, all of which are significantly reduced following treatment with intravenous immunoglobulin (IVIG).

Keywords: Atopic dermatitis; Kawasaki disease; Atopic triad; Exacerbation of dermatitis

INTRODUCTION

Kawasaki disease (KD) is a systemic vasculitis affecting small and medium-sized vessels, primarily occurring in infants and preschool-aged children between 6 months and 5 years of age. It is characterized by persistent fever lasting at least five days with temperatures above 38°C, along with the presence of at least four of the following

five principal clinical criteria (according to the American Heart Association): bilateral conjunctival injection without exudate, changes in the oral mucosa (cracked lips, strawberry tongue, or erythematous pharynx), peripheral extremity changes (erythema around the nails in the acute phase), generalized rash, and cervical lymphadenopathy (with a diameter greater than 1.5 cm).[1] In contrast to the AHA criteria, Japanese guidelines do not consider a fever lasting at least five days as a mandatory criterion.[2]

KD is the leading cause of acquired heart disease in children, primarily manifesting as coronary artery abnormalities in its initial stages.[1] However, timely administration of intravenous immunoglobulin (IVIG) within the first 10 days of illness reduces this risk to 2-4%.[3,4] If left untreated, up to 25% of cases progress to coronary artery ectasia or aneurysms, with the risk of acute myocardial infarction and sudden death.[5] Acetylsalicylic acid (ASA), due to its antiplatelet effect, is used at moderate to high doses in combination with IVIG as the standard treatment.[6]

The exact etiology of KD remains unknown, but recent studies suggest a possible link to infectious agents and dysregulated immune function.[5] Eosinophils play a crucial role as the most significant predictor of KD when present at levels >1.5%. KD is associated with increased expression of various T helper (Th)-1 cytokines (IL-6, IL-12, TNF-alpha, CXCL10, and IFN-gamma), as well as elevated levels of Th2 cytokines (IL-4, IL-5, IL-13, and IL-31). Both Th1 and Th2 immune responses are heightened during the acute phase of KD, and it has been observed that the Th2 response appears to exert certain anti-inflammatory effects, similar to what occurs in atopic dermatitis.[7-10] On the other hand, atopic dermatitis is associated with immune dysfunction and is characterized by a state of immune hyperreactivity predominantly mediated by the Th2 response.[10] Evidence has been found that both Th1 and Th2 cytokines activate immune systems triggered by an etiological agent of KD.[11] Children with a prior history of combined allergic diseases (atopic dermatitis, allergic rhinitis, and urticaria) are at a higher risk of developing KD, which is diagnosed on average 2.83 years later in life.[12-13]

CLINICAL CASE

A 2-year-old male patient with a recent diagnosis of atopic dermatitis, which was adequately controlled and without active lesions, presented with periorbital edema and periungual desquamation on the hands and feet, as well as scaling on both knees. Nine days prior to admission, the patient developed fever of 38.5°C for one day, clear rhinorrhea, strawberry tongue (Figure 4), abdominal pain, loose stools (Bristol Stool Scale 6), and bilateral conjunctival injection (Figure 3).

The patient was initially evaluated by a pediatrician in a private setting, who ordered laboratory tests. Results were within normal ranges except for an elevated eosinophil count of 8%. Tests for SARS-CoV-2 antigen, IgG and IgM antibodies, dengue NS1 antigen, and influenza detection were all negative. Given the clinical suspicion of Kawasaki disease (KD), the patient was referred to pediatric cardiology. An echocardiogram revealed ectasia of the left coronary artery, pericardial hyperreflectivity, and dilation of the left coronary artery. Consequently, the patient was admitted to our unit for further management.

The patient presented with a disseminated dermatosis affecting the head, particularly the facial region with a predominance on the cheeks, the entirety of the upper extremities, the anterior and posterior regions of the

thorax, and the lower extremities, primarily the knees. The lesions consisted of multiple erythematous, well-defined papules measuring 1 to 3 mm in diameter, which tended to coalesce on the abdomen and the dorsum of the hands (Figures 2,5 and 6). Additionally, intense erythema and distal periungual desquamation were observed on the palms and soles (Figures 1 and 2).

