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SHORT COMMUNICATION

Erosive Pustular Dermatosis of the Arm Following Burn Injury

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Erosive pustular dermatosis (EPD) is a rare inflammatory skin disease of unknown aetiology, characterized by multiple chronic, amicrobial pustules and crusted erosions. Since 1979, when it was first described (1), approximately 150 cases have been reported in the literature, most of which have been EPD of the scalp and legs. Typically, EPD occurs on atrophic skin caused by actinic damage on the scalp, or on legs with venous insufficiency. We describe here a case of EPD on the arm following burn injury.

CASE REPORT

A 48-year-old woman with no previous significant medical history had a superficial dermal burn on her back and a deep dermal burn on the medial side of her left arm. Initially, we treated her with clobetasol propionate ointment for 5 days. Subsequently, we used petroleum jelly (Vaseline®, Hashimacity, Gifu prefecture, Japan), sulphadiazine silver cream, and bucladesine sodium. The burn on her back healed normally within 2 weeks; however, the lesion on her left arm, which caused severe burning pain, showed no progress. One month later, the lesion became infected, and we treated her with antibiotic tablets containing sulphamethoxazole and trimethoprim and a local sucrose and povidone-iodine dressing. Initially this was partially successful, but complete clearance was not achieved. Two months after the burn there were still confluent erosions with pustules and hyperkeratotic debris on her upper arm and forearm (Fig. 1). She could not tolerate the use of sucrose and povidone-iodine, sulphadiazine silver cream, or vulnerary covering materials, such as hydro-fibre or hydrocolloid, because they all caused erythema and pain, and the condition deteriorated. The patient reported irritation when sweating or even when the area was lightly rubbed.

Laboratory tests for complete blood count, liver transferase, blood urea nitrogen, and serum creatinine were normal. Direct microscopy of pustule scrapings was negative. Microbiology revealed mild growth of methicillin-resistant *Staphylococcus aureus*. Treatment with sulphamethoxazole and trimethoprim combination tablets was re-started. Subsequent microbiological swabs were negative. However, the number of pustules did not decrease. We performed a patch-test of ointments used for external preparations, such as betamethasone dipropionate cream, betamethasone butyrate propionate ointment, petroleum jelly (Vaseline®), sulphadiazine silver cream, bucladesine sodium, hydro-fibre, and hydrocolloid, but all were negative. A biopsy on her left arm showed subcorneal and intraepidermal pustules filled with neutrophils, spongiosis at the epidermis, and diffuse dermal inflammatory infiltrate consisting of neutrophils, lymphocytes and erythrocytes. No signs of granuloma or vasculitis were observed (Fig. 2). The area taken for biopsy ulcerated.

Considering the distinctive characteristic clinical course and histologically non-specific inflammatory findings, we concluded that the patient had EPD. Although we initially chose topical betamethasone dipropionate cream, the condition did not improve; therefore, we started oral prednisolone, 20 mg daily. The lesion resolved gradually and the dosage of prednisolone was tapered by 5 mg every week. When tapered to 10 mg daily, the condition did not improve further, therefore the dose was increased again to 15 mg daily and tapered carefully while monitoring her condition. If the lesion flared up, the dose of prednisolone was increased again. During the first year, 10–20 mg oral prednisolone was administered daily. It was tapered gradually to 5 mg daily over the following years. In addition, the topical steroid was changed to betamethasone valerate cream. The patient still takes oral prednisolone, 5 mg daily. The lesion on her upper arm resolved completely after approximately one year, while the lesion on her forearm has continued as erosions covered by pustules and crusted plaques until now, 3.5 years later.

DISCUSSION

EPD was first described in 1979 (1). The original patients were all women with EPD of the scalp. In 1987 a similar condition was reported on legs with chronic venous insufficiency (2). Since then, approximately 150 cases have been reported. The clinical features of EPD are recalcitrant, erythematous, pustular, crusted erosions. This marked pustule formation often appears to be infectious; however, microbial and mycological examinations prove

