

Management of chronic wounds in patients with pemphigus

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Abstract: Pemphigus is a group of chronic IgG-mediated autoimmune blistering diseases that involves both the skin and mucous membranes. It is caused by autoantibodies disrupting intercellular keratinocyte adhesion. Loss of epidermal integrity clinically manifests as flaccid thin-walled blisters, erosions, and crusts on the skin, and painful mucosal erosions. Pemphigus vulgaris and pemphigus foliaceus are the two most common types of pemphigus. Although rare, pemphigus can be life-threatening. Substantial morbidity and mortality can occur as a result of complications of these diseases and their treatments. In general, pemphigus often results in extensive skin loss, leading to scar formation and adherence of articular skin folds, limiting joint and limb movements. The primary goal of treatment is to induce durable remission, decrease blister formation, prevent infections and promote healing of blisters and erosions. The mainstay of treatment for pemphigus is systemic corticosteroids combined with an adjuvant immunosuppressant and wound care. We performed PubMed and Google Scholar searches of the English-language literature for pemphigus disease and wound management for blistering disease (1966–2019). In this review, we discuss wound care in patients with pemphigus disease.

Keywords: pemphigus, chronic wounds, blisters, dressing, wound healing, infection

Introduction

Pemphigus is a group of rare yet potentially fatal IgG-mediated blistering autoimmune diseases affecting the skin and mucous membranes.¹ The term pemphigus was derived from the Greek word “pemphix” meaning bubble or blister.² Pemphigus can be grouped into four major types: vulgaris, foliaceus, paraneoplastic, and IgA pemphigus. Pemphigus vulgaris (PV) is the most prevalent of the autoimmune bullous diseases in the pemphigus family. In PV, the blister occurs in the deeper part of the epidermis, just above the basal layer, and in pemphigus foliaceus (PF), also called superficial pemphigus, the blister occurs in the granular layer. PV is characterized by the presence of circulating IgG autoantibodies against desmoglein, a keratinocyte cell surface molecule, leading to disruption of normal cell adhesion between keratinocytes, and hence separation and rounding up of epidermal cells (acantholysis) and intraepidermal clefting leading to blister formation. The target antigens in PV are desmoglein (Dsg) 1 and 3. In the mucocutaneous type, autoantibodies against Dsg1 and Dsg3 are present, whereas the mucosal-dominant type of PV is caused by autoantibodies against Dsg3. PF is characterized by cutaneous involvement only and is caused by autoantibodies directed against Dsg1, leading to subcorneal blisters.³

The incidence rate of PV is estimated to be between 0.1 and 0.5 per 100,000 people per year. Pemphigus is more common in adults but can occur at any age. The average

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age of onset is 40–60 years for PV and nonendemic PF.⁴ Overall, the sex ratio for PV and PF appears to be equivalent or close to equivalent.⁵ It is rare in young people.

Clinical manifestations

The disease has a chronic course. The primary lesion of PV is a flaccid blister, which may occur anywhere on the skin surface, but typically not the palms and soles.⁶ The skin lesions in PV can be pruritic or painful. Because the blister is forming in the epidermis, it is flaccid and easily ruptured and hence, the most common skin lesions observed in patients are moist erosions resulting from broken blisters.⁷ These erosions are often quite large, as they tend to spread at their periphery. Large areas of the epidermis can denude, creating risk for bacterial infection and sepsis. Some patients will only have mucosal involvement, usually in the oral cavity.⁶ In the mouth, short-lived bullae quickly rupture to involve most of the mucosa with slow-healing painful erosions. A characteristic finding in pemphigus patients is that erosions can be extended into visibly normal skin by pulling the remnant of the blister wall or rubbing at the periphery of active lesions; additionally, erosions can be induced in normal-appearing skin distant from active lesions by lateral pressure or mechanical shear force. This phenomenon is known as the Nikolsky sign.¹ This sign helps differentiate pemphigus from other blistering diseases of the skin such as pemphigoid; however, similar findings also can be elicited in staphylococcal scalded skin syndrome, Stevens-Johnson syndrome, and toxic epidermal necrolysis. Exposure to ultraviolet radiation may intensify disease activity.⁸

