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Refractory Bullous Pemphigoid Ulcer Successfully Treated With Dermal Substitute: A Case Report

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Abstract

Bullous pemphigoid (BP) is an autoimmune blistering disorder of the dermoepidermal junction, observed in individuals over the age of 70 years, and is conventionally managed with corticosteroids. In some cases, the traditional approach of medical management in combination with wound care is insufficient to resolve the persistent bullae and ulcers characteristic of the disease. We describe here the disease course, diagnosis, and treatment of a 57-year-old male with a four-year history of BP and treatment-resistant ulcers of the lower extremities. The wound addressed in this case was located on the distal anterior left lower extremity and further complicated by uncontrolled diabetes. The patient was managed in accordance with current guidelines under the care of a multidisciplinary team without resolution of the wound. Ultimately, success was found with a surgical approach using the dermal substitute, Integra® Bilayer Matrix Wound Dressing (Integra LifeSciences, Plainsboro, New Jersey).

Categories: Plastic Surgery
Keywords: autoimmune blistering disorder, bullous pemphigoid, chronic ulcers, dermal substitute, integra, lower extremity wounds, negative pressure wound therapy, refractory disease, surgical reconstruction, wound healing

Introduction

Bullous pemphigoid (BP) is an autoimmune subepidermal blistering disorder characterized by tense bullae and pruritus, often observed in individuals over the age of 70 years [1,2]. The annual incidence of BP is 2.4-23 new cases per million people in the general population, based on European studies [3,4].

The European Academy of Dermatology and Venereology (EADV) suggests medical management in combination with wound care, emphasizing corticosteroids as the mainstay of treatment [5-9]. In most cases, medical management is sufficient treatment of disease; however, some patients continue to have persistent ulcers that are unresponsive to conservative measures, requiring alternative treatment options.

With the limited amount of literature on management of nonhealing ulcers in the setting of BP, we present a case where Integra® (Integra LifeSciences, Plainsboro, New Jersey) dermal substitute was used in the successful treatment of a persistent wound of the anterior shin after a year of medical management in accordance with current guidelines.

Case Presentation

A 57-year-old male presented for the management of multiple nonhealing ulcers of the lower extremities. The patient has a history of BP, hypertension, paroxysmal atrial fibrillation, and type 2 diabetes mellitus with poor glycemic control.

The patient initially developed skin ulcerations and pruritus three years before seeking medical care. He was first evaluated by dermatology, where BP was diagnosed based on biopsy findings demonstrating a linear IgM U-serrated pattern and positive C3 deposition along the basement membrane zone. Further evaluation with enzyme-linked immunosorbent assay (ELISA) was positive for BP180 antibodies. Indirect immunofluorescence was negative at the time of evaluation; however, the patient was concurrently taking oral prednisone, which may have influenced the results.

Over the following year, the patient was managed primarily by outpatient dermatology with various treatment modalities, including Unna boot compression dressings with triamcinolone, oral prednisone, mycophenolate, and doxycycline. Specifically, dressing changes occurred weekly with Vaseline and triamcinolone ointment, and collagenase ointment applied to each ulcer to help debride the slough. The dressing applied was a multilayer compression system consisting of calamine/zinc, Kerlix gauze, and 4-inch Coban, wrapped in three separate layers from the base of the toes to the knee. Despite these interventions,

the patient continued to develop persistent ulcerations, predominantly involving the lower extremities, and subsequently developed osteomyelitis of the left heel. This progression necessitated multidisciplinary surgical management, including debridement by orthopedic surgery and subsequent wound coverage by plastic surgery.

