New onset bullous pemphigoid arising in Mohs surgical site

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Bullous pemphigoid (BP) is the most common autoimmune vesiculobullous disease affecting the elderly population.[1,2] Many triggers for BP have been identified, including medications, vaccines, infections, surgery, and radiation.[1,2] Rare cases of BP induced by orthopedic, podiatric, and general surgery procedures have been reported. We present a case of BP presenting weeks after Mohs micrographic surgery (MMS) within the procedure scar before generalizing. The patient responded well to therapy and flares were prevented during subsequent MMS with pulse corticosteroids.

Abbreviations used:

BP: bullous pemphigoid
MMS: Mohs micrographic surgery

CASE REPORT

A 91-year-old White man underwent MMS for an invasive squamous cell carcinoma of the dorsal surface of the right hand. The first stage yielded a 1.1 × 1.3 cm defect to the mid-subcutis with clear margins. An intermediate layered repair with 4/0 poliglecaprone-25 dermal sutures and 5/0 fast-absorbing plain gut epidermal sutures was used to close the wound (Fig 1, *A*). His initial postoperative course was uneventful with appropriate healing. One month later, he presented with a noninflamed serous bulla over the healed incision (Fig 1, *B*). The bulla was neither pruritic nor purulent.

The differential diagnosis included seroma, bullous impetigo, contact dermatitis, edema bulla, porphyria cutanea tarda, pseudoporphyria, and BP. The bulla resolved with application of a potent topical steroid. It recurred several weeks later and resolved again after debridement. Three months after surgery, the patient returned with a large heme-crusted plaque covering the MMS site and new pruritic bullae with an erythematous base on the contralateral hand, thigh, and great toe (Fig 2). The patient denied any history of autoimmune or dermatologic disease aside from keratinocyte carcinomas. Direct immunofluorescence confirmed the clinical diagnosis of BP. Excellent disease control was obtained with a 1-month prednisone taper, doxycycline 200 mg/day, niacinamide 1000 mg/day, and topical clobetasol.

DISCUSSION

BP presents in individuals genetically predisposed to autoimmunity after development of antibodies against the hemidesmosomal proteins BP180 and BP230.[3] Autoantibody binding leads to complement activation, inflammatory cell recruitment, and, ultimately, subepidermal cleavage. Linear deposition of IgG and complement C3 along the basement membrane zone on direct immunofluorescence is diagnostic.[1] Consequently, tense, pruritic bullae with an erythematous base develop. The prognosis is variable but control can be achieved through immunosuppressive or anti-inflammatory regimens.

Except for advanced age, our patient had no risk factors for BP. The temporal and spatial relationship between his MMS and disease onset suggested a triggering role. BP has been reported to develop after various surgical procedures, including arthroplasty, grafting, foot surgery, abdominal surgery, and amputation and at ostomy sites.[3-9] Our patient's course

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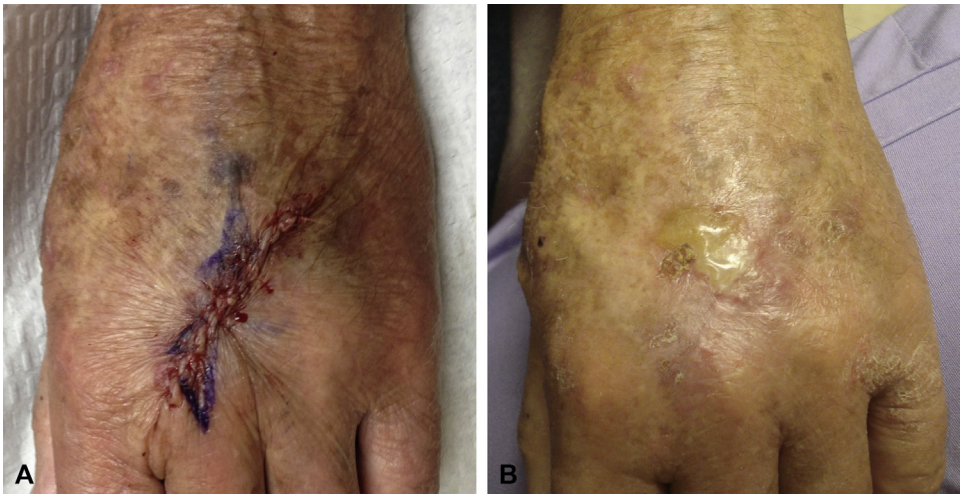

Fig 1. Mohs micrographic surgery site. **A**, Layered repair on day of MMS. **B**, Serous bulla 1 month after Mohs micrographic surgery.

