Diffuse Symmetric Intertriginous Erythema in a 39-year-old Woman

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CASE REPORT

A 39-year-old woman presented to our out-patient department with a diffuse skin rash symmetrically distributed over the neck, submammary areas, axillae, antecubital fossae, groins, buttocks, and upper inner thighs. Four days previously, she had been exposed to mercury vapor after accidentally breaking a thermometer at home. About one day after the mercury exposure, a slightly itchy erythema began to appear on the flexural and friction areas of her body. It then rapidly expanded to involve her trunk and extremities (Fig. 1). She denied any systemic disorders or allergy. Also, she could not recall whether she had any previous exposure to mercury or mercury compounds. The eruption cleared with systemic and topical corticosteroid therapy. Patch tests were positive on day 2 for metallic mercury, ammoniated mercury chloride, and mercury chloride (Fig. 2).

Fig. 1
Diffuse symmetric erythematous patches and purpuric erythema predominantly on the intertriginous areas and buttocks, with a V-shaped pattern on the anteromedial thighs.

Fig. 2
Positive patch test reactions to metallic mercury, ammoniated mercury chloride, and mercury chloride at 48 hours after application.
**DIAGNOSIS: Baboon Syndrome**

**DISCUSSION**

The term baboon syndrome was introduced in 1984 to denote a characteristic dermatologic response, i.e., diffuse erythema with particular involvement of the buttocks and upper inner surface of the thighs resembling the red bottom of baboons. It occurs in response to the systemic administration of contact allergens and to certain drugs. The originally implicated causative agents were mercury, nickel, and ampicillin.1

In fact, in 1983 Nakayama et al. had published 15 cases of diffuse symmetrical erythema predominantly on major flexural areas that appeared a day or two after breaking a mercury thermometer or during dental treatment.2 This specific eruption was recognized as a mercury exanthem. Later, Menn et al. proposed the term systemic contact dermatitis for dermatitis caused by systemic administration of substances either with or without previous topical exposure.3 Baboon syndrome was classified as one of the five clinical patterns seen in systemic contact dermatitis.

Metals and drugs are the most common causative agents of baboon syndrome. Of the metals, mercury is most often reported, but it can occur as well with exposure to nickel, cobalt, and gold. Amoxicillin and β-lactam antibiotics account for most drug-induced cases.4 There is typically no known or documented prior sensitization to the agent. The skin lesions are characteristically well-defined V-shaped erythematous patches, with occasional tiny papules, vesicles, or pustules in inguinal and gluteal areas as well as in other flexural areas. The reason for the flexural predilection is unclear, although localized occlusion and sweating have been suggested as contributory.5,6 The palms, soles, face and mucosal areas are spared. The rash usually appears hours to a few days following exposure to the causative agent. Systemic symptoms are rarely present, and laboratory examinations are normal. The histology of the skin lesion is nonspecific and nondiagnostic, usually demonstrating only superficial perivascular infiltrates of mononuclear cells.7

Systemically applied mercury may result in cutaneous reactions with different manifestations. As baboon syndrome is the most common presentation, acute generalized exanthematous pustulosis (AGEP) accounts for about 10% of the cases.2 The skin rash is most commonly provoked by the inhalation of metallic mercury vapor from a broken thermometer.2,7 It is not unusual to obtain a history of previous exposure, such as to mercurochrome or dental amalgam. Delayed-type hypersensitivity to mercury could be confirmed by patch test in most cases of baboon syndrome as well as in AGEP.2

In baboon syndrome, it is suggested that systemic rechallenge with mercury in a previously sensitized individual may induce the classical antigen-presenting cell-T-cell activation pathway and directly stimulate lymphocyte proliferation. The excretion of mercury by sweat glands might explain attraction and localized accumulation of activated and proliferating T cells in the flexural areas.7 Though baboon syndrome and AGEP may share similar latency time, the same causative agents, and the potential common immunologic pathogenesis, the explanation for the distinct systemic signs and cutaneous manifestations are still lacking.

**REFERENCES**