Report

# Pyoderma gangrenosum: clinicopathologic correlation and proposed diagnostic criteria

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**Abstract**

Pyoderma gangrenosum is a rare but significant cause of ulcerations. It is a diagnosis of exclusion. Herein, we suggest diagnostic criteria and some historical perspectives on the diagnosis of pyoderma gangrenosum.

## Introduction

The rapid progression of a painful, necrolytic ulcer with an irregular, undermined border typifies ulcerative pyoderma gangrenosum (PG). PG is thought to be a reactive inflammatory dermatosis and part of the spectrum of neutrophilic dermatoses. Because no diagnostic criteria have been established for clinical use, PG is a diagnosis of exclusion.[1]

Despite the lack of diagnostic criteria, the distinctive clinical features of PG, which are outlined below, are apparent enough to permit the diagnosis of most cases. It would greatly simplify diagnosis and management of PG if a diagnostic laboratory test were available to confirm the condition, but such a test does not currently exist.

Herein, we describe our perspective on the essential components of PG and propose criteria to guide diagnosis.

## Historical perspectives from the Mayo Clinic

The condition of PG was initially described at the Department of Dermatology, Mayo Clinic.[2] Since that original publication describing this syndrome, clues to establishing a diagnosis of PG have been proposed by and presented from our department.[3–39] Initially, one of us (W.P.D.S.) proposed three major and seven minor criteria for the diagnosis of PG. Since then, we have modified and refined these criteria, and now we present them for formal use in the clinical setting.

The essential elements of the clinical diagnosis of PG were present in the original description of the entity by Brunsting, Goeckerman and O'Leary in 1930.[2] They considered PG to be an infectious dissemination from a distant focus of infection, for example, from the bowel in ulcerative colitis or the lungs in empyema. Over time, the clinicopathologic variability and other clinical properties of PG began to be recognized.

The first large case series (19 patients) with PG was reported by one of us (H.O.P.) in 1957.[4] In time, PG was recognized as a dermatologic emergency. Patients could be rescued from aggressive debridement and even amputation by making an accurate diagnosis of PG and by providing appropriate treatment.

The 1950s saw a revolution in the treatment of this disorder after the discovery of cortisone, for which Drs Kendall and Hench of the Mayo Clinic won the Nobel Prize in 1950. Cortisone was used initially to alleviate the symptoms of rheumatoid arthritis. It was also found to be effective in managing PG. The conventional course of corticosteroid treatment was similar to that with antibiotics (i.e. prescribed for 1–2 weeks at a time). However, PG would rebound after discontinuation of the corticosteroid. Dermatologists managing PG were among the first clinicians to use corticosteroids in larger doses for longer periods. Doses as high as 160 mg daily were used for initial treatment. Over the decades, various other immunosuppressive medications have been reported to be helpful in the management of PG.



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**Table 1** Proposed diagnostic criteria of classic, ulcerative pyoderma gangrenosum (PG). Diagnosis requires both major criteria and at least two minor criteria

| |
|---|
| **Major criteria** |
| 1. Rapid[a] progression of a painful,[b] necrolytic cutaneous ulcer[c] with an irregular, violaceous, and undermined border |
| 2. Other causes of cutaneous ulceration have been excluded[d] |
| **Minor criteria** |
| 1. History suggestive of pathergy[e] or clinical finding of cribriform scarring |
| 2. Systemic diseases associated with PG[f] |
| 3. Histopathologic findings (sterile dermal neutrophilia, ± mixed inflammation, ± lymphocytic vasculitis) |
| 4. Treatment response (rapid response to systemic steroid treatment)[g] |

[a]Characteristic margin expansion of 1 to 2 cm per day,[2] or a 50% increase in ulcer size within 1 month.
[b]Pain is usually out of proportion to the size of the ulceration.
[c]Typically preceded by a papule, pustule, or bulla.
[d]Usually necessitates skin biopsy and other investigations to rule out causes (outlined in Table 2) and for work-up (outlined in Table 3).
[e]Ulcer development at sites of minor cutaneous trauma.
[f]Inflammatory bowel disease, arthritis, IgA gammopathy, or underlying malignancy.
[g]Generally responds to a dosage of 1 mg/kg to 2 mg/kg per day, with a 50% decrease in size within 1 month.

