Case report

Systemic contact dermatitis due to nickel

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Abstract

Introduction: Systemic contact dermatitis (SCD) is a systemic reactivation of a previous allergic contact dermatitis. The initial exposure may usually be topical, followed by oral, intravenous or inhalation exposure leading to a systemic hypersensitivity reaction. A case of a 27 year-old male with SCD due to nickel is reported.

Case Report: A 27 year-old male presented with recurrent pruritic eruption consist of deep seated vesicles on both palmar and left plantar since 6 months before admission. This complaint began after patient consumed excessive amounts of chocolate, canned food, and beans. The patient worked as a technician in a food factory. History of allergy due to nickel was acknowledged since childhood. The clinical presentation was diffuse deep seated vesicles, and multiple erythematous macules to plaques, with collarette scale. Patch test using the European standard showed a +3 result to nickel. The patient was diagnosed as systemic contact dermatitis due to nickel. The treatments were topical corticosteroid and patient education of avoidance of both contact and systemic exposure to nickel. The patient showed clinical improvement after 2 weeks.

Discussion: SCD was diagnosed due to the history of massive consumption of food containing nickel in a patient who had initial sensitization to nickel, with clinical features and the patch test result. Advice to be aware of nickel and its avoidance is important in SCD management.

Key word: Systemic contact dermatitis, nickel-sensitization, dietary nickel, patch test, food induced systemic contact dermatitis

Introduction

Systemic contact dermatitis (SCD) is a systemic reactivation of a previous allergic contact dermatitis. The initial exposure may usually be topical, followed by oral ingestion, intravenous or inhalation contact leading to systemic hypersensitivity reaction. Various types of skin eruptions have been reported, from a recall reaction (dermatitis at the site of prior topical sensitization), symmetrical intertriginous and flexural exantheme, widespread dermatitis, and erythroderma. Metals such as nickel, cobalt, and zinc are abundant in our environment. They can act as an allergen of SCD. This case reports a 27 year-old male with SCD due to nickel.

Case

A 27 year-old male presented with recurrent pruritic eruption of deep seated vesicles on both hands and left foot since 6 months before admission. This complaint began after the patient consumed excessive amounts of chocolate, canned food, and beans at his working place. The vesicles on both hands and left foot appeared at the same time. The patient often scratched and rubbed them until the
lesions ruptured, exhibited redness, and scales. The patient worked as a mechanic in a food factory. His complaints improved when he was on vacation and stopped eating chocolate or canned food but exacerbated when consumption resumed. The patient used unspecified medication and improved. The patient showed normal overall health. Patient always used gloves and washed his hands with soap after work. The patient had a history of allergy due to nickel since child and a history of skin atopy. The clinical presentation was deep seated vesicles, multiple circumscribed to diffuse erythematous macules to plaques, with collarette scale on both palms and left plantar (Figure 1). The patch test using European standard result after 48 hours was +1, after 72 hours was +3, and after 96 hours was +3 to nickel (Figure 2,3,4). The patient was diagnosed with systemic contact dermatitis due to nickel. The patient was given topical corticosteroid and advised to avoid contact and systemic exposure to nickel. The patient showed clinical improvement after 2 weeks.

Discussion

Systemic contact dermatitis (SCD) is a contact hypersensitivity reaction in which ingestion or other systemic exposure to a contact allergen occurs in an already sensitized person. It is also known as endogen contact eczema, systemic contact-type dermatitis, systemically induced contact dermatitis, internal-external contact-type hypersensitivity, symmetrical drug related intertriginous and flexural exanthema, baboon syndrome, systemic reactivation of allergic contact dermatitis, and systemic allergic dermatitis. SCD happens through two stages: sensitization and elicitation phase. Although the initial sensitizing exposure is usually by topical application, there are multiple routes of exposure for the elicitation of SCD: subcutaneous, intravenous, intramuscular, inhalation, and oral ingestion. Even percutaneous exposure through inflamed or broken skin may result in enough systemic absorption to cause SCD. These reactions may occur not only after ingestion of the primary allergen, but also after ingestion of other immunochemically related allergen.
The diagnosis of systemic contact dermatitis in this patient was made based on the recurrence of extremely pruritic eruption of deep seated vesicles on both hands and feet after massive consumption of high nickel diet even there was no contact to nickel or other metals. This patient worked in a factory that produced a lot of chocolate, canned food, beverage cans and beans. The patient often gets free food and beverage. The complaints began after working at the food factory and regular consumption of a diet high in nickel. There exists a wide spectrum of clinical presentation from a recall reaction (dermatitis at the site of prior topical sensitization), to widespread dermatitis and erythroderma.