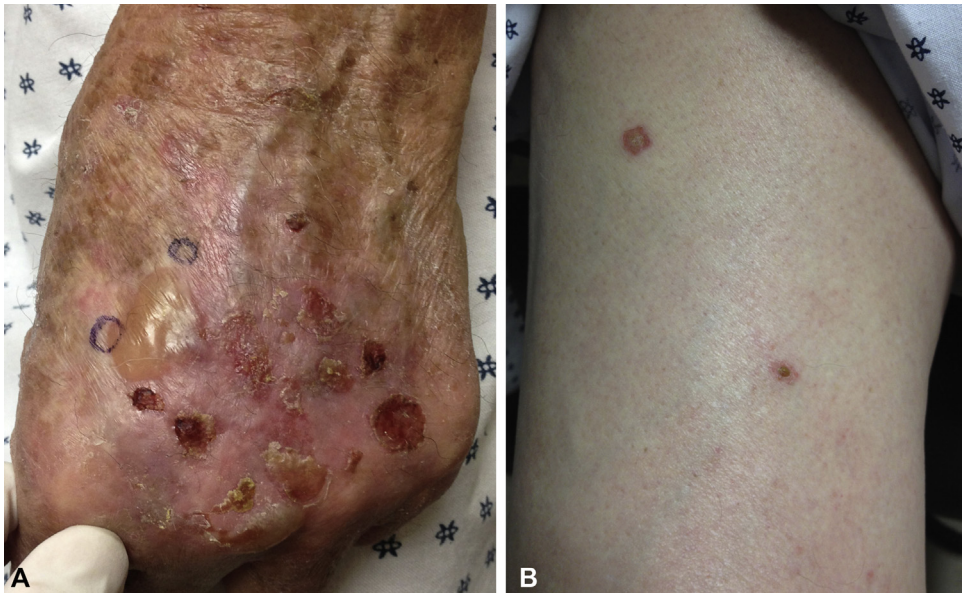

Fig 2. Three-month follow-up. **A**, Mohs micrographic surgery site with bulla and crusted plaques. **B**, Contralateral thigh with evolving bullae.

mirrored that of other reported postsurgical cases, beginning in the surgical incision before generalizing.[4,8] BP can develop within several days after the implicated trauma or surgery.[4,8] Our patient's eruption arose within weeks of MMS, similar to the majority of trauma-induced BP reports we reviewed.[4,5,7-9]

It is uncertain whether MMS was causal or coincidental to BP developing in this patient. Two hypotheses have been presented to explain trauma-induced BP. First, the patient may have had low titers of circulating autoantibodies and subclinical disease unmasked by the surgical event. Incision and subsequent wound repair would trigger an inflammatory cascade involving granulocyte recruitment and cytokine-induced vascular permeability. This would promote circulating autoantibody binding of exposed antigens with complement activation and subepidermal cleavage.[2] Alternatively, cutaneous wounding may expose epitopes from the basement membrane zone with de novo and pathologic development of antibodies in susceptible patients.[1-3] Disease generalization, the patient's age, and his history of prior, uneventful skin cancer surgeries support the former hypothesis. This model is further supported by studies showing that the BP

phenotype will develop after UV exposure in animals with transferred or induced BP autoantibodies.[10,11]

When surgery-induced BP occurs, the subsequent duration of therapy is variable. Kim et al[5] reported a patient in whom BP developed after total knee arthroplasty. After the diagnosis, a course of corticosteroid therapy was initiated, with resolution of findings within 4 months. The patient underwent contralateral knee arthroplasty 1 year later with BP recurring within 2 days after surgery. All symptoms resolved after 2 weeks of prednisolone. The authors posited that early diagnosis and intervention after surgery-related BP may decrease the duration of therapy. Our patient underwent MMS for an unrelated keratinocyte carcinoma on the ear 3 months after initiating BP therapy. His BP was well controlled at that time and was given 40 mg of prednisone for 3 days beginning on the day of surgery without any flare.

Dermatologic surgeons should be aware of the potential for induction of BP at cutaneous surgery sites. There is considerable overlap between the BP-susceptible population and those undergoing MMS. New onset BP should be considered when an unusual postoperative bullous eruption occurs. In patients with a confirmed diagnosis of BP who are undergoing skin cancer procedures, surgeons may consider short courses of corticosteroids perioperatively in anticipation of flares.

Conflicts of interest

None disclosed.

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