



Exanthema following a spider bite mimicking baboon syndrome

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Baboon syndrome (BS) is characterized by V-shaped erythema in inguinal and perianal areas with exanthema in other flexural areas, occurring after systemic exposure to contact allergens.¹ The non-contact allergic variant of BS is referred as symmetrical drug-related intertriginous and flexural exanthema (SDRIFE).² We report an unprecedented case of exanthema following a spider bite mimicking BS and misdiagnosed as SDRIFE caused by cotrimoxazole.

CASE REPORT

A 69-year-old woman was referred with an erythematous eruption that appeared 1 day after taking cotrimoxazole for an inflammatory lesion of the chin caused by a spider bite (Figure 1). The patient presented with an erythematous rash with tiny pustules, symmetrically involving the buttocks (Figure 2), inner thighs, popliteal folds, and upper limbs. There was no fever, and no mucosal or other involvement. The results of laboratory tests were normal. Skin biopsy showed



FIGURE 1 Inflammatory lesion on the chin



FIGURE 2 Symmetrical erythema on the buttocks mimicking baboon syndrome

spongiosis in the epidermis and a perivascular lymphohistiocytic infiltrate with eosinophils and some neutrophils in the dermis. The patient met all of the criteria of SDRIFE established by Häusermann et al.¹ SDRIFE caused by cotrimoxazole was diagnosed. Cotrimoxazole was discontinued and topical steroids were prescribed. The skin lesions regressed within a few days. A patch test with cotrimoxazole 30% pet. gave negative results on day (D) 2 and D3. An oral provocation test gave a negative result, showing that cotrimoxazole was not responsible. The diagnosis of exanthema induced by a spider bite mimicking BS was made.

DISCUSSION

We report a case of exanthema following a spider bite mimicking BS and misdiagnosed as SDRIFE caused by cotrimoxazole. The patch test and, especially, the oral provocation test allowed us to revise the diagnosis. Some cases of acute generalized exanthematous pustulosis (AGEP) have been reported to be induced by spider bites, including three cases in Tunisia.³ The venom of the spider contains sphingomyelinase, which stimulates the liberation of cytokines, including great amounts of interleukin-8 and granulocyte-macrophage colony-stimulating factor, which would trigger AGEP.⁴ Except for systemic signs, AGEP and SDRIFE have similar latency times, potential immunological pathogeneses, and causative drugs.¹ As BS is a closely related entity, clinically and physiopathologically, to AGEP, it seemed logical to incriminate the spider bite in our case. However, according to the original publication from 1984,⁵ BS is defined as systemically induced allergic contact dermatitis. The current case reproduces the same clinical picture as BS, but does not fulfil the criteria, as this is not a case of allergic contact dermatitis. Our observation seems to be unprecedented.

CONFLICTS OF INTEREST

The authors have no conflicts of interest to report.

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