

CONTACT POINTS

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Erythema multiforme due to contact with laurel oil

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Erythema multiforme is a relatively common skin disorder. The best known cause is herpes simplex virus infection. We report the first case of erythema multiforme due to contact with laurel oil. The diagnosis was confirmed by the positive patch test to laurel oil, the histopathological studies of the lesions and the histopathological studies of the positive patch test to laurel oil.

Key words: contact dermatitis; erythema multiforme; laurel oil.

Erythema multiforme (EM) is an acute, self-limited, occasionally recurrent skin disease characterized by the abrupt onset of symmetrically fixed red papules. Some of the papules evolve into target lesions with or without mucous membrane involvement. EM is relatively common; it may account for up to 1% of dermatologic outpatient visits.

Contact erythema multiforme (CEM) is a rare non-eczematous manifestation of allergic contact dermatitis. We report a case of a patient with CEM due to contact with laurel oil.

Case Report

A 63-year-old German woman presented to our department with a

2-month history of recurrent, severe pruritic erythemas on the extremities, and reduced general condition. She had no history of drug intake, herpes simplex or other infectious disease during the previous months. The patient had a chronic arthropathy of both knees, but denied any medical therapy.

With systemic and topical steroids a partial response was seen, but recurrences were observed after cessation of the therapy. After further enquiries, she suddenly remembered the occasional use of laurel oil as topical treatment for her knee arthropathy.

On physical examination, the patient demonstrated multiple red and dull red macules and papules, 0.5–2.0 cm in diameter, in target-like configuration (Fig. 1) on her legs and right hand. Some of the lesions pre-

sented with mild scaling. Multiple petechiae, hyperpigmentations and excoriations were seen on both upper and lower extremities. The oral cavity, eyes and the genitals were free of symptoms. There were no signs of a herpes simplex infection. On blood examination, CBC and routine serum parameters were within normal limits. Antistreptococcal- and antistaphylococcal-serology were negative. The HSV- and EBV-serology did not indicate an acute infection. Direct microscopic examination and culture of scales collected from the lesions were negative for fungal infection. Chest X-ray demonstrated no pathological findings. The histologic evaluation of a biopsy taken from her left upper leg revealed apoptotic keratinocytes and perivascular as well as epidermal lymphocytic infiltration (Fig. 2a),



Fig. 1. Multiple red and dull red macules and papules in target-like configuration on the legs.

typical features of erythema multiforme (EM). The direct immunofluorescence study was negative.

With a potent steroid ointment, skin lesions had cleared within 1 week. With strict avoidance of laurel oil no further skin lesions appeared.

Free of symptoms, she was patch tested to the German standard series and to her own laurel oil. Positive reactions were obtained to compositae, balsam of Peru and fragrance mix. A strongly positive reaction to laurel oil, with the clinical aspects of a 'tar-

get' lesion, was seen (Fig. 3). A biopsy of this positive patch test showed vacuolization of basal keratinocytes (Fig. 2b) and therefore histological features of an early stage of EM. The immunohistological studies of the positive patch test showed CD8+ T-cells at the basal membrane (Fig. 2c). The direct immunofluorescence study of the same lesion was negative.

Based on the medical history, the symptoms, the positive patch test to laurel oil, the histopathological studies of the lesions and the histopathological studies of the positive patch test to laurel oil, the diagnosis of EM due to contact with laurel oil was made.

After a 3-month period of follow up and continued avoidance of laurel oil, the patient remained completely free of symptoms.

Discussion

Topical antigen contact through the skin is a relatively rare cause of EM. Here we report the first case of EM due to contact with laurel oil. The diagnosis was confirmed by a positive patch test to laurel oil, which revealed clinical and histological features typical of EM.

Allergic contact hypersensitivity can cause EM-like eruptions, or, extremely rare, a true EM (contact erythema multiforme/CEM). EM-like eruptions due to contact sensitization to sulphonamides, antihistamines, diphencyclopropenone, exotic woods, various plants, tea tree oil, nickel sulfate, colophonium, fragrance mix and buprenorphine are well documented. On cases of EM-like eruptions, the histology of the lesions is not specific and typical signs of EM such as keratinocyte apoptosis are not observed (1).