## Need for diagnostic criteria

PG has a characteristic clinical appearance. However, other ulcerating conditions may have a similar appearance. Thus, there is room for diagnostic error. Indeed, of 157 patients evaluated for a diagnosis of PG at our institution, 15 (approximately 10%) were found not to have PG.[1]

The diagnostic criteria that we propose comprise what we consider the essential elements for the diagnosis of PG (Table 1). No criterion can be used in isolation because each can be seen in many disease states, but, when used together, they support a diagnosis of PG. Overvaluation of either clinical presentation or histopathologic findings may lead to the wrong diagnosis.

For his study of 44 cases of PG, von den Driesch[79] independently used inclusion criteria for the diagnosis of his PG patients that are almost identical to those we propose for clinical use.

When difficulty is encountered in either the diagnosis or the therapy of a patient suspected of having PG, we suggest referral to a physician experienced with the disease.

## Neutrophilic dermatoses

PG is part of the spectrum of the neutrophilic dermatoses, which are reactive processes that have in common noninfectious dermal neutrophilia. Other features the neutrophilic dermatoses share include an associated disorder (e.g. inflammatory bowel disease, paraproteinemia, or arthritis), a tendency for pathergy, and similarities in treatment (e.g. prednisone and dapsone). In addition to PG, the neutrophilic dermatoses include acute febrile neutrophilic dermatosis (Sweet syndrome), bowel-associated dermatosis–arthritis syndrome, neutrophilic eccrine hidradenitis, subcorneal pustular dermatosis (Sneddon-Wilkinson disease), and rheumatoid neutrophilic dermatitis, as well as some disorders considered primarily vasculitic (e.g. Behçet disease, pustular vasculitis, and erythema elevatum diutinum).[80]

## Clinical manifestations

The clinical manifestations of PG have been well described.[4,5,7,10,26,81,82] The classic ulcerative form begins as a nodule or sterile pustule that progresses to a necrotic and mucopurulent ulcer, with an edematous, violaceous, serpiginously expanding, undermined border (Fig. 1a). The ulcer is exquisitely painful. Pustular,[83] bullous,[8] and vegetative or superficial granulomatous[21] variants have been described and are outlined later in this article. The clinical manifestations of PG are characteristic if clinical changes are correlated with the age of the lesion. Acute lesions of PG progress rapidly. A cutaneous nodule develops into a furunculated lesion with central necrosis. The lesion enlarges rapidly, with an acneiform and serpiginous necrotizing red-blue border, again with marked pain.

The term "necrolytic" well describes these ulcers. PG is a dynamic process. It rapidly destroys skin tissue, and the liquefactive necrosis gives rise to the red-blue, undermined border.

Close inspection of the lesions reveals a 1- to 2-cm halo of erythema, depending on the stage of the disease, that surrounds the lesion. The halo has been interpreted as an area of the skin already involved but not yet showing necrosis. The lesions then progress to central necrosis and ulceration, with a red-violet, necrolytic, arcuate, or serpiginous undermined border, all surrounded by a halo of edema and erythema.

When acute lesions are present, necrosis of the skin characteristically develops quickly. Pain is usually a prominent feature and offers a means of following the progress of the disease and its response to therapy.

Pain is present in early lesions or as lesions spread, and it is out of proportion to the size of the lesion. When adequate doses of systemic corticosteroids are administered, pain may subside in 48–72 h.

Older and subsiding lesions of PG heal with a thin cribriform scar. The presence of a cribriform scar is of minor importance in clinical diagnosis. No dermatologic disease can be diagnosed by scars alone, but this finding can support the diagnosis of PG.

About 25% of patients with PG give a history of skin trauma, or pathergy (Fig. 1b), in the area of lesion development,