A diagnosis of KD was established, along with an exacerbation of atopic dermatitis. Treatment was initiated with a single dose of intravenous immunoglobulin (IVIG) at 2 g/kg and high-dose acetylsalicylic acid (80 mg/kg/day) for 6 days, followed by a reduction to a maintenance dose. Additionally, syndet cleansers and emollients were used to restore the skin barrier. Complete clinical remission was achieved after one month of follow-up.

Figure 1: Lingual papillary hypertrophy (raspberry tongue).

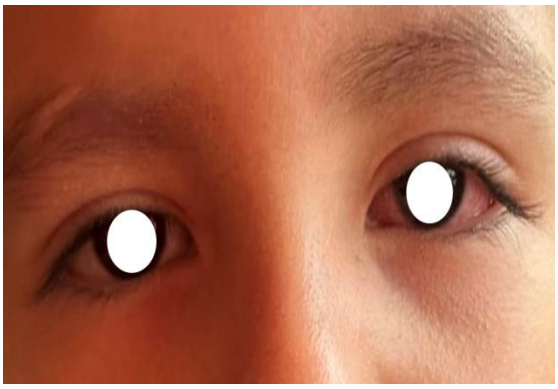

Figure 2: Conjunctival bilateral injection with periocular edema.

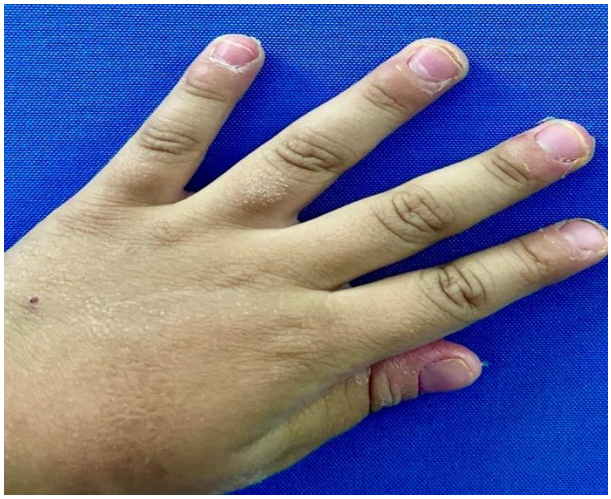

Figure 3: Periungual peeling on hands.

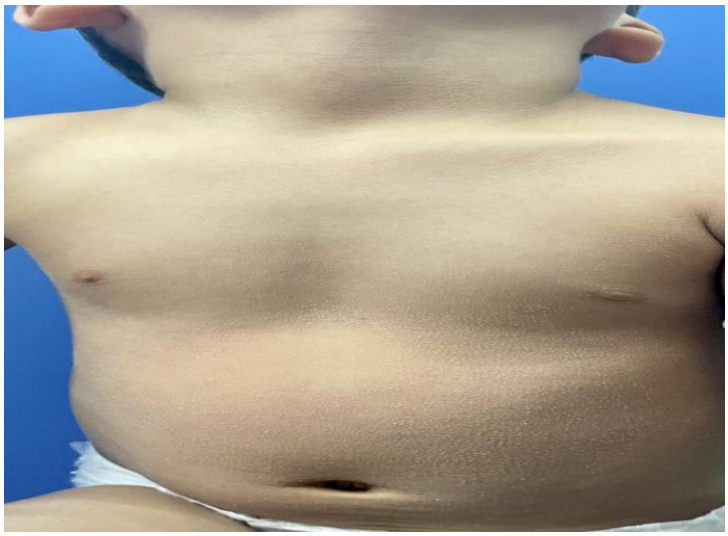

Figure 4: Anterior thorax with multiple papules 0.1-0.3 mm in diameter, erythematous, delimited that converge to form surface plaques.

