

Clinical characteristics, associated comorbidities, and treatment approaches in pyoderma gangrenosum: a single-center retrospective analysis

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Abstract

Introduction: Pyoderma gangrenosum (PG) is a rare destructive neutrophilic dermatosis of unknown etiology, associated with systemic diseases in approximately 50% to 75% of cases.

Methods: We conducted a search of the hospital database to retrieve medical records of patients diagnosed with PG at our facility between 1995 and 2019. The diagnosis was validated through clinical characteristics, histopathological examination, and necessary tests to rule out other dermatoses. Data on demographics, disease presentation, comorbidities, and treatment strategies were collected and evaluated.

Results: The analysis included 44 patients, 27 (61.4%) females and 17 (38.6%) males. The median age at presentation was 46.5 years (range 15–73). The most common location was the lower leg, in 32 (72.7%) patients. The ulcerative variant was found in 37 (84.1%) patients. In 11 patients (25%) an association with inflammatory bowel disease (IBD) was found. Hematological disorders occurred in five (11.4%) patients and rheumatoid arthritis in four (9.1%). Treatment was started with systemic corticosteroids (CS) in 36 (83.7%) patients, and pulse corticosteroid therapy was administered in five (11.4%) patients. The most frequently used steroid-sparing agent was dapsone, in 18 (40.9%) patients.

Conclusions: PG often presents with associated systemic conditions, but it may also appear idiopathically. CS remain the mainstay of treatment, complemented by immunosuppressants and biologics such as infliximab for IBD-associated cases.

Keywords: associated comorbidities, biologic therapy, inflammatory bowel disease, neutrophilic dermatosis, pyoderma gangrenosum

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Introduction

Pyoderma gangrenosum (PG) is a rare chronic inflammatory condition, not caused by infection, that falls within the spectrum of neutrophilic dermatoses. It typically begins with sterile pustules that rapidly progress into painful necrotic ulcers surrounded by erythematous tissue (1, 2). The age of onset can vary between the 20s and 50s, and it is rare in childhood and more frequent in females. The etiology of PG is unknown, and genetic predisposition, neutrophilic dysfunction, and aberrant cellular immunity play a role in pathogenesis (3). PG may occur alone or in syndromic forms, or it may be associated with systemic diseases. Approximately 50% to 75% of PG cases are associated with systemic diseases, including inflammatory bowel disease (IBD), rheumatological conditions, blood disorders, and cancers (4–8).

Four clinical types of PG were suggested by Powell et al.: ulcerative, pustular, bullous, and vegetative (9). Although lesions most frequently appear on the lower extremities, they can also affect other areas, including the hands, trunk, breasts, head and neck, and peristomal skin.

Extracutaneous manifestations have been reported in the pulmonary (including severe respiratory distress syndromes), ocular (keratitis, scleritis, or ulcers of the eyelids), renal, cardiac, and gastrointestinal systems, and as neutrophilic infiltration of the spleen and bone (10–13). The treatment of PG is empirical due to the small number of randomized controlled trials (14). Current treatment options include high-dose prednisone, cyclosporine A

(CsA), azathioprine (AZA), cyclophosphamide (CYC), mycophenolate mofetil (MMF), intravenous immunoglobulin (IVIG), infliximab (IFX), adalimumab (ADA) and other biologics (15–22).

This article provides a retrospective review of a series of PG cases from a single institution, focusing on clinical features, related conditions, treatment approaches, and follow-up outcomes.

Methods

We accessed the hospital database to review the records of 44 patients that were diagnosed with PG and treated at our facility from 1995 to 2019. Demographic data, clinical characteristics (localization, clinical variants, and number of lesions), associated systemic diseases, and treatment modalities were analyzed. The initial workup included erythrocyte sedimentation rate; C-reactive protein; blood cell count; liver and renal functional testing; quantitative serum immunoglobulin (Ig)A, IgM, and IgG levels; serum electrophoresis; urinalysis; cytoplasmic and perinuclear anti-neutrophil cytoplasmic antibodies, antinuclear antibodies; antiphospholipid antibodies; extractable nuclear antigen screening; cryoglobulins; and immune complexes. Enzyme-linked immunosorbent assay (ELISA) for HIV 1/2, as well as hepatitis B and C, and syphilis serologies were performed. Additional procedures included colonoscopy in patients with suspicion of IBD, bone marrow biopsy in patients with signs of hematological disorders, and color Doppler ultrasonography to exclude vascular etiology in cases with ulcerative lesions. Diagnoses were based

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on clinical presentations, histopathological evidence confirming PG, and tests to differentiate it from other similar skin conditions. Data analysis was performed using descriptive statistics.