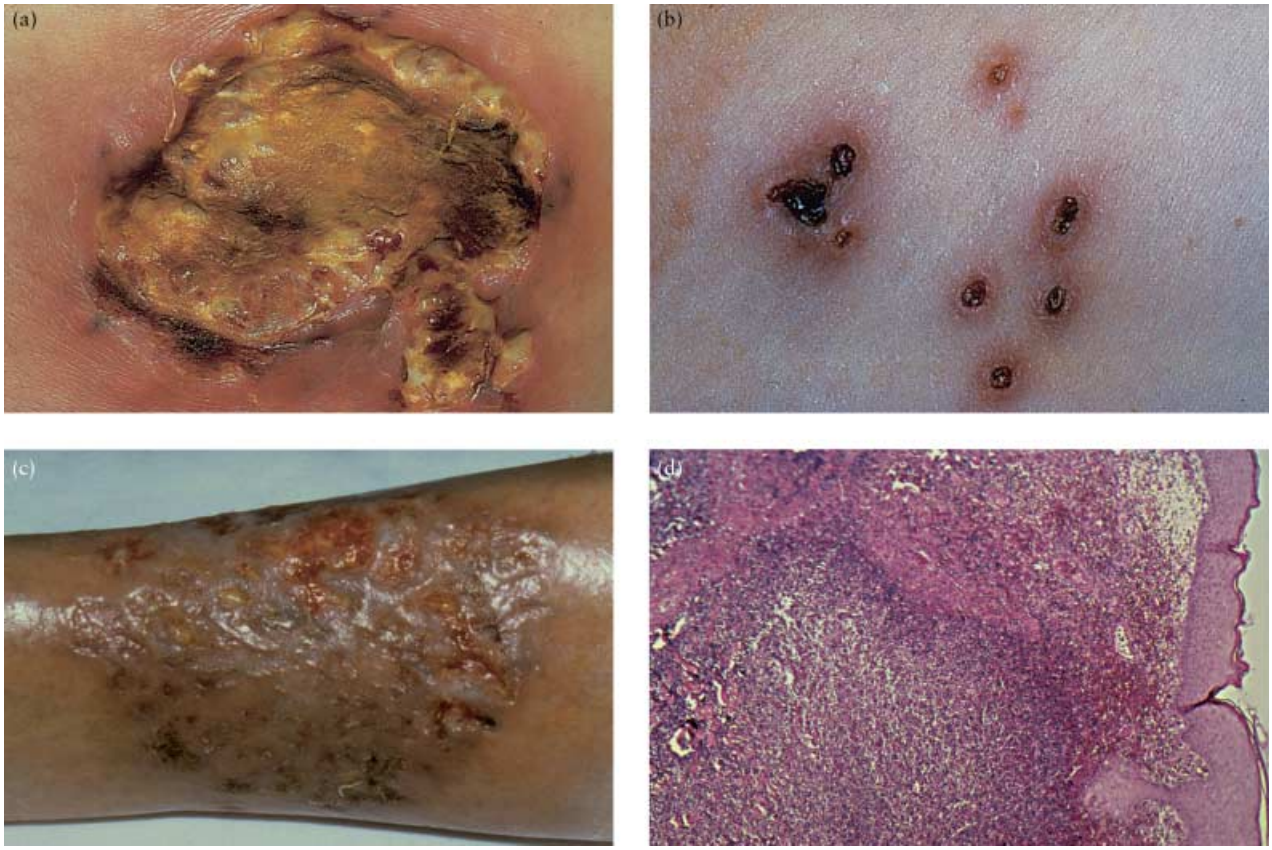


**Figure 1** Ulcerative PG. (a) An irregular, violaceous, and undermined border surrounds the necrolytic cutaneous ulcer. (b) Ulcers occurring at areas of injections (pathergy). (c) Healing ulcerative PG demonstrating cribriform scarring. (d) Incisional biopsy specimen of inflamed border showing intense dermal edema and neutrophilia

but most lesions develop de novo. The ulcers characteristically heal with cribriform scarring (Fig. 1c).

PG most frequently affects the lower extremities but can involve virtually any cutaneous site (e.g. the scalp, the face, periocular and perioral areas, the arms, the hands, the trunk, and genital, perianal, peristomal, and mucocutaneous areas). Involvement of the perineum is extraordinary but more common in children.[84–86]

## Associated diseases

In as many as 70% of cases, PG may be associated with a variety of diseases, including inflammatory bowel disease,[2,87] seropositive or seronegative arthritis,[88–90] multiple myeloma,[91] paraproteinemia (particularly IgA),[12,92–94] diverticulitis,[95] pulmonary disease,[96–98] malignancies (particularly leukemia),[8,15,22,99–104] and other conditions.[14,81,105–113] Yet, diagnosis of PG should not rely on this criterion alone because these diseases may be associated with ulcers due to other conditions.[1]

## Pathogenesis

The pathogenesis of PG is poorly understood, but neutrophil dysfunction (i.e. defects in chemotaxis or hyperreactivity) has been suggested. Evidence of abnormal neutrophil trafficking and metabolic oscillations was described recently in a patient.[114] Furthermore, interleukin-8 (IL-8), a potent leukocyte chemotactic agent, has been shown to be overexpressed in PG ulcers and to induce similar ulceration in human skin xenografts transfected with recombinant human IL-8.[115] The factors inciting or maintaining these abnormalities are unclear but likely are multifactorial (i.e. genetic predisposition, undefined infectious agents, or paraneoplastic or paraimmune phenomena).