PF typically presents in a seborrheic distribution. The scalp, face, and trunk are common sites of involvement.⁹ Lesions usually start on the trunk. The skin lesions typically consist of small, scattered superficial blisters that rapidly evolve into scaly, crusted erosions. Because the blisters are superficial, patients often appear to have impetiginized eczema of the face and upper trunk. Nikolsky sign is often present in active disease. The lesions may remain localized or may coalesce to cover large areas of skin. In its most severe form, PF progresses to involve the entire skin surface as an exfoliative erythroderma.¹⁰ The erosions may cause local pain and/or burning sensation.⁹ Unlike PV, PF very rarely has mucosal lesions. Systemic symptoms are usually absent. Key clinical and laboratory findings in different types of pemphigus are listed in (Table 1).^{2,3,11}

Pharmacological treatment

The approach to the treatment of PV and PF is similar. Treatment is aimed at achieving a rapid and sustained remission with the resolution of cutaneous lesions and elimination of circulating antibody, with the eventual goal of maintenance with low-dose therapy, or ideally tapering off all treatment.¹ Without treatment, PV does not spontaneously remit. The mortality of PV was 75% on average before the introduction of corticosteroids (CS) in the early 1950.¹² The management approach of PV may be considered in 2 general phases: remission induction and remission maintenance. It is important to note that relapse rates can be high initially (up to 47% if treatment is stopped after 1 year). If PV is localized in distribution and mild in severity, therapy with potent topical corticosteroids can be attempted. In moderate to severe cases, systemic corticosteroids with adjuvant immunosuppressants are the primary therapy (Table 2).^{13–17} Clinical response may be observed within days of starting treatment, although cessation of new blister formation may take a few weeks,^{18,19} with complete healing occurring at 6–8 weeks.²⁰ Rituximab can also be considered as first-line therapy.¹⁶ Patients requiring long-term use of corticosteroids and/or cytotoxic drugs must be monitored carefully for adverse effects. Sun avoidance and sun-protection measures (eg, using sunscreens, wearing barrier clothing) should be recommended. Following successful therapy, cutaneous manifestations of pemphigus tend to resolve in lighter skin phototypes, whereas post-inflammatory pigmentary changes often occur in patients with darker skin phototypes.

Oral mucosal lesions are usually painful during active disease. Patients should maintain oral hygiene (toothbrushing and antiseptic mouthwashes) and avoid spicy, sharp, very hot, or abrasive foods.²¹ Topical anesthetics such as topical lidocaine 2% gel or solution can be applied to reduce pain.⁷ Medium or high potency topical corticosteroids have shown to improve oral symptoms of pemphigus in some patients.^{21,22} In one case report, topical trichloroacetic acid (TCA) 33% seemed promising for the treatment of persistent, old, and refractory oral PV lesions in a patient who is otherwise in clinical remission.²³

PV and PF are associated with considerable morbidity and mortality.² Mortality has considerably decreased to 1.6–12% since the regular use of corticosteroids and adjuvant immunosuppressants was introduced but is approximately 2–3-fold higher in patients with pemphigus than in the general population.² The most frequently reported causes of death in