Results

Forty-four patients with PG were included in the study: 27 (61.4%) females and 17 (38.6%) males (male:female ratio = 1:1.6), age 15–73 (median = 46.5) years. The median ages of the female and male patients were 42 and 51 years (range: 22–73 and 15–69 years), respectively. The demographic and clinical characteristics of the patients are presented in Table 1.

The ulcerative variant was most frequently observed, in 37 (84.1%) patients. The bullous and pustular variants were identified in three (6.8%) patients each, and the vegetative form was observed in two (4.5%) patients. One patient exhibited both ulcerative and bullous forms. Lesions were predominantly localized on the lower limbs (32 patients, 72.7%) and trunk (15 patients, 34.1%), and only four (9.1%) patients showed lesions in the head and neck region. In one patient (2.3%), PG appeared at the site of a surgical procedure. Disseminated lesions on the trunk and extremities appeared in two (4.5%) patients.

No correlations were observed between the clinical variants of the disease, lesion localization, and gender. The ulcerative variant was most common on the lower extremities (29 patients, 78.4%) and less frequent on the trunk (nine patients, 24.3%) and upper extremities (six patients, 16.2%). Bullous and pustular lesions were mainly localized on the trunk and extremities, whereas the vegetative variant affected the extremities in two patients. The pustular variant never appeared on the lower extremities. The ulcerative form manifested most frequently, with three to five lesions in 24 (54.5%) patients, and more than six lesions in nine (20.5%) patients. Histopathologic examination of the skin lesions revealed nonspecific findings in the largest number of patients (20 patients, 45.5%); neutrophilic infiltration with abscess formation without vasculitis was present in 17 (38.6%) patients, and five (11.4%) patients exhibited signs of vasculitis.

Table 1 | Demographic data and clinical characteristics of pyoderma gangrenosum patients.

	Total	Male, n (%)	Female, n (%)	p-value
Sex	44	17 (38.6)	27 (61.4)	0.897
Median age (years)	46.5	51.0	42.0	
Localization				
Head and neck	4 (9.1)	3 (17.6)	1 (3.7)	0.282
Trunk	15 (34.1)	5 (29.4)	10 (37.0)	0.603
Upper extremities	10 (22.7)	5 (29.4)	5 (18.5)	0.473
Lower extremities	32 (72.7)	12 (70.6)	20 (74.1)	1.000
Generalized	2 (4.5)	1 (5.9)	1 (3.7)	1.000
Post-surgical	1 (2.3)	0 (0.0)	1 (3.7)	1.000
Variants				
Ulcerative	37 (84.1)	13 (76.5)	24 (88.9)	0.662
Bullous	3 (6.8)	1 (5.9)	2 (7.4)	1.000
Pustular	3 (6.8)	2 (11.8)	1 (3.7)	1.000
Vegetative	2 (4.5)	1 (5.9)	1 (3.7)	1.000
Lesions				0.904
1–2	11 (25.0)	5 (29.4)	6 (22.2)	
3–5	24 (54.5)	8 (47.1)	16 (59.3)	
6 or more	9 (20.5)	4 (23.5)	5 (18.5)	
Associated diseases				1.000
Present	24 (54.5)	9 (52.9)	15 (55.6)	
Absent	20 (45.5)	8 (47.1)	12 (52.9)	

Associated diseases

Among the 44 patients, 24 (54.5%) had an associated systemic disease, with 15 (62.5%) female and nine (37.5%) male.

