Traumatic Fat Necrosis: A Case Report

Cecilia Tillman, Rolf Holst and Cecilia Svedman
Department of Dermatology and Venereology, Malmö University Hospital, SE-205 02 Malmö, Sweden.
E-mail: cecilia.tillman@skane.se
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Sir,

Panniculitis has a multiple aetiology. One of the less common causes is trauma and hence traumatic fat necrosis. These soft tissue injuries usually appear on the shins, thighs, breasts, arms and buttocks. Women are particularly susceptible (1, 2). The initial injury is usually bruising of the skin with a haematoma, and later deeper indurated lesions can be palpated (2, 3). Traumatic panniculitis is a benign subcutaneous lesion with a distinct histological appearance. Focal liquefaction can occur in the injured fat, leading to discharge (2, 4). We describe an interesting case of a male patient with a fat necrosis developing into a discharging wound. The histology confirms the diagnosis.

CASE REPORT

Traumatic fat necrosis was diagnosed in a 39-year-old man with a clear previous trauma of the hip (Fig. 1). The patient had been operated on for hyperparathyroidism several years earlier, but was otherwise healthy. Two years previously, while playing ice hockey, he fell and hit his left hip and since then had felt a hard palpable lesion in the same region. The lesion had not shown any tendency to disappear. The patient could not recall any additional trauma. Two months before visiting our clinic the patient consulted his general practitioner (GP) and was advised to apply an ointment, Hirudoid® (mucopolysaccharide polysulphate, glycerol, potassium hydroxide, stearic acid, dehydrated Eucerin, emulsifying cetostearyl alcohol (type A), myristyl alcohol, preservatives (thymol, methyl paraben, propyl paraben), isopropyl alcohol and purified water), which is used to treat haematoma and thrombophlebitis. The patient applied Hirudoid, thoroughly rubbing it onto his left hip every day for 2 months. The patient noticed that during treatment the skin became reddish and finally began to ulcerate. His GP suspected an infection and started treatment with flucloxacillin. Cultivation from the area showed enterococcus, pseudomonas and anaerobic bacteria. Treatment with ciprofloxacin was initiated. About 10 days later the patient was admitted to the Department for Infectious Diseases. The wound had now enlarged and had begun to ache. An ultrasonic investigation did not show any encapsulated haematoma or abscess, while an X-ray of the hip, pelvis and femur showed no skeletal or joint changes. The wound was described as about 5×3 cm and had a yellow-green appearance. Therefore clindamycin and pivampicillin were added. Four days later the patient was seen at the Department of Dermatology. The patient had not noticed any improvement. On his left hip there was now a crater-like wound measuring about 4×4 cm, with a yellow necrosis. The surrounding skin area was indurated and erythematous. Although two 4-mm punch biopsies were taken, one from the indurated skin and one from the margin of the wound, no definite diagnosis was made. About one month later the patient was seen again, but there was no great change in the ulceration. The diagnosis was not clear, but pyoderma gangrenosum was suspected and treatment with 60 mg prednisone daily was initiated and continued for 3 weeks. An X-ray of the lungs was found normal. The hard palpable area surrounding the ulceration became softer and there was a black necrosis at the bottom of the wound. No other changes were observed. A new treatment with antibiotics (cefadroxil) was given for 10 days since infection was suspected. The bacterial culture was, however, negative.

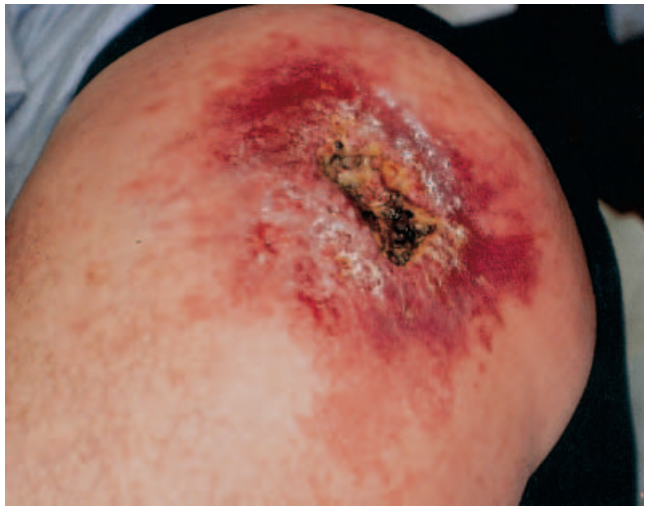

Fig. 1. Fat necrosis of the hip with a discharging wound.

