Systemic allergic dermatitis reaction to nickel released from an eyelet in an intravenous catheter

Contact Dermatitis 2009: 61: 180–182

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Key words: baboon syndrome; intravenous catheter; nickel; symmetrical drug-related intertriginous exanthemata; systemic allergic dermatitis reaction.

The term systemic allergic dermatitis reaction has been used for patients sensitized to contact allergens who, upon its systemic exposure, develop particular clinical manifestations (1). These include dyshidrotic hand eczema, flare-up of patch tests, disseminated maculopapular exanthemata, disseminated eczemas and vasculitis-like reactions (2) as well as symmetrical drug-related intertriginous exanthemata (3) or baboon syndrome (4). Contact allergens responsible for this clinical manifestation include nickel and mercury and drugs such as amoxicillin among many others (1,3).

Case Report

A 53-year-old female patient had suffered from three episodes of a severe flexural exanthem (Fig. 1) during three hospital admissions. She was referred to evaluate a suspected drug hypersensitivity. Her medical history included Crohn's disease and contact allergy from jewellery. During all admissions, she was on complete intravenous nutrition using an Optiva® catheter. She had received different drugs (enoxaparin, methylprednisone, methotrexate, mesalazine, metronidazole, ciprofloxacin, paracetamol, metamizol, morphine) and underwent two radiographic examinations with contrast media.

As most probable elicitors, enoxaparin, ciprofloxacin, paracetamol and radio contrast media (3,5) were suspected.

Prick tests were done with undiluted drugs from commercial vials, intradermal test with dilutions from 1:10 to 1:1000 depending on the drug (heparin, ciprofloxacin, metronidazole, paracetamol). They were read at 20 min and 1D. Patch tests applied for 2D with the European series extended with additional allergens as recommended by the Information network of Departments of Dermatology (IVDK), an antibiotic (penicillin G, amoxicillin trihydrate, dicloxacillin, cefotaxim, clindamycin phosphate, cefradine, cefalexin, doxicycline, minocycline, erythromycin, spiramycin, clarithromycin, pristinamycin, co-trimoxazole, norfloxacin, ciprofloxacin), a corticosteroid (amcinonide, hydrocortisone, triamcinolone acetonide, clobetasol-17-propionate, hydrocortisone-17-butyrate, betamethasone-17-valerate, budesonide, prednisolone, dexamethasone-21-phosphate, tixocortol pivalate) and a radio contrast screening series (iodixanol, ioxaglate, iopamidol, amidotrizoate) were performed. Prick and intradermal tests with the anticoagulants, the radio contrast media and the antibiotics series were all negative. Patch tests read according to the recommendations of the International Contact Dermatitis Research Group revealed a ++/+++ test at D2/3 to nickel sulfate only. After giving informed consent, the patient was re-exposed orally with 0.5 mg nickel sulfate, followed after 1 hr by 250 mg mannitol (placebo) and, after another 2 hr, with 1 mg nickel. After 4 hr, an erythematous flare-up was observed in the inguinal folds. Re-exposure with 1250 ml intravenous glucose 5% over 3 hr through an Optiva® catheter resulted in no reaction.

