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 nonsurgical 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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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 exanthematic 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-

ative 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 ins-
luts 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 les-

ions [8]. Therefore, this case may be FDE occurring in a photo-
distributed pattern due to some nonspecific factors including aromatic compounds. In phototesting, the appearance of an eruption in UVB-irradiated areas of the back might induce a reactiva-
tion of cells resident in the skin of the trunk and extremities. Our case suggests that an EM-like eruption recurring in UV-exposed areas may be FDE caused by the recall phenomenon, and we pres-

tent this case to facilitate recognition of this entity.

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We congratulate Arnold et al. [1] for their interesting report on symmetrical drug-related intertriginous and flexural exanthema (SDRIFE, baboon syndrome). We especially commend the first criterion of this syndrome stating, what we emphasized some days ago: SDRIFE should be distinguished from he-matogenous or systemic contact-type dermatitis, as suggested by Andersen et al. [3], when they first reported this syndrome. We would like to make two comments that by no means detract from the scientific and educational value of this paper.

First, the authors state that ‘the particular pattern of SDRIFE has not been observed so far’ as a reaction to radio contrast medium (RCM). We refer them to our report [4] 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.

Key Words
Symmetrical drug-related intertriginous and flexural exanthema • Radio contrast medium • Drug rash with eosinophilia and systemic symptoms

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.

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,