Table 1 Key clinical and laboratory findings in pemphigus disease

	Epidemiology	Autoantigens	Clinical features	Histopathology	Direct immunofluorescence	Indirect immunofluorescence	Variants
Pemphigus vulgaris	Primarily middle-aged adults	Desmoglein 1 and desmoglein 3, others	Mucosal erosions; flaccid bullae and erosions on skin	Suprabasal acantholysis; "row of tombstones" pattern of basal keratinocytes	Intercellular deposition of IgG	Intercellular deposition of IgG; monkey esophagus is preferred substrate	Pemphigus vegetans and pemphigus herpetiformis
Pemphigus foliaceus	Primarily middle-aged adults; endemic form most common in children and young adults	Desmoglein 1	Fragile blisters, shallow erosions, erythematous patches, crusts; mucosal involvement absent	Subcorneal or granular layer acantholysis	Intercellular deposition of IgG	Intercellular deposition of IgG; normal human skin or guinea pig esophagus is preferred substrate	Endemic pemphigus foliaceus (fogo sevalgem), pemphigus erythematosus, and pemphigus herpetiformis
Paraneoplastic pemphigus	Primarily adults, but may occur at any age	Envoplakin, periplakin, others	Severe stomatitis, variable cutaneous findings (eg, blisters, erosions, lichenoid lesions), bronchiolitis obliterans	Variable findings; suprabasal acantholysis, keratinocyte necrosis, and lichenoid interface dermatitis are most common	Intercellular and/or basement membrane zone deposition of IgG and/or C3	Intercellular deposition of IgG; rat bladder is preferred substrate	
Subcorneal pustular dermatosis-type IgA pemphigus	Any age	Desmoglein 1	Vesicles, pustules, crusts on skin; annular, circinate, or herpetiform morphology; mucosal involvement usually absent	Subcorneal clefts and pustules; minimal acantholysis; mixed infiltrate in dermis	Intercellular deposition of IgA	Intercellular deposits of IgA on monkey esophagus*	
Intraepidermal neutrophilic dermatosis	Any age	Poorly defined	Vesicles, pustules, crusts on skin; annular, circinate, or herpetiform morphology; mucosal involvement usually absent	Intraepidermal pustules; minimal acantholysis; mixed infiltrate in dermis	Intercellular deposition of IgA	Intercellular deposits of IgA on monkey esophagus*	

Notes: *Indirect immunofluorescence is negative in around 50 percent of patients with IgA pemphigus. Test availability restricted to specialized laboratories. Reproduced with permission from: Hertl M, Sitaru C. *Pathogenesis, clinical manifestations, and diagnosis of pemphigus*. In: UpToDate, Post TW (Ed), UpToDate, Waltham, MA. (Accessed on July 22, 2019). 11 Copyright © 2019 UpToDate, Inc. For more information visit www.uptodate.com.

Table 2 Pharmacological treatment for pemphigus

Topical options	First line	Other corticosteroid-sparing agents
For isolated lesions: Potent topical or intralesional steroids (clobetasol, betamethasone, triamcinolone)	- Oral prednisone (0.5–1.0 mg/kg every 24 hrs as a single dose) – may increase every 5–7 days if blistering continues, or - Pulsed intravenous prednisone for initial treatment of severe disease, or - Anti-CD20 monoclonal antibodies: Rituximab (2 infusions of 1 g, 2 weeks apart – rheumatoid arthritis protocol)	If treatment failure occurs with a first-line adjuvant drug, consider switching to an alternate adjuvant agent: - Methotrexate - Cyclophosphamide - Plasmapheresis or plasma exchange - Intravenous immunoglobulin (IVIg) - Dapsone
For oral lesions (paste or mouthwash): - Triamcinolone acetonide 0.1% in adhesive paste (Adcortyl in Orabase®) - Benzydamine hydrochloride 0.15% (Diffiam Oral Rinse®) - Betamethasone sodium phosphate 0.5 mg dissolved in 10 mL water - Topical cyclosporine (5 mL of 100 mg/mL as mouthwash) - Chlorhexidine gluconate 0.2% (Corsodyl), hexetidine 0.1% (Oraldene®) antiseptic mouthwash	May be combined with corticosteroid-sparing agents: - Azathioprine 2–3 mg/kg daily, or - Mycophenolate mofetil 2–3 g daily	