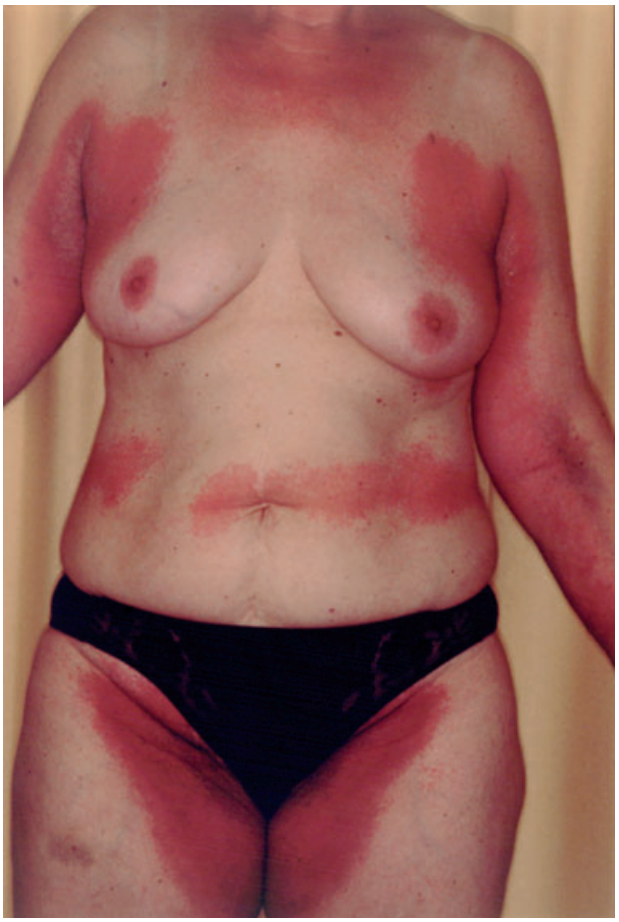

Fig. 1. Symmetrical, sharply demarcated erythematous plaques in the large folds.

The Optiva® catheter was cut open and the metal eyelet in the device was removed. Scanning electron microscopy including the embedded energy-dispersive X-ray spectroscopy (XL 30 ESEM-FEG/EDAX, Philips, Eindhoven, The Netherlands) was applied to analyse the eyelet's composition and surface morphology. The weight percentages of the interior and exterior surfaces were determined to be 83–88% nickel and 12–14% phosphorus, which corresponds to the nickel–phosphorus alloy Niphos®. The bulk of the eyelet consists of brass (68% copper and 30% zinc). The SEM image showed micrometre-sized nickel–phosphorus globules on the surface (Fig. 2).

Discussion

Systemic allergic dermatitis manifesting as symmetrical maculo-papular, sharply demarcated eruption of the

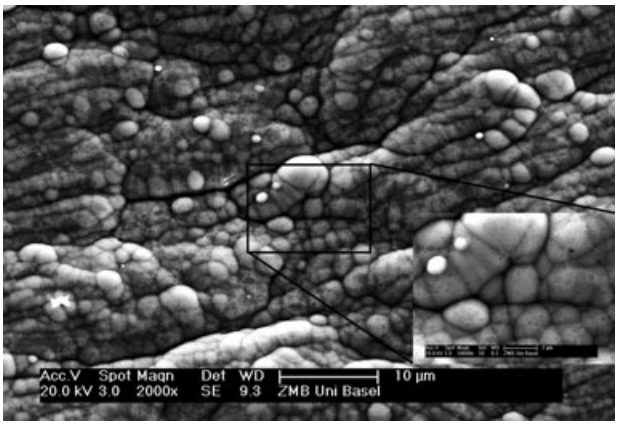

Fig. 2. The SEM image represents the morphology of the interior surface of the metallic eyelet. Loosely bonded globules may be easily removed by the shear stress of fluid flow.

great folds and the genital and gluteal area is a well-known phenomenon (3, 4). The contact allergen or drug may be absorbed through the skin, by parenteral administration, the gastrointestinal or respiratory tract.

In a previous report, two patients with a very similar clinical manifestation and findings have been described (6). A nickel spot test on the dry metallic eyelet did not detect any nickel. However, after incubation for 2D, and by analysing the nickel content of a glucose infusion passed through the catheter, a considerable nickel concentration was measured. In our patient, re-exposure over 3 hr with a glucose infusion through an Optiva® catheter was apparently not sufficient to elicit symptoms. However, the oral provocation with cumulative 1.5 mg nickel was sufficient to trigger a mild flare-up.

We suggest that particularly in hospitalized patients with long-term intravenous therapy who develop signs of systemic allergic dermatitis, eczematous lesions or maculo-papular exanthemata, the culprit nickel from such catheters may go unrecognized and consequently they may be falsely labelled as drug allergic.

Because, in the European Union, the limit of nickel release from jewellery is regulated, it is also essential that the use of medical devices that contain metal parts, which release considerable amounts of nickel, should be prohibited.

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