Rare familial forms of PG have been reported.[85,116] The recently described "PAPA syndrome" (pyogenic sterile arthritis, PG, and acne) is an autosomal dominant disorder that maps to chromosome 15q.[27,29] The *IL-16* gene maps to 15q25 and is perhaps overexpressed in this disorder, because the IL-16 protein is chemotactic to neutrophils.

**Table 2** Differential diagnosis of pyoderma gangrenosum[a]

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|---|
| **Vascular occlusion or stasis** |
| Antiphospholipid–antibody syndrome[40–46] |
| Livedoid vasculopathy[b] |
| Venous stasis ulceration[b] |
| Klippel–Trénaunay–Weber syndrome[47] |
| Small-vessel-occlusive arterial disease[b] |
| Type-1 cryoglobulinemia[48] |
| **Vasculitis** |
| Wegener granulomatosis[49–52] |
| Polyarteritis nodosa[b] |
| Cryoglobulinemic vasculitis (mixed cryoglobulinemia)[53] |
| Takayasu arteritis[54,55] |
| Leukocytoclastic vasculitis[b] |
| **Cutaneous involvement by malignant process** |
| Angiocentric T-cell lymphoma[56] |
| Anaplastic large-cell T-cell lymphoma[57] |
| Mycosis fungoides bullosa[58] |
| Unspecified lymphoma[59] |
| Leukemia cutis[60,61] |
| Histiocytosis X (Langerhans cell histiocytosis)[62] |
| **Primary cutaneous infection** |
| Sporotrichosis[63–66] |
| Aspergillosis[24] |
| Cryptococcosis[9] |
| Herpes simplex type 2 virus[67,68] |
| Cutaneous tuberculosis[69] |
| Amebiasis cutis[70] |
| Zygomycosis[71] |
| Penicillium marneffei[72] |
| **Drug-induced and exogenous tissue injury** |
| Munchausen syndrome and factitious disorder[73,74] |
| Hydrea-induced ulceration[b] |
| Bromoderma[75] |
| Contact vulvitis[b] |
| Drug-induced lupus[76] |
| Loxoscelism (Brown recluse spider bite)[77,78] |
| Injection drug abuse with secondary infection[b] |
| **Other inflammatory disorders** |
| Cutaneous Crohn disease[b] |
| Ulcerative necrobiosis lipoidica[b] |

[a]Diseases initially diagnosed or treated as pyoderma gangrenosum.
[b]Weenig *et al*.[1]

**Table 3** Approach to the patient with suspected ulcerative pyoderma gangrenosum

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|---|
| **Important historical data** |
| Markedly painful ulcer |
| Rapid progression of ulceration |
| Type of skin lesion (papule, pustule, or vesicle) preceding the ulcer |
| Minor trauma (pathergy) preceding the ulcer |
| Symptoms of an associated disease (e.g. inflammatory bowel disease or arthritis) |
| Drug history (e.g. bromides, iodide, hydroxyurea, or granulocyte-macrophage colony-stimulating factor) |
| **Identify characteristic features of ulcer on physical examination** |
| Tenderness |
| Necrosis |
| Irregular violaceous border |
| Undermined, rolled edges |
| **Skin biopsy** |
| **Aim:** to rule out diagnoses that mimic pyoderma gangrenosum |
| **Protocol** |
| Elliptical incisional biopsy preferable to punch biopsy; include inflamed border and ulcer edge at a depth that includes subcutaneous fat |
| Specimen from inflamed border: routine histology (hematoxylin and eosin staining) and special staining (e.g. Gram, methenamine silver, or Fite) to detect microorganisms |
| Specimen from ulcer edge: culture in appropriate culture medium (to detect bacteria, fungi, and atypical mycobacteria) |
| **Laboratory investigations** |
| **Aims:** to identify associated diseases and to rule out diagnoses that mimic pyoderma gangrenosum |
| Complete blood count |
| Erythrocyte sedimentation rate |
| Blood chemistry (liver and kidney function tests) |
| Protein electrophoresis |
| Chest radiography |
| Colonoscopy |
| Coagulation panel (including antiphospholipid antibody screen) |
| Antineutrophil cytoplasmic antibodies (ANCA) |
| Cryoglobulins |
| Venous and arterial function studies |
| **Close, continuous follow-up** |
| Monitor response and side-effects of therapy |
| If patients have no response to treatment, reconsider diagnosis and repeat biopsy |