Note: Data from Murrell et al,¹³ Ahmed et al,¹⁴ Gregoriou et al,¹⁵ Joly et al¹⁶ and Kridin.¹⁷

patients with pemphigus include septicemia, respiratory tract infections, and cardiovascular disease.^{2,24}

Wound management

The four main goals of wound care are (1) prevention of infection, (2) maintenance of a moist environment, (3) protection of the wound, and (4) minimizing scar formation.²⁵ small blisters should be left intact, to prevent secondary infection. However, large blisters should be punctured and aspirated with a large (18-gauge) sterile needle, to keep the blister roof in place for wound protection. Gentle cleansing is an early priority and can be accomplished by normal saline or antibacterial soap/antiseptic twice a day. Allow it to dry well. Next, a bland emollient ointment, such as 50% white soft paraffin +50% liquid paraffin, is applied directly to the wound bed, or apply the ointment to a dressing. If a wound is wet, the dressing should absorb excess exudate. If a wound is dry, the dressing should provide moisture to the wound bed.²⁶

Pemphigus is characterized by superficial wounds, where the damage is generally limited to the epidermis and dressing is comparable to dressing surgical wounds except that extra attention should be paid to early closure and prevention of fluid losses through evaporation. This can be accomplished by using moisture retentive dressings, either occlusive or semioclusive, help promote reepithelialization and help restore the skin barrier. Non-adherent gauze, transparent film, hydrocolloid, and hydrogel dressings may be used directly on the wounds. When selecting a dressing, one should take into account not only wound characteristics, but also specific dressing features such as absorptive capacity, hydrating quality, adhesive quality, and comfortability. Various types of wound dressing are highlighted in (Table 3).^{10,27–30} All dressing products used for pemphigus patients must be non-adherent.

Films are thin, elastic dressing made of see-through transparent material (generally composed of polyurethane), available in a wide variety of shapes and sizes to conform to different wounds. They are excellent for erosions and superficial ulcers.²⁸ Films provide a barrier to shield from bacterial invasion.¹⁰ They are waterproof but provide one-way exit passage for gas (CO₂) and excess moisture. Films are non-absorbant and are only suitable for delicate and minimally exudative, superficial wounds. It is not recommended to use films in high-exudate wounds. To avoid potential tearing or damage to the newly-formed epidermis, films should be non-adherent to

the wound area, especially in patients with fragile skin, including the elderly or patients with cutaneous atrophy.¹⁰

Hydrogel dressings are made of polyethylene and up to 97% water. They are available in two main forms, sheets (traditional form) and amorphous form. They are useful for dry wounds as they provide a moist environment. Hydrogels also have a marked cooling and soothing effect on the skin, which is valuable in burns and painful wounds. Most hydrogels are nonadhesive, thus requiring a secondary dressing.²⁶ Xeroform gauze or a film can be placed over the hydrogel sheet to stabilize it and to prevent it from drying out.³¹ Hydrogels should not be used on infected or heavily draining wounds since they are not absorptive.²⁶

Hydrocolloid dressings are available in two forms: sheets and a hydrocolloid gel. Both are composed, for the most part, of carboxymethyl cellulose, gelatin, and pectins. The sheet hydrocolloid dressing has an external semipermeable layer and an internal layer of hydrophilic carboxymethyl cellulose molecules suspended in a hydrophobic mass of gelatin and pectins.¹⁰ Hydrocolloid dressings are a cost-effective and convenient option because they can be worn for several days before changing. In addition to having some absorptive properties, they seem particularly useful in causing painless debridement. However, hydrocolloids may cause contact dermatitis. Many hydrocolloids contain adhesives which could damage fragile skin in pemphigus. Hydrocolloid dressings may also produce a malodorous yellow gel on the inner side of the dressing referred to as “gel and smell.” Patients should be informed to expect this as it may be confused with infection.¹⁰