The patient had been tapered off oral prednisone six months before surgery. At the time of surgical intervention, approximately one year after his initial dermatologic evaluation, his BP was being managed with mycophenolate, doxycycline, and niacinamide. Two wounds were addressed during the procedure performed by plastic surgery, one heel defect measuring 4x8 cm and one anterior shin defect measuring 4x3 cm. Orthopedic surgery performed calcaneal debridement and washout of the heel, which was then managed with an anterolateral thigh free flap [10]. The second wound addressed during the procedure was 4x3 cm on the anterior shin without underlying bone exposure. Sharp debridement was performed, followed by coverage with a dermal substitute secured with 3-0 chromic sutures and bolstered with negative pressure wound therapy. Following negative pressure wound therapy, wound care consisted of a silicone foam dressing and compression wrap to manage lower extremity edema. At four-month follow-up, the wounds demonstrated re-epithelialization without evidence of recurrent ulceration or infection. No BP flare or complications were reported during the follow-up period (Figures *1*-*4*).



FIGURE 1: Left distal anterior leg preoperatively (4×3 cm). Proximal on the left, distal on the right



FIGURE 2: Left distal anterior leg two weeks after debridement and placement of Integra® Bilayer Matrix Wound Dressing. Proximal on the left, distal on the right



FIGURE 3: Left distal anterior leg three weeks after debridement and placement of Integra® Bilayer Matrix Wound Dressing. Proximal on the left, distal on the right



FIGURE 4: Left distal anterior leg four months after debridement and placement of Integra® Bilayer Matrix Wound Dressing. Proximal on the left, distal on the right

Discussion

Dermal substitutes support healing by providing an acellular scaffold for revascularization and neodermal formation [11,12]. These are commonly used as adjuncts in burn reconstruction and complex wound management [13-16]. The decision to use a dermal substitute in this case stems from a combination of the complexity of the patient's disease and the pathophysiology of BP itself.

Since BP occurs at the dermoepidermal junction and can be precipitated by surgery or at skin graft donor sites, Integra® Bilayer Matrix Wound Dressing was chosen for this patient rather than a traditional split-thickness or full-thickness skin graft to mitigate the risk of trauma-induced disease exacerbation at the

donor site [17,18]. This dermal substitute is composed of a bilaminate sheet of bovine tendon collagen and chondroitin-6-sulfate glycosaminoglycans covered by a semipermeable silicone sheet. In this case, negative pressure wound therapy was used in conjunction with the dermal substitute to increase adherence to the wound bed and to promote cellular migration and neovascularization through the dermal matrix [19].

In this case, the combination of a dermal substitute and negative pressure wound therapy provided a unique treatment alternative that facilitated successful healing of the patient's persistent ulcer despite the refractory nature of the underlying disease process.

Literature review

Epidemiology/Prevalence

BP is an autoimmune subepidermal blistering disorder first described by Lever in 1953, characterized by tense bullae-containing eosinophil-rich infiltrate and severe pruritus [20-22]. BP is commonly observed in older adults over the age of 70 years and generally resolves spontaneously over the course of a few months, but many cases require treatment and may last for years [1,2].

Pathophysiology

BP is a type II hypersensitivity reaction of immunoglobulin G (IgG) against the hemidesmosomal anchoring proteins BP antigen 180 (BP180, type XVII collagen) and BP antigen 230 (BP230) [23]. The binding of autoantibodies activates complement protein C3, triggering an inflammatory cascade [24]. The inflammatory response is predominantly eosinophilic, accompanied by a limited amount of neutrophil involvement. The degranulation of these cells releases cytokines, chemokines, and proteolytic enzymes such as metalloproteinase 9, neutrophil elastase, and reactive oxygen species [25]. The destruction of hemidesmosomal anchoring proteins leads to subepidermal blister formation [26].

Although rare, BP may present in childhood. Drug-induced BP can be observed in younger patients who have been exposed to drugs such as nonsteroidal anti-inflammatory drugs, amoxicillin, programmed cell death protein 1/programmed death-ligand 1 inhibitors, gliptins, tumor necrosis factor-a inhibitors, spironolactone, and furosemide [27]. Beyond drug exposure, BP has been associated with UV radiation exposure, burns, surgery, and specific human leukocyte antigen class II alleles [28]. Skin grafting also may precipitate BP at the donor site [18,29,30].

