Delayed Burn Blister

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Sir,

Delayed post-burn blistering is an interesting and unusual phenomenon which has received little attention in the literature.\(^1,2\) We herein report a case of a 20-year-old man who presented to the dermatology outpatient department with complaints of recurrent, apparently spontaneous blistering, occurring over a post-burn scar. The patient had sustained a thermal burn with accidental spillage of boiling water, over his left forearm, 5 years ago. The same healed over a 2 month period but left behind a hypertrophic scar. The first episode of blistering was a year after scar formation which healed over 1-2 weeks. There was history of 5-6 similar episodes till the time of presentation to us. These blisters arose spontaneously without any predisposing factors in the form of trauma, contact with irritants, or local site infection. The blisters were confined to the scar and healed over a few days with depigmentation. There was also no suggestion of dermatitis artefacta.

On examination, there was a hypertrophic scar with well-defined irregular margins, measuring approximately 20 × 6 cm, over the dorsal aspect of left forearm, extending to the flexor aspect as well. Overlying it was a blister filled with sero-sanguinous fluid, surrounded by crusted erosion [Figure 1]. Nikolsky’s sign as well as Bulla spread sign were negative.

Both Grams-stained and Giemsa-stained smears from the blister fluid showed only neutrophils without any evidence of microorganisms or multinucleate giant cells. A histopathologic examination of the blister edge biopsy revealed a sub-epidermal blister, along with loss of dermal appendages, marked fibrosis, and presence of neutrophils [Figure 2]. The periodic acid Schiff (PAS)-stained sections showed a poorly formed basement membrane [Figure 3]. Direct immunofluorescence (DIF) for IgG, IgM, IgA and C3 were all negative. Immunoelectron microscopy could not be performed due to financial constraints. Based on the clinical and investigational outcomes, a diagnosis of delayed burn blister was kept. The patient was reassured and counselled regarding the condition. The area was dressed with non-adherent, paraffin dressing. No oral antibiotic or any other medications were prescribed. The raw area healed completely over a course of 10 days. For the management of the hypertrophic scar, intralesional triamcinolone acetonide 40 mg/ml injections were started after the complete healing of the ulcer. These were given at 3 weekly intervals. There was a marked reduction in hypertrophic scar at the end of 2-5 months with significant flattening of the lesion and improved

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Figure 1: Clinical photograph showing hypertrophic scar with blister containing sero-sanguinous fluid

Figure 2: Photomicrograph showing thinned out necrotic epidermis associated with bulla formation at the dermoepidermal junction. The dermis shows areas of marked fibrosis with loss of dermal appendages (Hematoxylin and Eosin stain ×40)

Figure 3: Periodic acid Schiff stain showing an irregular and discontinuous basement membrane. (×100)
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symptomatology. At this stage the injections were discontinued and the patient was kept under regular follow-up.

Acute burn blisters are a well-known entity and a subject of intensive study. In contrast, the phenomenon of delayed burn blisters has received very little attention in the literature.\textsuperscript{[1-3]} Delayed blisters, observed weeks to months after initial healing in cases with second-degree thermal burns, on donor sites, and on recipient sites of split-thickness skin grafts have been known.\textsuperscript{[1-3]} Clinically these are seen in the form of tense vesicles or blisters, developing spontaneously after a prolonged interval of complete healing of the injured skin.\textsuperscript{[1-3]} They remain confined to the injured site not encroaching on the surrounding normal skin which can be an important pointer towards this possibility. They tend to heal spontaneously and can have a distinct tendency for recurrence over a period of weeks to months.

The exact pathogenesis is not known. It has been reported that the antigenic components of the normal dermo-epidermal junction appear sequentially during wound healing as they do in fetal life. An enhanced fragility of the newly synthesized, ‘immature’ dermo-epidermal junction could be responsible for this delayed blistering.\textsuperscript{[4,5]} It has been shown that a discontinuity of the basement membrane may be the main anomaly, with a disturbed reassembly of the basement membrane components contributing to it.\textsuperscript{[4,5]}

Histologically, delayed burn blisters are sub-epidermal blisters with minimal inflammation. Direct immunofluorescence is negative and show characteristic diminished or variable expression of dermo-epidermal junction antigens, including type IV and type VII collagens, laminin, and bullous pemphigoid antigen.\textsuperscript{[4,5]}

Blistering associated with a healed injured site can also be due to herpetic infection, or autoimmune bullous diseases. These can be easily excluded by a negative Tzanck smear, histology, DIF findings. These blisters tend to heal spontaneously with simple care, topical antibiotics and wound dressings, as was seen in our case.\textsuperscript{[1-3]}

We present this case to make the clinician’s aware about this lesser known and poorly understood entity. Further reports of similar cases can add to a deeper understanding of this phenomenon.

References


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