Polymeric membrane dressings (PMDs) are multifunctional dressings composed of a hydrophilic polyurethane membrane matrix with a continuous semipermeable polyurethane film backing, which comes in different thicknesses based on wound exudate.¹⁰ PMDs have been used successfully in superficial abrasions without overdrying.^{32,33} PMDs contain ingredients that work synergistically to cleanse wounds and expedite healing continuously.³⁴

When the disease becomes severe with extensive skin involvement, patients are typically admitted to the burn unit. Therefore, supportive care resembles that performed for severe thermal burns TEN. It aims at minimizing potential complications, which may ultimately lead to patient mortality. For instance, it aims to avoid hypovolemia, electrolyte imbalance, renal insufficiency, and sepsis

Table 3 Properties of dressing materials and topical agents

Type	Actions	Indications/use	Precautions/contraindications	Brand Names
Alginates	Absorb fluid Promote autolytic debridement Conformability to wound bed	Moderate to high exuding wounds. Special cavity presentations in the form of rope or ribbon. Can combine with silver for antimicrobial activity.	Do not use on dry/necrotic wounds. Use with caution on friable tissue (may cause bleeding). Do not pack cavity wounds tightly.	Algiderm® (Bard) Algisorb™ (Calgon-Vestal) Algosteril® (KCI) Curasorb™ (The Kendall Co.) Kalginate® (DeRoyal) Kaltostat® (ConvaTec)
Foams	Absorb fluid Moisture control Conformability to wound bed	Moderate to high exuding wounds. Special cavity presentations in the form of strips or ribbon. Can combine with silver or PHMB for antimicrobial activity.	Do not use on dry/necrotic wounds or those with minimal exudate. Low-adherent versions are available for patients with fragile skin.	Allewyn® (Smith & Nephew United) Curafoam™ (The Kendall Co.) Hydrasorb® (Tyco/Kendall Co.) Mepilex® (Molnlycke Health Care) V.A.C. GranuFoam™ (KCI)
Hydrocolloids	Absorb fluid Promote autolytic debridement	Clean, low to moderate exuding wounds. Combined presentation with silver for antimicrobial activity.	Do not use on dry/necrotic wounds or high exuding wounds. May encourage overgranulation. May cause maceration.	Comfeel® (Coloplast Sween, Inc.) Cutinova® (Smith & Nephew) Duoderm® (ConvaTec) NuDerm® (KCI)
Hydrogels	Rehydrate wound bed Moisture control Promote autolytic debridement Cooling sensation	Dry/low to moderate exuding wounds. Combined presentation with silver for antimicrobial activity.	Do not use on highly exuding wounds or where anaerobic infection is suspected. May cause maceration.	Clearsite® (Conmed Corp.) Curafill™ (The Kendall Co.) Elasto-Gel™ (SW Technologies) Normgel® (SCA Hygiene Products) 2nd Skin® (Spenco Medical, Ltd.) Transigel™ (Smith & Nephew) Vigilon® (CR Bard)
Iodine	Antimicrobial action	Low to high exuding wounds. Critically colonized wounds or clinical signs of infection.	Do not use on dry necrotic tissue. Do not use with known sensitivity to iodine. Short-term use recommended (reduce systemic absorption).	Iodosorb gel (Cadexomer iodine)

(Continued)

Table 3 (Continued).