A total of 20 (45.5%) patients exhibited the idiopathic form of the disease (Table 2). The most frequent associated disease was IBD in 11 (25%) patients, including ulcerative colitis (UC) in six (13.6%) patients and Crohn’s disease (CD) in five (11.4%) patients. The ulcerative variant was observed in nine (24.3%) patients, and the pustular form was noted in two patients with IBD. Hematological disorders—including monoclonal gammopathy of undetermined significance (MGUS), acute myeloid leukemia (AML) or chronic lymphocytic leukemia (CLL), and autoimmune hemolytic anemia (AIHA)—were found in 11.4% of the patients, and the disease manifested as the ulcerative variant in four patients and the bullous variant in one patient. Notably, one case of IgA MGUS was linked to a sterile neutrophilic spleen abscess, an extracutaneous manifestation of PG. A diagnosis of rheumatoid arthritis (RA) was confirmed in four patients (9.1%), and two had uric arthritis (UA),

Table 2 | Associated comorbidities in pyoderma gangrenosum patients.

Comorbidity	Total (n = 44)	Male (n = 17)	Female (n = 27)
Inflammatory bowel disease			
Ulcerative colitis, n (%)	6 (13.6)	2 (11.8)	4 (14.8)
Crohn’s disease, n (%)	5 (11.4)	2 (11.8)	3 (11.1)
Hematological			
Monoclonal gammopathy, n (%)	2 (4.5)	1 (5.9)	1 (3.7)
Acute myeloid leukemia, n (%)	1 (2.3)	1 (5.9)	0 (0.0)
Chronic lymphocytic leukemia, n (%)	1 (2.3)	1 (5.9)	0 (0.0)
Autoimmune hemolytic anemia, n (%)	1 (2.3)	0 (0.0)	1 (3.7)
Rheumatological			
Rheumatoid arthritis, n (%)	4 (9.1)	1 (5.9)	3 (11.1)
Uric arthritis, n (%)	2 (4.5)	1 (5.9)	1 (3.7)
Diabetes mellitus, n (%)	2 (4.5)	0 (0.0)	2 (7.4)
Miscellaneous			
Hidradenitis suppurativa, n (%)	1 (2.3)	1 (5.9)	0 (0.0)
Pilonidal sinus, n (%)	1 (2.3)	1 (5.9)	0 (0.0)
Tuberculosis, n (%)	1 (2.3)	0 (0.0)	1 (3.7)
Bladder tumor, n (%)	1 (2.3)	1 (5.9)	0 (0.0)
Autoimmune hepatitis, n (%)	1 (2.3)	1 (5.9)	0 (0.0)

both presenting with the ulcerative variant. One patient with RA and PG also exhibited hidradenitis suppurativa (HS) and pilonidal sinus (PS). Two (4.5%) patients had diabetes mellitus (DM), a solid tumor was found in one (2.3%) patient, and one patient each suffered from tuberculosis (TB) and autoimmune hepatitis (AIH). In patients \leq 65 years old, rheumatological disorders and IBD were more frequent, whereas hematological disorders were more frequent in older patients, but without significant correlations.

Treatment pattern

Data on treatment are summarized in Table 3. Therapy with CS and immunosuppressive or biological agents was most frequently initiated based on the clinical manifestations and associated diseases. The majority of the patients (36 or 83.7%) were treated with CS (1 mg/kg/d of prednisone). Pulse corticosteroid therapy was administered in five (11.4%), DDS in 18 (40.9%), CsA in eight (18.2%), and CYC and AZA in three (6.8%) patients. The patient with widespread PG associated with CD was refractory to the previously mentioned immunosuppressive agents and was treated with IFX with an unsatisfactory outcome.

Follow-up

The idiopathic form of the disease was not linked to any associated diseases during a follow-up of 13 patients spanning 1 to 27 years (median: 8.75 years; Table 4). For maintenance therapy, CS were administered in combination with DDS and AZA in seven and two patients, respectively. Eleven patients achieved complete remission (CR) of their lesions without relapses during long-term observation. The median duration of systemic treatment in patients with CR was 12 months (range: 6–36 months). Continuing

Table 3 | Treatment of pyoderma gangrenosum patients.

Therapy	n (%)
Systemic corticosteroids	36 (83.7)
Pulse corticosteroid therapy	5 (11.4)
Azathioprine	3 (6.8)
Cyclophosphamide	3 (6.8)
Cyclosporine A	8 (18.2)
Dapsone	18 (40.9)
Infliximab	1 (2.3)
Topical steroids	27 (61.4)

Table 4 | Follow-up of pyoderma gangrenosum patients.

