Encapsulated fat necrosis, also named ‘nodular cystic fat necrosis’, mobile encapsulated lipoma or ‘posttraumatic degeneration and herniation’, was first described in 1975 by Schmidt-Hermes and Loskant [4]. It is characterized by solitary or multiple, subcutaneous nodules, mostly located on the lower extremity and consisting of degenerated or necrotic fat tissue encapsulated by thin to thick fibrous tissue. The exact causes remain unclear; however, trauma and subsequent interruption of blood supply are speculated to play a major role [5]. Histopathology shows a fibrous capsule with degenerated or necrotic fat tissue in between, sometimes accompanied by inflammation and calcification.

The synopsis of clinical aspect, ultrasound, aspirated tissue and histopathology confirmed the diagnosis of encapsulated fat necrosis in our patient. It seems that the injected PPC-containing substance led to fat necrosis with the subsequent formation of surrounding fibrosis [5]. The edema, typically induced by injection lipolysis, may also play an additional role, probably by reducing the local blood supply due to pressure. In our patient, further treatments with PPC were rejected due to the complication. Both physician and patient did not observe sufficient fat reduction after the performed 2 injections.

To the best of our knowledge, our patient is the first case to present encapsulated fat necrosis after injection lipolysis for the reduction of localized fat accumulations. We assume that encapsulated fat necrosis has to be seen as a possible complication of the non-surgical procedure described above. Considering the supposed mechanism of action of PPC and the frequency of the procedure, we assume that encapsulated fat necrosis is under-reported.

References

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Letters to Dermatology

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Erythema-Multiforme-Like Eruption Recurring in Ultraviolet-Exposed Skin
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Key Words
Erythema multiforme · Fixed drug eruption · Recall phenomenon · Ultraviolet B

We report a case of an erythema multiforme (EM)-like eruption in a 48-year-old Japanese woman who had suffered from recurrent edematous eruptions of the face, trunk and extremities for a year. Eruption seemed to recur at the same sites with ultraviolet (UV) exposure. In phototesting, UVB irradiation induced macules and papules extending beyond the exposed area in the back, and eruptions resembling the target lesions appeared in the trunk and extremities. The patient was diagnosed with EM-like eruption induced by UVB irradiation. Recently, cases of drug eruptions occurring in a photo-distributed pattern have been reported. In these reports, the involvement of photo recall phenomenon was discussed. EM-like eruption is known to be triggered by UV exposure, however, the mechanism has not been revealed yet. Our case suggests that EM-like eruption recurring at UV-exposed areas may be involved in recall phenomenon.

The patient was a 48-year-old Japanese woman who had suffered from recurrent edematous eruptions of the face, trunk and extremities for a year. She had taken no medication during this period. Her medical history including drug allergy was insignificant. She had worked as an aromatherapist for a year. Eruptions often appeared within several hours after UV exposure on a sunny day and disappeared within about a week. Eruptions seemed to recur at the sites of UV exposure. These findings suggested an abnormal response to UV irradiation; therefore, phototesting was performed by illuminating the back with UV A (5, 10 and 15 J/m²) and UVB (10 doses: 20–200 mJ/m²) using a Dermaray M-DMR-100. UVB-induced macules and papules extending beyond the exposed area appeared 24 h after UVB irradiation at doses above 160 mJ/m² (fig. 1), and eruptions resembling target lesions appeared at the same site as previous eruptions on the trunk and extremities (fig. 2). UVB irradiation did not cause erythematous lesions. To assess whether there was a possible allergy to essential oils, patch and photopatch testing was performed. All test reactions were negative. Despite careful history-taking, other agents suspected to be causative were not found.

A skin biopsy specimen from the right forearm showed satellite cell necrosis of keratinocytes, lymphocytic infiltration around the upper dermal vessels and along the dermoeipidermal junction, and edema of the papillary dermis. Laboratory findings were normal. Serological tests were negative for herpes simplex virus (HSV) and mycoplasma infection, and tests for antinuclear and anti-SS-A/B antibodies were also negative. The patient was diagnosed as having an erythema-multiforme (EM)-like eruption induced by UVB.
irradiation, based on the provocative UVB tests and clinical and histological findings. Because the patient had suffered from recurrent eruptions since beginning to work as an aromatherapist, the possible involvement of aroma was strongly suspected despite the negative patch and photopatch reactions. Prednisolone 30 mg/day was started and then tapered with improvement of clinical symp-
toms. With the subsequent avoidance of UV and essential oils, there was no recurrence of the eruptions.

EM is a self-limited, usually mild and relapsing exanthematous reaction of the skin that is characterized by target-shaped, urticarial plaques and, histologically, satellite cell necrosis of the epi-
dermis. These features are the expression of an archetypal polyeti-
ological reaction pattern of the skin, i.e. a cytotoxic immunological attack on keratinocytes expressing non-self antigens [1]. The three common triggers of EM are HSV infection, mycoplasma infection and drug reactions. Recurrent EM is preceded by HSV episodes in up to 80% of patients [2]. Sun exposure is also known to trigger EM [1]; however, the mechanism has not been revealed yet.