From Weenig *et al*.,[1] by permission of the Massachusetts Medical Society.

## Laboratory findings

No laboratory finding is diagnostic of PG, but patients often demonstrate a neutrophil leukocytosis and an elevated erythrocyte sedimentation rate. Laboratory investigations should be tailored to consider potentially associated diseases (e.g. inflammatory bowel disease, arthritis, or gammopathy) and to exclude diseases that may mimic PG (Tables 2 and 3).

## Histopathology

The histopathologic findings of PG are not specific, and the primary objective in obtaining a biopsy specimen is to rule out other causes of ulceration (e.g. infection, vasculitis, and malignancy).

Skin biopsy specimens taken from the necrotic, undermined ulcer border of PG reveal mixed cellular inflammation with neutrophil predominance. There also may be disruption or necrosis of dermal or pannicular blood vessels (with lymphocytes as the primary inflammatory cells) in skin biopsy specimens of erythematous regions adjacent to ulcers (Fig. 1d).

Studies of direct immunofluorescence,[11] histopathologic findings,[17] and additional clinicopathologic findings[16] have been reported from our institution. The direct immunofluorescence of PG demonstrated vascular deposition of IgM, C3, and fibrin in 36 of 65 (55%) biopsy specimens. IgM, C3, and fibrin were found in the papillary and reticular dermal vessels. IgG and IgA were present only occasionally.[17] These findings are neither sensitive nor specific for PG and usually immunofluorescence studies are conducted to exclude immunobullous disease, lupus, or vasculitis as potential causes of cutaneous ulceration.

**Table 4** Treatments reported effective in the management of pyoderma gangrenosum (PG)[a]

**Corticosteroids**
Systemic
Intralesional[117]
Topical[118]

**Antimicrobial agents**
Benzoyl peroxide[119]
Clofazimine[120]
Diaminodiphenylsulfone (dapsone)[121]
Rifampicin[122]
Lymecycline[119]
Tetracycline[21]
Minocycline[123]
Mezlocillin[124]
Potassium iodide[125]
Sulfapyridine[34]
Vancomycin[124]

**Steroid-sparing immunosuppressive agents**
5-aminosalicylic acid (topical)[126]
6-mercaptopurine[127]
Azathioprine[128]
Chlorambucil[129]
Cyclophosphamide[130]
Cyclosporine (systemic[131], topical[132])
Methotrexate[133]
Mycophenolate mofetil[134]
Nitrogen mustard (topical)[135]
Tacrolimus (systemic[136], topical[137])
Melphalan[138]

**Immune modulation**
Infliximab[139]
Interferon-$\alpha$[140]
Intravenous $\gamma$-globulin[141]
Plasmapheresis[142]
Thalidomide[143]

**Miscellaneous**
Colchicine[144]
Nicotine (topical)[145]
Sodium cromoglycate (topical)[146]

[a]Earliest reference of each treatment cited, as per Medline search from 1966 to December 2001.

## Differential diagnosis

Other causes of cutaneous ulceration may simulate or mimic PG clinically (Table 2).

## Treatment

In addition to corticosteroids and some miscellaneous medications, efficacious treatments for PG include antimicrobial agents, steroid-sparing immunosuppressive agents, and immunomodulating agents (Table 4). A comprehensive review of treatment approaches is beyond the scope of this article, but various approaches have been reported from our institution.[18,20,147]

Because PG typically responds to moderate doses of systemic corticosteroids, dramatic improvement after initiation of corticosteroid therapy lends support to the diagnosis. Thus, we include such a response as a minor diagnostic criterion. However, care must be taken to avoid overemphasizing the corticosteroid response, because other diseases simulating PG may be improved (e.g. antiphospholipid syndrome, vasculitis, and lymphoma) or exacerbated (e.g. infection and lymphoma) by treatment directed at PG. Indeed, 36% of patients with epidermal ulcers misdiagnosed as PG experienced improvement in or worsening of the underlying disorder while receiving treatment for PG.[1]

Reduced pain and decreased erythema are markers of response to treatment. Classically, within 24–72 h after initiation of treatment, the pain of PG is rapidly alleviated. The ulcer may show signs of clinical improvement (halt of enlargement, less induration, or less erythema), but the most remarkable feature is disappearance of pain.

