Plaque psoriasis induced after Kawasaki disease

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Kawasaki disease is an acute febrile mucocutaneous lymph node syndrome and a multisystem vasculitis affecting children under five years of age. The induction of chronic plaque psoriasis following Kawasaki disease can be mediated by common pathomechanisms. A recently suggested pathomechanism is based upon superantigens, which may trigger both diseases in patients with suspected genetic predispositions. Herein, a case who developed psoriasis after Kawasaki disease is presented together with the related literature.

Key words: Kawasaki disease, psoriasis, superantigen, trigger.

Kawasaki disease (KD) is an acute febrile mucocutaneous lymph node syndrome and a multisystem vasculitis affecting children under five years of age. Psoriasis following KD is rarely reported. Herein, a case who developed psoriasis after KD is presented.

Case Report

A seven-month-old girl was consulted for an eruption that had started five days before. One month prior to the appearance of the rash, she was admitted to the Pediatrics Department for fever above 38°C persisting longer than one week, nausea and vomiting. She had concurrent conjunctival hyperemia and periorbital edema. On physical examination, swollen palms and soles, reddened lips and strawberry tongue were noted. Cervical lymphadenopathies were palpated bilaterally. Electrocardiography was normal. According to the clinical symptoms and physical examination, a diagnosis of KD was made. Intravenous immunoglobulin therapy (2 g/kg) was given for five consecutive days and aspirin (3 mg/kg/day) was administered. After the resolution of KD, she was followed otherwise healthy for two weeks until a rash appeared over the entire body. Dermatological examination revealed erythematous scaly plaques on the cheeks, trunk, and bilateral upper and lower extremities (Fig. 1). Physical examination was normal. A punch biopsy was performed from one of the lesions over the left thigh and histopathologic examination confirmed a diagnosis of psoriasis (Fig. 2). After clarifying the dermatologic diagnosis, a detailed family history revealed that the patient's mother also had plaque psoriasis. Hydrocortisone-17-butyrate cream was commenced for the psoriatic lesions once a day and the patient was free of lesions at the follow-up visit three weeks later.

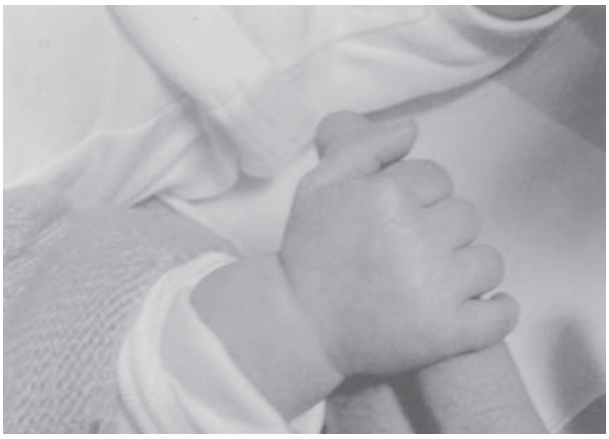

Fig. 1. Erythematous, scaly psoriatic plaques over the hand.

Discussion

Kawasaki disease (KD), also known as mucocutaneous lymph node syndrome, infantile polyarteritis and Kawasaki syndrome, is a complicated vasculitis that affects many organs,