Clinical Presentation/Diagnosis/Lab and Histology Analysis

Subepidermal blisters and inflammation are the hallmarks of BP. Large tense bullae are observed on normal or erythematous skin, accompanied by pruritus. Pruritus may precede the eruption of bullae by several months. In 80%-90% of cases, BP spares the head, neck, and mucosal tissues and is associated with a negative Nikolsky sign: lateral pressure to skin does not cause the epidermis to blister or shear [25].

Histologic examination reveals inflammatory cell infiltrate-containing eosinophils. Direct immunofluorescence of punch biopsies shows linear deposits of complement (C3) and IgG along the basement membrane and is the gold standard for evaluation of autoimmune blistering disorders [31]. In most cases, indirect immunofluorescence shows that circulating IgG binds the epidermal basement membrane [31]. Immunohistochemical staining of formalin-fixed biopsies for C3d and C4d can support the diagnosis if paraffin-embedded tissue is available. ELISA testing may be used to detect antibodies to the NC16A domain of the transmembrane BP180 protein, with a sensitivity of 85% and specificity of 98% [26,32].

Management

Management of BP is largely guided by severity, with corticosteroids being the mainstay of treatment; they work to control the immune response and nonspecifically decrease autoantibody production [33,34]. Superpotent (class I) topical corticosteroids such as clobetasol are used as initial treatment, particularly for localized disease [5-7]. The alternative of oral corticosteroids such as prednisone offers a lower cost and better patient adherence due to ease of use and is generally used for moderate-to-severe disease [8,9]. Resolution of clinical disease is achieved in a few weeks, indicated by a lack of new lesion formation, existing blisters beginning to heal, and a significant decrease in pruritus and inflammation. This is followed by a taper of corticosteroids over several months [7].

Immunosuppressive therapy is used as a second-line treatment after failure of corticosteroids or in patients with contraindications to steroid use. In addition, immunosuppressive therapies spare the patient from long-term corticosteroid use. Methotrexate, mycophenolate mofetil, and azathioprine are used for extensive disease [35,36]. Nonimmunosuppressive therapies may be used, such as doxycycline plus nicotinamide, dapsone, and dupilumab. In refractory cases, biologics may be used [37-43]. Rituximab targets IgG, and omalizumab is used in patients with high IgE levels and eosinophilia [44]. Intravenous immune globulin lowers serum levels of BP180 and BP230. Dupilumab slows eosinophil chemotaxis and inhibits preactivated

B-cell proliferation, showing a promising alternative in a recent case series [44-46].

Long-term steroid and immunosuppressant use presents wound healing issues such as skin atrophy, diabetes, and increased infection risk [7,47]. Steroids have an inhibitory effect on collagen synthesis by fibroblasts, which can be reversed with oral vitamin A to augment epithelialization and fibroblast proliferation. There is also concern about infection in patients with open wounds on immunosuppressive therapy.

Experimental adjunctive treatments are being investigated to address wound healing in BP. Ozone liquid dressings provide a molecule of three oxygen atoms in a liquid dressing that facilitates antibacterial activity and immunomodulatory effects, acting to facilitate wound healing [34,48]. Recombinant human type XVII collagen used in combination with systemic glucocorticoids has been shown to promote proliferation of primary human keratinocytes in patients with BP [49]. Surgical management is another area of exploration for management of BP, including free flap reconstructions and split-thickness skin grafts [50]. Skin grafting, in particular, is unfavorable due to the risk of disease exacerbation at the donor site [18].

Conclusions

Chronic wounds associated with BP are complex, and there is limited literature available specifically focused on surgical management of these nonhealing ulcers. The mainstay of treatment with corticosteroids is essential to control the overactive immune system at the root of the disease but carries the risk of skin atrophy and delayed wound healing. In this case, where medical management and wound care failed, success was found in a surgical approach. This case demonstrates the collaboration of a multidisciplinary team and an instance where a dermal substitute provided substantially better healing for a complex patient with treatment-resistant BP wounds.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

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