## Variants of pyoderma gangrenosum

The described variants of ulcerative PG have been summarized in review articles.[26,82] Our proposed diagnostic criteria for the PG variants are noted in Table 5.

### Bullous pyoderma gangrenosum

Rapidly evolving painful vesicles and enlarging bullae characterize bullous PG,[8] and grouped bullae coalesce. The bullae enlarge in waves, with central necrosis and erosion and a surrounding halo of erythema. This central necrosis results in a shallow erosion rather than a necrotic ulcer. The bullous form of PG is most probably due to the more rapid superficial

 

**Table 5** Diagnostic criteria for variants of pyoderma gangrenosum (PG)[a]

| | Major criteria | Minor criteria |
|---|---|---|
| **Bullous PG** | Painful, inflammatory bullae; rapidly enlarging painful vesicles and bullae; coalescing of grouped bullae<br>Exclude other causes of bullae | Histopathology compatible (neutrophilic dermal infiltrate, subepidermal bullae ± epidermal necrosis)<br>Associated hematologic malignancy in as many as 70%<br>Pathergy<br>Rapid response to steroids |
| **Pustular PG** | Painful pustules (0.5–2 cm diameter), with surrounding halo<br>Exclude other causes of pustules (e.g. infections, drug eruptions, or psoriasis) | Histopathology compatible (neutrophilic infiltrate; subcorneal/subepidermal neutrophils)<br>Associated inflammatory bowel disease<br>Improvement with control of inflammatory bowel disease |
| **Vegetative PG** | Chronic erythematous plaques, with sinus formation; shallow ulceration or erosions and discomfort<br>Exclude other causes (e.g. pyoderma vegetans, blastomycosis-like pyoderma, infection, or pemphigus vegetans) | Histopathology compatible (dermal and histiocytic dermal infiltrate, granuloma formation)<br>No associated disease<br>Response to minor treatment measures |

[a]Diagnostic requirement: both major criteria and at least two minor criteria.