Type	Actions	Indications/use	Precautions/contraindications	Brand Names
Low-adherent wound contact layer (silicone)	Protect new tissue growth Atraumatic to peri-wound skin Conformable to body contours	Low to high exuding wounds. Use as a contact layer on superficial low exuding wounds.	May dry out if left in place for too long. Do not use with known sensitivity to silicone.	ADAPTIC TOUCH™ (KCI) Cardinal Health™ Silicone Contact Layers (Cardinal Health) ComfiTel™ (DermaRite Industries) Cuticell® Contact (Essity) Mepitel® (Mölnlycke Health Care)
Odor control Dressing	Odor absorption	Malodorous wounds (due to excess exudate). May require additional antimicrobial if odor due to increased bioburden.	Do not use on dry wounds.	Exuderm Odorshield Dressing (Medline) CarboFLEX® (Convatec) ACTISORB® Silver 220™ (KCI)
PHMB (polyhexamethylene biguanide)	Antimicrobial action	Low to high exuding wounds. Critically colonized wounds or clinical signs of infection. May require secondary dressing.	Do not use on dry/necrotic wounds. Do not use with known sensitivity to PHMB.	Kerlix AMD™, Excilon AMD™, Telfa AMD™ (Tyco HealthCare Group) XCell® (Xylos Corp, Langhorne, Pa)
PMD (Polymeric membrane dressings)	Wound cleansing Absorb fluid Provide hydration Decrease pain	Exudative wounds. Venous, diabetic or pressure ulcers of all stages. Chronic wounds. Infected Wounds.	May require multiple dressing changes for high exudative wounds. Can be used on dry or infected wounds unlike traditional foam dressings.	Polymem® (Ferris Corp.)
Polyurethane film	Moisture control Breathable bacterial barrier Transparent (visualization of wound)	Primary dressing over superficial low exuding wounds. Secondary dressing over alginate or hydrogel for rehydration of wound bed.	Do not use on patients with fragile/compromised peri-wound skin.	BIOCLUSIVE™ (KCI) Finesse PU Film Dressing (Finesse Medical, LTD) Kendall™ Window Transparent Film Dressing (Cardinal Health)
Protease modulating	Active or passive control of wound protease levels	Cleans wounds that are not progressing despite correction of underlying causes, exclusion of infection, and optimal wound care.	Do not use on dry wounds or those with leathery eschar.	PROMOGRAN™ (KCI)
Silver	Antimicrobial action	Critically colonized wounds or clinical signs of infection. Low to high exuding wounds. Combined presentation with foam and alginates/CMC for increased absorbency. Also in paste form.	May cause discoloration. Do not use if known sensitivity to silver. Discontinue after 2 weeks if no improvement and reevaluate.	Aquacel Ag® (ConvaTec) Acticoat 7 (Smith and nephew) ACTISORB® Silver 220 (KCI) Silversorb (medline) Silvercel (KCI)

Note: Data from Dabiri et al,¹⁰ Armstrong,²⁷ Powers et al,²⁸ Broussard and Powers,²⁹ and Borda et al.³⁰

Approach to infected or colonized lesions

Superimposed infections are often a significant cause of morbidity and mortality in pemphigus, either secondary to the breakdown of the skin barrier or immune suppression related to treatment. Bacterial skin colonization and infection may render pemphigus refractory to treatment by preventing or delaying wound healing.³⁵ The most frequently reported cause of death in pemphigus is septicemia which is usually secondary to cutaneous *Staphylococcus aureus* infection.³⁶ Prevention of infections is a crucial factor in the overall clinical outcome. An ideal dressing should decrease the microbial burden without causing cytotoxic damage. Patients should be encouraged not to “pop” or drain blisters since this could provide a route for infection. Patients should also be informed about the warning signs of infection to facilitate prompt diagnosis and early treatment.

Infected wounds clinically present with erythema, warmth, edema, and pain or local tenderness. Purulence, increased wound drainage and new or worsening malodor can occur. Systemic signs such as fever and leukocytosis are indicators of progression to bacteremia or septicemia. In such cases, systemic antibiotics are warranted.³⁷

Culture and sensitivity should be considered to determine the most appropriate antibiotic for an infected wound. Superficial swab cultures are of limited utility because wounds, like skin, are covered with transient bacteria rendering the isolation and accurate identification of the pathogenic bacteria impossible. Quantitative swab cultures can help to identify the pathogenic microorganism.¹⁰ It is known that a quantitative culture of 1×10^6 organism/gram of tissue confirms the diagnosis of infection.³⁸