There are two primary situations in which dietary nickel should be considered as a possible cause or exacerbating factor of dermatitis. The best known situation is vesicular hand dermatitis in which there is a positive patch test reaction to nickel. The other situation is that of a widespread dermatitis or even isolated pruritus in the setting of a positive nickel patch test. Dietary nickel reactions should be particularly strongly considered when patients demonstrate...
widespread dermatitis or hand dermatitis. Our patient developed hand dermatitis every time he had a high nickel diet. He was certain that the hand dermatitis always came together with the dermatitis on his left foot.

The immunologic basis for SCD is not completely understood and may not be identical for all allergens. As reviewed recently by Jacob and Zapolanski, during allergen sensitization, a hapten penetrates the skin and reacts with resident antigen-presenting dendritic cells that transfer the bound antigen to T lymphocytes. Once these cells are primed and reproduce, they return to the skin, ready to act on target cells when the antigen is encountered again. It seems that the immune system can be activated by allergen exposure as well through oral and other systemic routes, triggering the activated CD8+ effector T cells. The differential diagnoses in this case was tinea, allergic contact dermatitis, and dyshidrosis. The potassium hydroxide examination from skin lesions result was negative for fungal infection. The lesion on left foot that had no history of contact to metal eliminated the diagnosis of allergic contact dermatitis. Dyshidrotic eczema is a type of eczema of multi-factorial etiology characterized by a pruritic vesicular eruption on the fingers, palms, and soles. Exogenous factor like nickel, balsam, cobalt, ingested metals, dermatophyte infection, and bacterial infection may trigger recurrent episodes. Evidence shows that the ingestion of metalions can induce type I and type IV hypersensitivity reactions. They can also act as atypical haptens, activating T lymphocytes human leukocyte antigen-independent pathways, causing systemic allergic dermatitis in the form of dyshidrotic eczema.

Patch test can be performed in every case of chronic and recurrent itchy dermatitis. It helps in the identification and avoidance of the offending hapten. Patch tests use certain amounts of suspected haptens that are applied onto the skin for 48 hours, with the assessment of skin reactions at defined times, after 2,3 and 4 days. Test substances should be chosen accordingly to clinical history. We used the European standard patch test in this patient. The patch test result in our patient was only positive and relevant to nickel. The first reading at 48 hours showed that the patch test result was only erythematous (+1). The second reading at 72 hours showed coalescing vesicles (+3). The third reading in 96 hours was more coalescing vesicles and bullae. We concluded that there is no cross sensitization to other metal. Nickel is a very common cause of allergic contact dermatitis (ACD).

We suspected nickel as the causative allergen due to the history of allergy due to nickel since childhood and massive consumption of chocolate, canned food, and beans at his workplace. Sensitized individuals generally have a predictable localized response following cutaneous exposure to nickel, including erythema, vesicles, scales, and pruritus. Systemic reactions, such as hand dermatitis or generalized eczematous reactions, can occur due to dietary nickel ingestion.

SCD is more complex to diagnose than allergic contact dermatitis. Oral provocation test using suspected allergen after elimination diet can be used to diagnose SCD. The patient was given a topical corticosteroid and education to avoid contact and systemic exposure to nickel. It is very hard to eliminate nickel from our diet. Using a low nickel diet, an 80% improvement can be expected. The skin condition depends on the nickel concentration in body. Vitamin C has been shown to reduce plasma nickel concentration when ingested concurrently with nickel. According to Hindsen (2003), the flare-up induced by nickel appears to be linked not only with the dose, but also with the intensity of the previous reaction and its proximity in time. Patient must know the list of food with high nickel content such as chocolate, beans, broccoli, cauliflower, coffee, salmon, soybean, tomatoes, and tuna. Total elimination of nickel is not possible to do, as nickel is a metal ubiquitously present. Suggested diets contain less nickel than normally assumed, but there is no certainty of the nickel content in any given diet, and it is impossible to know the exact amount of nickel taken daily. Elimination diet should result in an improvement of the disease attributed to nickel at least 80%, in absence medications. The double blind placebo controlled challenge test is the gold standard in the diagnosis of oral allergy to nickel and is highly recommended and generally necessary for a correct diagnosis.
References