Clindamycin-induced Maculopapular Exanthema with Preferential Involvement of Striae Distensae: A Koebner phenomenon?

Clindamycin is a lincomycin-derived antibiotic useful for the treatment of anaerobic and Gram-positive aerobic bacterial infections. Cutaneous adverse reactions are usually maculopapular exanthemas, although hypersensitivity syndrome, acute generalized exanthematous pustulosis, and Stevens-Johnson syndrome have also been reported (1).

We report the case of a patient with a maculopapular rash triggered by clindamycin who developed cutaneous lesions on striae distensae (SD).

A 47-year-old woman was referred to our clinic for pruritic cutaneous lesions which had started 6 days earlier. Her past clinical history included hypertension, hypothyroidism, hyperuricemia, cholecystectomy, caesarean section, and endometriosis-related abdominal surgery, and she was taking levothyroxine, allopurinol, imidapril, and omeprazole. The skin rash first developed on her neck and back on the 3rd day of clindamycin oral treatment (300 mg every 6 hours), which was prescribed as antibiotic prophylaxis for a tooth implant. General malaise (but not fever) was also reported.

Physical examination revealed an erythematous maculopapular eruption symmetrically distributed on the neck, abdomen, and back (Figure 1, A), with isolated lesions involving the proximal upper and lower limbs (Figure 1, B). There was a striking vertical distribution of skin lesions along the SD on the lateral sides of the abdomen (Figure 1, C). No mucosal involvement was found, and laboratory studies showed no abnormalities.

Clindamycin withdrawal was followed by prescription of a course of oral deflazacort, starting at 30 mg daily and tapering down during a 9-day period. On the 5th day of treatment, the rash had almost cleared with minimal desquamation (Figure 1, D).

Eight weeks after clearance of the skin rash, informed consent was obtained in order to perform an

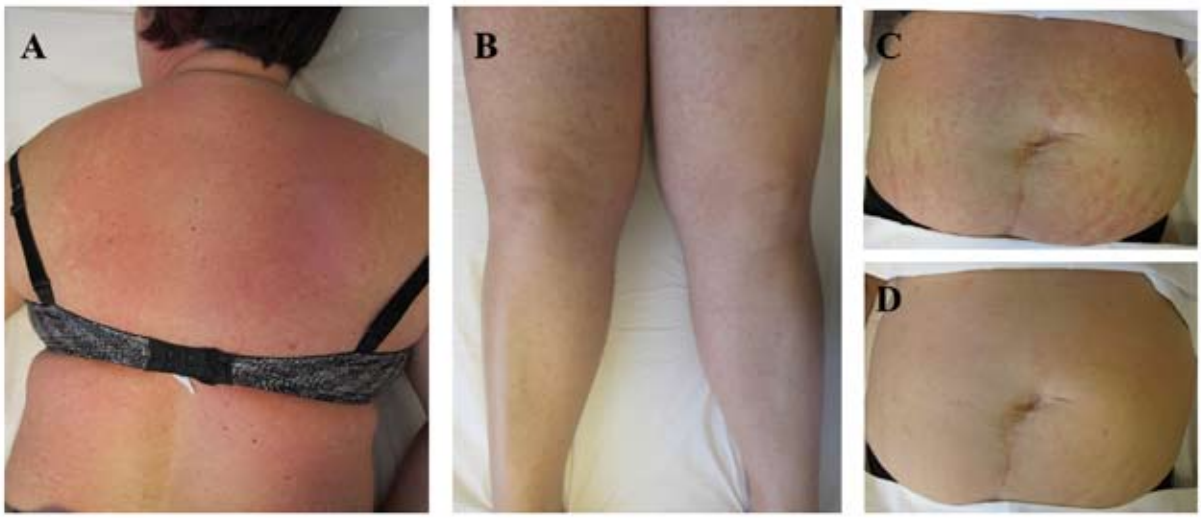

Figure 1. (A) Maculopapular confluent exanthema on the back. (B) Isolated erythematous lesions on the anterior aspect of the thighs. (C) Vertically disposed eruption along the striae distensae (SD) on the lateral wall of abdomen. (D) Lesions cleared 5 days after clindamycin withdrawal.

allergological evaluation of clindamycin, including prick and intradermal (ID) tests on the forearm and patch tests on the upper back (2). For patch testing, powder of the commercial capsules (Dalacin®) was diluted in petrolatum (pet.) and water (aq.), resulting in a final 1% clindamycin dilution. Parenteral clindamycin preparations were used in therapeutic concentrations for prick tests (150 mg/mL) and dilutions in saline of 1/100 and 1/10 for the ID test. Other authors have reported that these concentrations do not seem to irritate the skin (3-6). Prick and ID tests were assessed after 20 min and 24 hours, respectively. Patch tests were removed after the 2nd day, and late reactions were evaluated on day 2 and day 4. Prick and ID test results after 20 min were negative. Late results of ID tests with clindamycin (1.5 and 15 mg/mL) were positive: erythematous infiltrated papules about 7×7 mm and 18×15 mm were observed at 24 hours and lasted until the 8th day. Patch tests with clindamycin 1% in pet. and 1% in aq. were also positive (+ on day 2 and day 4). Positive late skin tests suggested delayed-type non-IgE-mediated allergic clindamycin hypersensitivity. Oral challenge tests are considered to be the gold standard to establish or exclude drug hypersensitivity. Due to the positive result of late skin test to clindamycin, oral challenge was not performed in our patient (3,5).

The Koebner isomorphic phenomenon has been described in cutaneous reactions induced by drugs, such as antibiotics and chemotherapy. Chronic pressure on the skin is probably involved in the onset of skin lesions in hand-foot eruptions induced by tyrosine kinase inhibitors (sorafenib and sutinib). Solar exposure and cutaneous trauma also seem to play a role in the location of papulopustular eruptions caused by endothelial growth factor receptor inhibitors (erlotinib) (7). More frequent involvement in traumatized skin and surgical scars has been reported in the context of linear IgA bullous dermatosis and leukocytoclastic vasculitis triggered by vancomycin and cefuroxime (8).

SD are produced by non-penetrating physical trauma, similar to friction or pressure. Different dermatoses can develop along SD skin lesions (like plaque psoriasis, pustular psoriasis, lichen planus, vitiligo, discoid lupus erythematosus, lupus vasculitis, urticarial vasculitis, or chronic graft-versus-host disease) (9).

Bevacizumab, etretinate, and corticosteroid-induced ulcers, hyperpigmentation caused by bleomycin, and urticariform lesions triggered by diclofenac are examples of different type of drug-induced abnormalities involving SD (10).

In summary, we identified clindamycin as the cause of the cutaneous reactions that occurred in our patient on the basis of the results of the skin tests and clinical history. Our findings confirmed a delayed-type hypersensitivity reaction, possibly involving a T-cell-mediated immunologic mechanism. Intradermal and patch tests were found to be useful in order to confirm the diagnosis (4,5). We did not find reports in the literature of drug-induced cutaneous eruptions along the SD as a manifestation of a Koebner phenomenon. Clinical underreporting of this phenomenon could explain the scarce literature on this cutaneous adverse reaction.

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