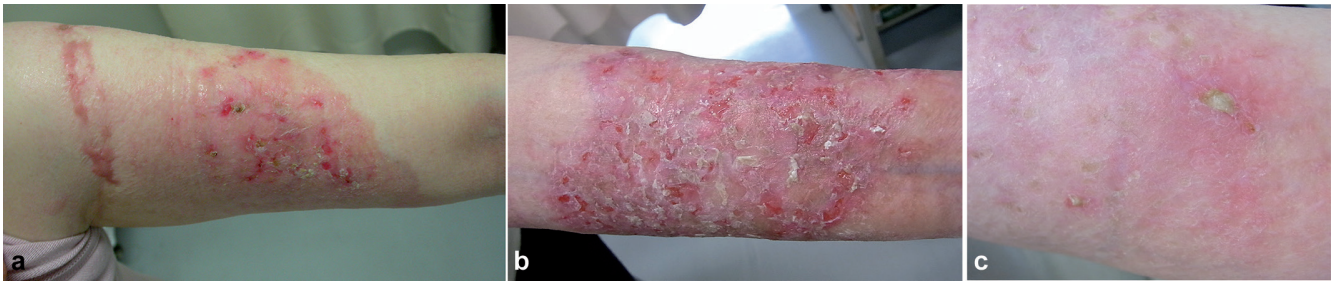

Fig. 1. Confluent erosions with pustules and hyperkeratotic debris on (a) the upper arm and (b) the forearm. (c) Close-up of pustules.

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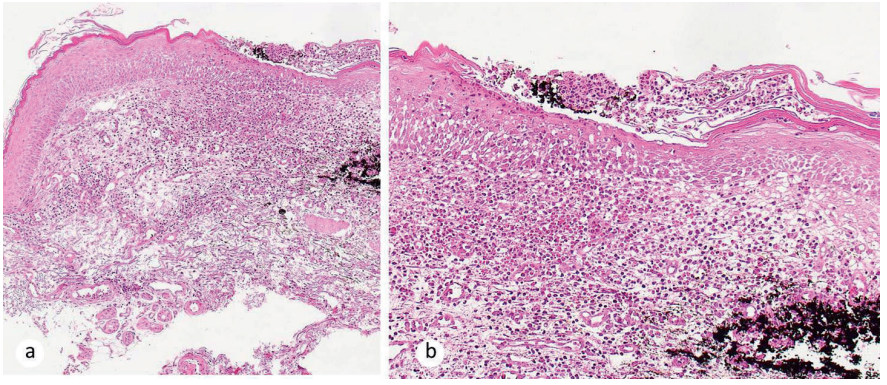

Fig. 2. Histological appearance of biopsy from the patient's left arm. (a) Scanning magnification (b) Medium-power magnification. Subcorneal and intraepidermal pustules filled with neutrophils, spongiosis at the epidermis, and diffuse dermal inflammatory infiltrate consisting of neutrophils, lymphocytes, and erythrocytes were noted. No sign of granuloma or vasculitis was observed. (Haematoxylin-eosin stain a: × 4, b: × 20).

negative in most cases. Any micro-organism cultured from involved sites is thought to be due to contamination, because antibiotics and antimycotic medication are almost always ineffective, as in our patient. Histology shows non-specific chronic inflammation, consisting of neutrophils, lymphocytes and plasma cells.

Differential diagnosis includes chronic bacterial or fungal infection, actinic keratosis, squamous cell carcinoma, pyoderma gangrenosum, and superficial granulomatous pyoderma. Therefore, EPD is a diagnosis of exclusion, based on histological and microbiological examinations.

Treatment includes high-potency topical steroids, tacrolimus, calcipotriol, dapsone, and oral retinoids. However, there have been intractable cases resistant to numerous therapeutic attempts (3).

Although its aetiology has not yet been clarified, cutaneous atrophy due to actinic damage or chronic venous insufficiency is present in many cases. Exogenous preceding factors that may induce or exacerbate EPD include: physical trauma, such as radiation therapy, photodynamic therapy, cryosurgery, carbon-dioxide laser treatment, birth trauma, skin grafting, and surgery; chemical agents, such as 5-fluorouracil cream and tretinoin; viral infection, such as herpes zoster; and mechanical pressure, such as compression therapy (3–9). A case that arose on an infant's scalp without preceding trauma has also been reported (10). Involved lesions are almost always limited to the scalp and legs. We found only that initially appeared on the big toe and quickly disseminated on the upper arm, the dorsal tongue, and scalp (7). The lesion on the arm was on the scar of old burn. The present case is the first report of EPD occurring directly after a burn, and the second report of EPD occurring on the arm. In our patient, there was no skin atrophy on the lesion. The patient had not undergone compression or exhibited irritant contact dermatitis; however, her lesion is very sensitive to irritants, as it flares up easily due to any kind of irritation, rubbing, sweating, and some kinds of ointments. We speculate that a preceding burn may be the main cause of her condition and that the sensitivity to any kind of irritant may be related to her recalcitrant condition.

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