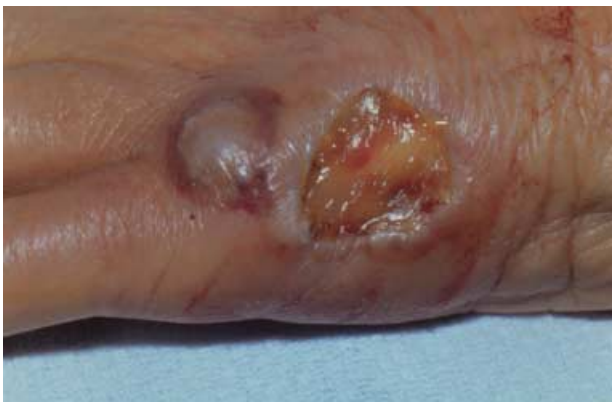

**Figure 2** Bullous PG. Bulla and adjacent ulceration developed at the site of previous bulla

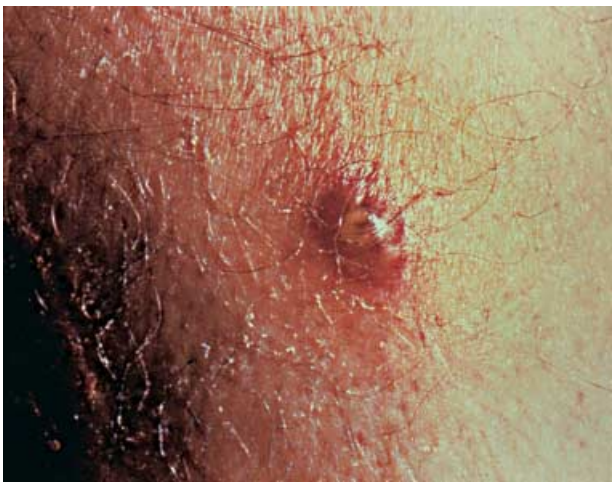

**Figure 3** Pustular PG. An intact pustule on normal skin is surrounded by intense erythema

necrosis that occurs in this form of the disease. The blistering seems to develop in concentrated rings at the periphery of the lesion. The superficial nature of the necrosis induces a gray color in surrounding tissue (Fig. 2). The necrolytic tissue produces the characteristic blistering common in this variant of PG. Histologic study typically shows dermal neutrophilia and subepidermal bullae formation. Bullous PG is associated with hematologic malignancy in as many as 70% of cases. Cribriform scarring is not associated. The appearance of bullous PG in a patient with leukemia or polycythemia rubra vera is often an ominous sign; such patients may develop progressive unresponsive hematologic malignancy and die within a short period. However, the disease may respond rapidly to moderate doses of systemic corticosteroids or dapsone.

### Pustular pyoderma gangrenosum

Although many lesions of ulcerative PG start as pustules, the pustules may not evolve to ulceration. This "pustular PG"[83] (Fig. 3) is associated with inflammatory bowel disease and may be a marker of bowel disease activity. Pustular eruption often improves with treatment of the underlying inflammatory bowel disease.

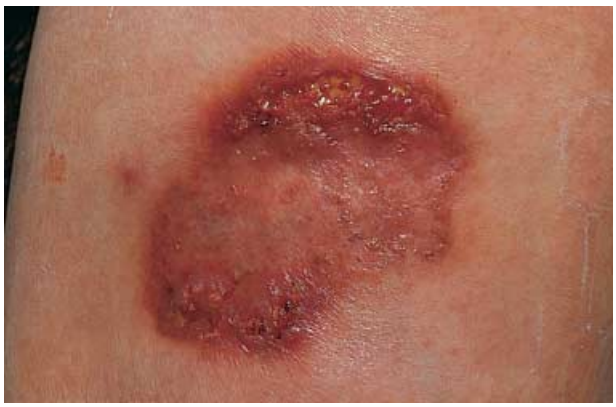

**Figure 4** Vegetative (superficial granulomatous) PG. A superficial boggy edematous plaque contains sinus tracts

#### Vegetative pyoderma gangrenosum

The localized, limited form of chronic superficial PG known as "vegetative PG" or "superficial granulomatous pyoderma"[21] has verrucous and ulcerative lesions (Fig. 4) and a granulomatous histologic appearance that represents a unique pattern in some patients. This variant usually progresses slowly and does not have an undermined border. Histologically, vegetative PG shows granuloma formation with sinus tracts. In general, patients with vegetative PG, which does not usually require systemic steroid therapy, do not have systemic disease.

## Conclusions

Many conditions produce severe cutaneous ulceration that simulates PG. Some of these alternative diseases may be improved or aggravated by PG-directed treatment, potentially delaying diagnosis or increasing morbidity due to the underlying condition. Therefore, PG imitators must be excluded (major criterion 2) before a diagnosis can be established.

PG may be associated with several systemic disorders. In all cases, a careful physical examination, appropriate laboratory investigations, and treatment of any underlying disease are indicated. Finding diseases that are associated classically with PG lends support to the diagnosis, but, because this is a nonspecific finding, it is included as a minor criterion.

Histopathologic examination of the erythematous or grayish border adjacent to the ulceration is not diagnostic, but it may support a diagnosis of PG. Special stains for microorganisms and tissue cultures should be performed on biopsy specimens. We include the histopathologic findings of PG as a minor criterion for diagnosis.

For localized cases of PG, dapsone or sulfapyridine may suffice as monotherapy. Systemic treatment with corticosteroids is usually necessary for patients with extensive involvement. Other agents, such as cyclosporine, azathioprine, tacrolimus, or one of the antimetabolites, should be used as steroid-sparing agents. Rapid response to treatment lends support to the diagnosis, but other ulcerative skin diseases also respond to these treatments. Therefore, rapid response to treatment is included as a minor criterion for diagnosis.

In summary, PG should be regarded as a syndrome that presents with a spectrum of clinical and histopathologic features. Diagnostic criteria are needed to differentiate it from other dermatoses that also produce severe cutaneous ulceration. We propose both major and minor criteria for the diagnosis of PG. To be considered a definite case of PG, the condition should meet both major criteria and at least two minor criteria.

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