The presence of bacteria in a wound can be grouped into three distinct situations (a) contamination: the presence of bacteria on the surface without multiplication, and the absence of clinical disease. (b) colonization: the bacteria multiply, but without signs and symptoms of infection, and (c) infection: the proliferation of bacteria associated with local host reaction, delayed healing and tissue damage.^{39,40}

In critically colonized wounds, bacteria may delay wound healing without apparent signs of infection. Mild topical antiseptics frequently used to cleanse wounds include Dakins solution (bleach and water), eusol solution (bleach, boric acid, and water), and acetic acid (one tablespoon white vinegar in a cup of water). These solutions

cannot be used for a prolonged period of time due to their ability to cause cytotoxicity (each dressing change for no more than 10 min).

Topical antimicrobials are useful when the sensitivity of the organism is determined. Both Mupirocin and retapamulin are effective against gram-positive organisms.^{41,42} Topical metronidazole provides good anaerobic coverage. These agents can be applied directly to the wound bed with appropriate covering dressings.

Iodine - and silver-based dressings are commonly used as antimicrobial dressings in the management of wounds that are at risk of infection. These dressings have been formulated to be noncytotoxic.¹⁰ Cadexomer iodine (eg, Iodosorb) is a slow-release antimicrobial and has shown to be bactericidal against all gram-positive and gram-negative bacteria as well as fungi, and it provides a moist wound environment.⁴³ A recent meta-analysis reported that cadexomer iodine dressings might be associated with better healing compared with standard of care.⁴⁴

Silver is an antimicrobial agent with broad-spectrum bactericidal activity. Silver-based dressings are available in different forms such as foams, hydrofibers, and hydrocolloids.²⁶ An international consensus on the appropriate use of silver dressings recommends that silver dressings be used for infected wounds or wounds that are at high risk of infection for an initial period of 2-week, after which the wound should be reevaluated to decide if a silver dressing remains appropriate or if a more aggressive intervention is warranted.^{10,45} It is important to note that silver ions—not silver atoms—produce the antimicrobial effect. Thus, silver dressings require moist wound environments to release their active antimicrobial agent.^{46,47} Newer silver dressings aim to decrease the cytotoxicity seen in older silver dressings. PMD with silver is one such example. A marked improvement in wound healing and patient comfort were observed in a patient with PV after application of silver-containing hydrofiber dressings (SHD) such as Aquacel-Ag[®].⁴⁸ Acticoat[®] is another silver biologic dressing containing a 15 nm bactericidal coat of nano-crystallized ions of silver in a cluster structure.⁴⁹ In one study, Acticoat[®] shown complete healing in 13 cases of pemphigus wounds.⁴⁹

Nutrition is a crucial aspect of care, due to the loss of protein and other essential components in the serous sanguineous drainage. Patients should avoid excessive skin manipulation and minimize activities that may traumatize the skin and mucous membranes during active phases of the disease.¹⁰ These include contact sports and eating or

drinking food that may irritate the oral cavity (spicy, acidic, hard and crunchy foods).

Summary

Pemphigus presents with superficial wounds (erosions) in the skin and oral mucosa. Systemic corticosteroids are typically used for treatment initiation, however, due to the need for prolonged treatment and high morbidity of long corticosteroid courses, the early introduction of corticosteroid-sparing immunosuppressive agents is a key principle of treatment. Measures aimed at managing cutaneous wounds are additional important aspects of the management of pemphigus. There is more to wound care than just dressing. Wound care has to do with creating an optimal wound microenvironment and addressing the needs of the patient. Dressing selection is best guided by both the wound characteristics and dressing special features. The ideal dressing material should provide a moist environment to the wound while shielding it from bacterial invasion. In patients with pemphigus, wounds are superficial, and dressings such as films, hydrogels, and PMDs would be appropriate choices.

Disclosure

The authors report no conflicts of interest in this work.

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