A cell-mediated immune response to antigen that has been processed and presented within the skin has been postulated in EM pathogenesis. The end point is the immunologic attack on antigen-expressing epidermal keratinocytes, carried out by cytotoxic effector cells (CD8+ T-lymphocytes), which induce apoptosis of individual scattered keratinocytes. The immune processes involved in CEM and post-herpetic EM appear to be identical, with focal epidermal expression of ICAM-1, as well as HLA-DR molecules on keratinocytes and the

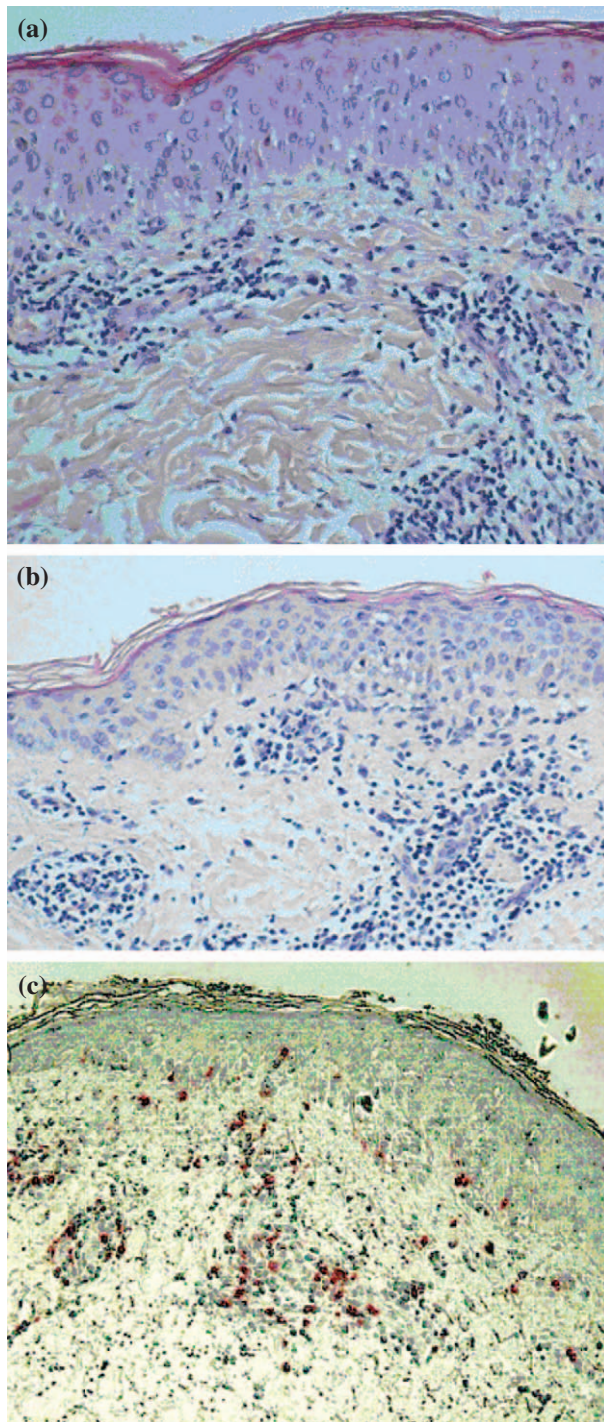


Fig. 2. (a) Photomicrograph showing apoptotic keratinocytes and perivascular as well as epidermal lymphocytic infiltration (haematoxylin and eosin). (b) Histologic evaluation of the positive patch test reaction to laurel oil showing vacuolization of basal keratinocytes (haematoxylin and eosin). (c) Immunohistochemical stains using monoclonal antibodies against CD8 (APAAP method).



Fig. 3. Patch test: a strongly positive reaction to laurel oil.

same immunophenotype of lymphocytes infiltrating the epidermis and dermis (2).

The keratinocyte is the target of the inflammatory process in EM. Early lesions exhibit lymphocyte accumulations at the dermal-epidermal interface, with exocytosis into the epidermis (CD8+ T-lymphocytes), scattered apoptotic keratinocytes with attached lymphocytes (satellite-cell necrosis), spongiosis, vacuolar degeneration of the basal layer, and focal junctional and subepidermal cleft formation. Advanced lesions show subepidermal blister formation and frank epidermal necrosis (3).

As mentioned before, our patient presented with lesions that were both clinically and histopathologically typical for an EM. Further workup of the patient rather suggested the diagnosis of CEM and not an EM-like eruption. The histopathological studies of the lesions and the histopathological studies of the positive patch test to laurel oil both support the diagnosis of EM. To our knowledge, this is the first case of EM to be reported in association with laurel oil. There is a case report of allergic contact dermatitis to tea tree oil (chemically similar to laurel oil) with erythema multiforme-like id reaction. In that case, the skin biopsy of the positive patch test showed a spongiotic dermatitis without histological features of EM (4). A generalized

EM reaction as a complication of patch testing is extremely rare. A research performed in the database Medical-line showed only 4 publications of that phenomenon (5–8).

The commercial production of products such as tea tree and laurel oil has considerably increased over the past 15 years in response to a strong demand for natural remedies and aromatic substances.

CEM is a rare manifestation of allergic contact hypersensitivity. A detailed, sometimes repeated medical history, including topical 'over-the-counter' remedies is mandatory in all patients with non-HSV dependent EM, since the prompt identification and removal of the aetiological agent is the mainstay of therapy.

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