Sex, age	Associated diseases	Initial treatment	Maintenance treatment	Treatment (months)	Follow-up (months)	Disease outcome
M, 56	UA	CS + DDS	CS + DDS	12	166	CR
F, 38	No	CS + CYC	CS + AZA	36	179	CR
F, 64	MG	CS + CsA	CS	13	160	CR
F, 61	No	CS + DDS	CS + CsA	7	15	CR
F, 34	UC	CS + CsA	CS	8	119	CR
F, 26	No	CS + CsA	CS pulse, CS + DDS	19	105	CR
F, 22	RA	CS + DDS	CS + DDS	12	89	CR
F, 47	UC	CS + AZA	CS + AZA	15	140	CR
M, 62	CD	CS + CsA	CS + DDS	6	95	CR
F, 68	UC	CS	CS	8	31	CR
F, 46	No	CS + DDS	CS + DDS	23	53	CR
F, 65	No	CS	CS + DDS	14	62	PR
F, 40	No	CS + CsA	CS + DDS	Intermittently 120, last 36 continuously	329	PR

M = male, F = female, AZA = azathioprine, CD = Crohn's disease, CR = complete response, CS = corticosteroids, CS pulse = pulse corticosteroid therapy, CsA = cyclosporine A, CYC = cyclophosphamide, DDS = dapsone, MG = monoclonal gammopathy, PR = partial response, RA = rheumatoid arthritis, UA = uric arthritis, UC = ulcerative colitis.

therapy was necessary to control the disease in two patients with partial remission (PR).

Discussion

PG is a challenging disease and is considered a prototype of neutrophilic dermatoses. It generally presents in adults between ages 20 and 50, with a greater frequency in females and a significant correlation with systemic diseases. Older patients are far more likely to have hematological malignant neoplasms and inflammatory arthritis, whereas younger patients exhibit IBD more commonly (6, 23). The age distribution and sex ratio observed in our cohort are consistent with previously published epidemiological data, further supporting the concept of PG as a disease of middle adulthood with a clear female preponderance. The ulcerative variant was predominantly observed, mainly affecting the distal third of the lower legs, consistent with observations from previous research (24–29). In one patient, coexistence of the ulcerative and bullous forms of disease was observed, and in another patient post-surgical PG appeared 5 days after surgery (30, 32).

Histopathological features are not pathognomonic but are useful in differential diagnosis; namely, to exclude vasculitis, malignancy, infection, vascular disorders, or other neutrophilic diseases that may mimic PG. The histopathologic changes are closely related to the type of PG, the timing of lesion onset, and the site of the biopsy. The occurrence of neutrophilic abscess formation and occasional leukocytoclastic vasculitis in our series aligns with observations by Pereira et al. (26) and additional studies in the field (33, 34) supporting the view that vascular involvement in PG is often secondary to intense neutrophilic inflammation.

Over half of patients with PG have a significant underlying condition, unlike 40% of patients that only have skin involvement (35). Current knowledge about diseases associated with PG is insufficient and is based on case series and a few retrospective cohort studies. The leading associated diseases are IBD, RA, hematological malignancies, and solid tumors (36). The onset of these diseases may precede, follow, or occur simultaneously with PG, and both conditions can run an independent course (37).

Gillard et al. (38) found that 53.2% of 126 patients suffered from an associated disease, including IBD (23.8%), hematological disorders (24.6%), and rheumatological disorders (7.1%). A higher

percentage of patients with associated diseases (66.3%) was reported in the largest retrospective study of 365 patients: IBD was found in 41% of these patients, and the corresponding numbers for arthritis, hematological disorders, and solid organ neoplasm were 20.5%, 5.9%, and 6.5%, respectively (6).

This study confirmed the findings in the literature. Associated diseases were found in 54.5% of patients, with 25.0%, 11.4%, 9.1%, and 2.3% suffering from IBD, hematological disorders, RA, and bladder cancer, respectively. Leukemia and MGUS are important associated diseases that were first noted by Sluis et al. (39). In our study, two (4.5%) patients had IgA MGUS without progression toward multiple myeloma during follow-up.

Extracutaneous manifestations of PG were observed in only one patient, with multiple sterile spleen abscesses identified via abdominal echography and computed tomography (13).