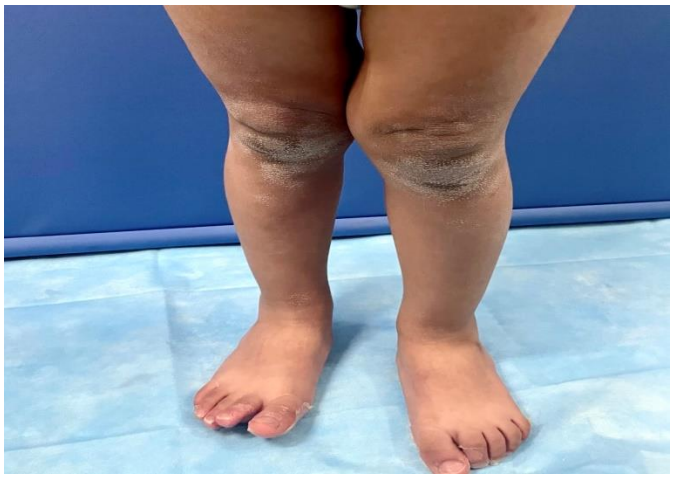

Figure 5: Bilateral peeling on knees.

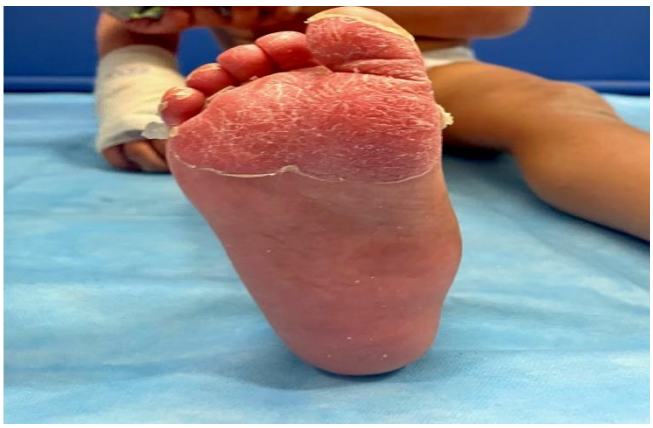

Figure 6: Periungual peeling on feet and plantar peeling.

DISCUSSION

A genetic predisposition to the atopic triad may be associated with an immunological susceptibility to developing Kawasaki disease (KD). Patients with atopic tendencies are prone to hyperreactivity in response to infections or antigens that come into contact with the skin and mucous membranes. Studies indicate that patients

with KD exhibit higher levels of eosinophils, Th2 cytokines (IL-4), and eosinophil cationic protein, all of which significantly decrease following treatment with intravenous immunoglobulin (IVIG). Elevated IgE levels, particularly during the second week of KD progression, suggest a key role of immunoglobulin-mediated immunity and proinflammatory factors related to atopic dermatitis.[12-13]

A meta-analysis published in 2013 in The Journal of Pediatrics examined 200 patients with KD under the age of 5. It found that 7.5% had a history of atopic dermatitis, while 3.5% presented with the complete atopic triad.[12] Similarly, a 2020 prospective study conducted in Taiwan, which followed 2,748 patients from childhood with KD up to 17 years of age, demonstrated statistically significant correlations between KD and the development of allergic rhinitis, atopic dermatitis, and urticaria.

CONCLUSION

Although Kawasaki Disease (KD) is a common pediatric condition, it is crucial for both dermatologists and pediatricians to identify patients at increased risk of severe manifestations and ensure prompt medical intervention to prevent complications such as coronary artery aneurysms. Further research is needed to clarify the relationship between KD and the atopic triad, with the goal of improving patient outcomes.

Given the overlap in susceptible populations and the shared association with immune dysregulation, investigating the relationship between the atopic triad and KD is essential. Specifically, it is important to determine whether the atopic triad precedes KD and whether it contributes to a more severe clinical course due to an underlying proinflammatory state. Additionally, it is necessary to evaluate the risk of patients developing components of the atopic triad following an episode of KD. These findings could inform more tailored follow-up strategies and optimize the clinical management of these patients.

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