After completing the prednisone treatment, a knife biopsy was taken from the indurated skin, about 2 cm from the wound. The histological examination showed a fat cell necrosis. The fat necrosis was excised and a histological examination showed a deep ulceration with fat necrosis, substantial fibrosis and foci of chronic and acute abscending inflammation (Fig. 2)

The patient was later patch-tested with the European Standard series and the ointment Hirudoid, to exclude contact allergy to Hirudoid. There was only a positive reaction to colophony, but not to Hirudoid.

Blood samples were all essentially normal, erythrocyte sedimentation rate 5, C-reactive protein <9. To exclude any underlying disease, the investigation was completed

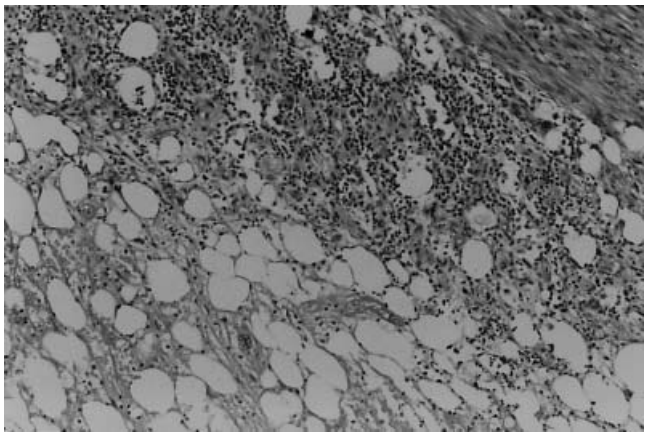

Fig. 2. Fat cell necrosis with inflammatory cells in the periphery and a prominent proliferation of fibroblasts in the corner.

with cytoplasmic, perinuclear and myeloperoxidase anti-neutrophil cytoplasmic autoantibodies (c-ANCA, p-ANCA and MPO-ANCA), which were negative. No complement deficiencies were seen. A protein analysis showed light signs of inflammation with a rise in polyclonal IgA, but was otherwise normal. There was no α1-antitrypsin deficiency. ANA, antibodies to DNA and ENA (extractable nuclear antigens) were all negative. Magnetic resonance (MR) imaging showed significant signal changes in the subcutaneous fat, extramuscularly. No changes in deeper structures were observed.

DISCUSSION

We think that the patient developed a fat cell necrosis after the trauma to the hip that occurred 2 years earlier. Trauma, pressure and surgery are known to cause fat damage (1, 5). The buttock region is previously reported to be a common site for traumatic fat necrosis and most reports indicate that women are more susceptible than men (2, 3, 6, 7).

The fat consists of microlobules of fat cells, each microlobule serviced by a blood vessel. When there is sudden pressure on the fat compartments, they burst and the surrounding septa and vessels rupture, leading to damage of the fat cells. The damaged fat cells are hydrolysed into glycerol and fatty acids, the latter then tending to irritate and aggravate the initial injury (2, 8).

The lesions often show fat cysts of varying size with surrounding fibrosis and sometimes lipomembranous change (9, 10). In the early lesions, there are fat necroses with cystic spaces and numerous neutrophils in the adjacent fat. Later there are more lymphocytes and then also histiocytes and multinucleated giant cells, some containing lipid vacuoles. Finally, the picture is dominated by fibrosis. Collections of necrotic fat cells can be seen in the dermis, sometimes in the process of being eliminated through a break in the epidermis (2, 4). A variant of fat necrosis is also reported, which is encapsulated and has fewer inflammatory changes. These smaller, nodular lesions also appear mostly at regions prone to trauma. An accepted theory is that they, too, are related to trauma and vascular insufficiency (9, 11). MR imaging is valuable in determining the diagnosis (12).

The lesion remained unchanged for 2 years in our patient. Possibly there was an encapsulation of the initial soft tissue injury that prevented further growth. Then the patient started frequently to rub and traumatize the lesion while applying the ointment. This probably led to a reactivation of the lesion. There was damage to the capsule surrounding the fat necrosis and finally it discharged to the skin surface.

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