Coexistence of HS and PG as a chronic inflammatory dermatosis has been reported in both syndromic (PASH, PAPASH, and PASS: PG, acne, and HS; pyogenic arthritis, PG, acne, and HS; and PG, acne, HS, and seronegative spondyloarthritis, respectively) and non-syndromic contexts. The overall prevalence of PG among patients with HS was 0.18%, and it is more frequently linked to CD (40). One patient with a 3-year history of HS and PS in our study simultaneously developed RA and PG. Although this coexistence is rare, these diseases share features due to pro-inflammatory cytokine dysregulation (41). DM was found in only two patients (4.5%), diverging from prior studies that reported a prevalence of 15.0% to 28.0% (8). This difference can be explained by their older ages compared to those of the patients in our case series.

Hepatitis has been noted as a rare comorbidity, and our study confirmed only one patient with AIH in contrast to Binus et al. (1), who reported seven (6.8%) patients with underlying hepatitis C and two (1.9%) with AIH. The other associated diseases in our case series may represent coincidental associations.

The treatment of PG is often empirical because of the lack of randomized controlled trials. Systemic CS and CsA have been shown to be effective in a large number of cases and are therefore considered first-line therapies (42). These treatments achieved a 47% response rate within 6 months, showing comparable results in terms of rapid healing, recurrence rates, and side effects.

Periera et al. (26) described complete healing in 83.0% of patients treated with steroids and CsA. Evidence of therapeutic response based on randomized clinical studies exists for only three agents: prednisolone, CsA, and IFX (14, 43, 44). In patients diagnosed with IBD, administration of IFX is currently regarded as a first-line therapeutic option (39). Other immunosuppressive agents successfully used in individual case reports include DDS, AZA, MMF, CYC, thalidomide, and IVIG. Patients that respond insufficiently to conventional immunosuppressants may be considered for novel targeted therapies, including ustekinumab, an interleukin (IL)-12/23 inhibitor, as well as anakinra and canaki-

numab, both IL-1 inhibitors (43, 45, 46).

In our case series, CS and DDS were the most frequently used therapeutic modalities, reflecting their established role as first-line treatment options in PG. In patients with severe or rapidly progressive disease, high-dose intravenous CS were employed to achieve prompt disease control. In addition, several steroid-sparing agents, including CsA and AZA, were introduced in selected cases, primarily to reduce CS exposure or to maintain disease remission. Treatment outcomes were heterogeneous, ranging from complete and partial remission to insufficient response or refractory disease, which is consistent with the well-recognized variability in clinical course and therapeutic responsiveness of PG.

Three patients died due to multiple systems organ failure, and two had uncontrolled underlying diseases (CD and AML) and reported side effects of therapy, such as sepsis and iatrogenic DM. Unfortunately, no post-mortem examination was performed to investigate a possible relationship with PG. Long-term follow-up was incomplete because many patients were lost to follow-up over time, which is a limitation of this study. However, among patients available for evaluation at the final follow-up visit in 2019, CR was noted in 11 patients and PR in two patients. The patients with CR were on systemic treatment for a median duration of 1 year.

In a patient with associated RA, HS, and PS, complete remission of cutaneous lesions was achieved with CS and CsA, and no recurrence was noted at subsequent follow-up visits. A patient with IgA MGUS and spleen abscesses (13) was treated with CS and CYC. This patient did not develop multiple myeloma during an extended period of observation. Complete remission was achieved with CS (a decreasing dose) and DDS in the patient with post-surgical PG, and preventive CS treatment was started before the next surgical intervention, as recommended by previous reports (30, 32). In three recalcitrant PG patients, CYC was used in addition to pulse CS therapy, but without satisfactory results. The treatment was stopped, and the therapy was replaced by other drugs, leading to a satisfactory response in one patient and complete remission in another. IFX was administered to one patient with IBD and widespread ulcerative lesions without any improvement in either condition, and the eventual outcome was fatal.

Conclusions

PG is a systemic immune-mediated inflammatory disease, often associated with underlying diseases, suggesting that all patients with PG should be assessed for the presence of systemic disease regardless of age. PG remains a diagnosis of exclusion due to the absence of definitive diagnostic and therapeutic markers. Early recognition of the disease's clinical manifestations can facilitate prompt diagnosis and treatment. Systemic immunosuppressive therapies and various biologic agents appear to be promising options and should be considered as initial treatment strategies.

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