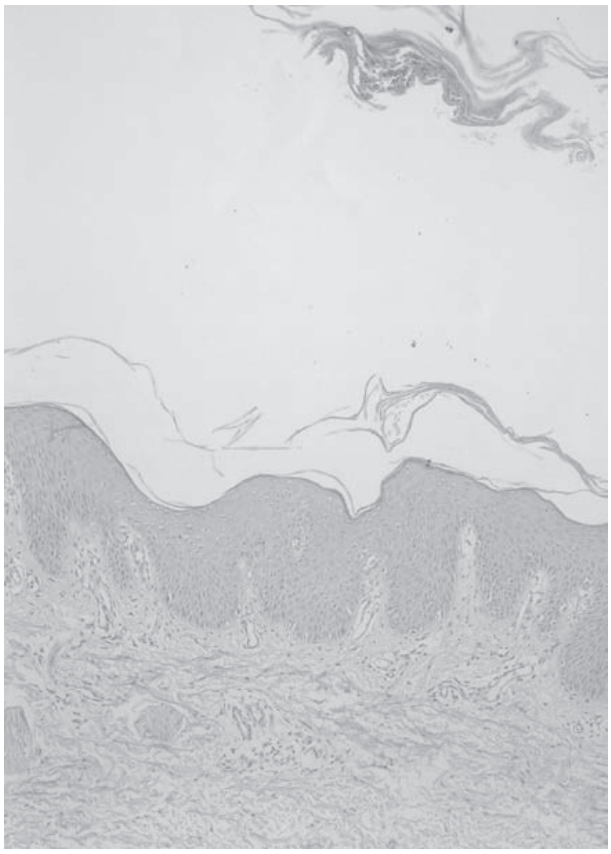

Fig. 2. Munro microabscess in the stratum corneum and regular acanthosis confirming psoriasis.

including the skin and mucous membranes, lymph nodes, blood vessel walls, and the heart. It was first described in 1967 by Dr. Tomisaku Kawasaki in Japan[1]. The disease is most prevalent in Japan and Asian-American populations. It especially affects young children; nearly 80% of the patients are younger than five years of age. KD typically presents with irritability, fever and malaise. The clinical findings of the disease confirm the diagnosis when fever for five days accompanies four of the following features: skin eruption, stomatitis (injected pharynx and strawberry tongue) and fissuring cheilitis, edema of the hands and feet, conjunctival injection, and cervical lymphadenitis. The skin eruptions may be morbilliform, urticarial or scarlatiniform and are thus better defined as polymorphous. Perianal desquamation is accepted as the other stigmata of the disease[2]. The most important complication of the disease is coronary artery aneurysms, which may occur in 20 to 25% of untreated patients[3]. Intravenous immunoglobulin therapy is the mainstay treatment for KD. Aspirin is added to the regimen to inhibit platelet aggregation and reduce inflammation.

Psoriasiform skin changes are described in the literature in both acute (1-4 weeks) and convalescent phases (4-12 weeks) in patients with KD. In a study among 476 patients with KD, pustules and psoriasiform plaques were noted in 10 patients, but only two of them had a histological confirmation of psoriasis[4]. The incidence of palmoplantar pustular involvement was reported to be >6.5% in Japanese patients with KD[5].

The interesting association of psoriasis and KD has brought a new additional insight into the disease pathomechanism. A respiratory infectious agent may be the triggering factor for KD because the disease is seen in the childhood population and shows seasonal peaks in winter and spring, and some epidemic cases are reported in the literature. Additionally, a recently growing idea is that superantigens pull the trigger in disease onset in KD. Superantigens are bacterial or viral proteins that directly bind to the major histocompatibility complex (MHC) peptide binding groove on the surface of the antigen presenting cells without any prior intracellular processing. The successful binding of the superantigen especially depends on the Vbeta region in the T cell receptor (TCR) complex. T lymphocytes exhibiting specific variable beta chains in their TCR complex are directly stimulated by these super antigens[6]. In patients with KD, an increase in Vbeta 2+ and Vbeta 8+ TCR was shown[7], and superantigen-producing bacteria were frequently isolated from KD patients[8]. In a similar manner, psoriasis is known to be triggered by superantigens. A previous study was able to show that the streptococci isolated from the pharynx of guttate psoriasis patients produce a superantigen called streptococcal pyrogenic exotoxin C that potently stimulates T lymphocytes exhibiting Vbeta2 molecule in their TCR complex. These activated T cells were shown to predominate in the newly advancing psoriatic lesions of the patients[9]. As similar T lymphocyte activation pathways can cause both diseases, the etiologic agent can be interpreted as a common environmental factor acting as a superantigen.

The family history of our patient revealed that her mother also had psoriasis, and we believe that this genetic susceptibility might have led to the easier induction of definite plaque psoriasis together with KD through the

above-explained pathomechanisms. The awaited reported disease associations of psoriasis will further enlighten the etiology of the disease. Although it has not been reported in any of the KD patients with psoriasiform eruption, we think further follow-up of patients with concurrent psoriasis and KD in order to monitor the development of chronic plaque psoriasis is mandatory.

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