UV exposure induces abnormal responses in some individu-
als. Recently, cases of fixed drug eruption (FDE) [3] and drug-in-
duced hypersensitivity syndrome [4] occurring in a photodistrib-
uted pattern have been reported. In these reports, the involvement of a recall phenomenon was discussed, i.e. an inflammatory re-
sponse that occurs in a previously UV-damaged tissue following drug administration [5]. In this phenomenon, immune cells re-
cruited from the circulation to the skin following UV irradiation are suspected to persist in the lesion and then to cross-react with administered drugs [5]. FDE recurs as solitary, erythematous macules in the same areas after each administration of the caus-
vative drug. Histologically, the FDE lesions consist of an interface dermatitis with lymphocytes at the dermal-epidermal junction and degenerative changes of epithelium with dyskeratosis [6], suggesting that skin lesions of EM may bear resemblance to FDE. According to Shiohara and Mizukawa [7], the mechanism of re-
currence of FDE lesions in exactly the same areas may depend on the recall phenomenon, which occurs upon various previous in-
sults such as X-ray irradiation and trauma as well as UV exposure [5]. Moreover, a wide variety of nonspecific factors other than drugs, such as cytokines, can trigger the development of FDE le-

Fig. 1. Erythematous plaques on UVB-exposed skin.

Fig. 2. Papular and urticarial target lesions on the right forearm and hand.

References
1 Fritsch PO, Ruiz-Maldonado R: Erythema multiforme, Stevens-John-
son syndrome, and toxic epidermal necrosis; in Freedberg IM, Eisen
AZ, Wolff K, Austen KF, Goldsmith LA, Katz SI (eds): Dermatology in
2 Aslanzadeh J, Helm KF, Epsy MJ, Muller SA, Smith TF: Detection of
HSV-specific DNA in biopsy tissue of patients with erythema multi-
3 Ee HL, Yosipovitch G: Photo recall phenomenon: an adverse reaction
4 So JS, Edwards SL, Ibbotson SH: Carbamazepine-induced hypersensi-
tivity syndrome occurring in a photodistributed pattern. Dermatology
2006;213:166–168.
5 Shiohara T, Mizukawa Y: Recall phenomenon: some skin-resident cells
6 Shear NH, Knowles SR, Sullivan JR, Shapiro L: Cutaneous reactions to
drugs; in Freedberg IM, Eisen AZ, Wolff K, Austen KF, Goldsmith LA,
Katz SI (eds): Dermatology in General Medicine, ed 6. New York,
15 years ago first criterion of this syndrome stating, what we emphasized some ma (SDRIFE, baboon syndrome). We especially commend the on symmetrical drug-related intertriginous and flexural exantheme which according to the authors describing 11 patients with SDRIFE, one of them (case 10) due to RCM, thus supporting a causal relationship between RCM and SDRIFE.

Second, the fifth criterion of SDRIFE is absence of systemic symptoms and signs, which according to the authors [1] is important to distinguish SDRIFE from drug rash with eosinophilia and systemic symptoms (DRESS). Here, we disagree. We contend that a patient with an eruption that has the cutaneous appearance of SDRIFE but with additional systemic organ involvement should still be classified as SDRIFE and not as DRESS. As we had suggested in an earlier publication [5], we believe that all drug eruptions – involvement of other organ systems notwithstanding – are first and foremost dermatological diseases and, as such, they should be classified according to the cutaneous lesions. In other words, SDRIFE with systemic symptoms and/or eosinophilia should be defined as SDRIFE with systemic organ involvement and not as DRESS.

We definitely agree with the authors that the description and diagnosis of cutaneous drug eruptions often require the expertise of a dermatologist. However, most patients with cutaneous drug eruptions are initially seen by nondermatologists such as general practitioners. Particularly these physicians should be aware of the specific danger signs of the severer drug eruptions with internal organ involvement [3]. On the other hand, we believe that it is less a particular morphological pattern, such as a maculopapular exanthema or SDRIFE, than the danger signs of severer reactions, needing new respect.

References

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Symmetrical Drug-Related Intertriginous and Flexural Exanthema – Reply

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Key Words
Symmetrical drug-related intertriginous and flexural exanthema · Flexural exanthema · Barium sulfate

We thank Dr. Wolf and Dr. Davidovici for their thoughtful comments on our case report [1].

We regret that the clinical case observation of a 79-year-old woman who developed the particular pattern of flexural exanthema a few hours after receiving barium sulfate by the gastrointestinal route [2] was not mentioned in our article. Since barium sulfate contains some additives, another allergen could have been responsible. To our best knowledge, iodinated radio contrast media have not been implicated in symmetrical drug-related intertriginous and flexural exanthema (SDRIFE) so far.

Definitely agree with the authors that the description and diagnosis of cutaneous drug eruptions often require the expertise of a dermatologist. However, most patients with cutaneous drug eruptions are initially seen by nondermatologists such as general practitioners. Particularly these physicians should be aware of the specific danger signs of the severer drug eruptions with internal organ involvement [3]. On the other hand, we believe that it is less a particular morphological pattern, such as a maculopapular exanthema or SDRIFE, than the danger signs of severer reactions,