Systemic disease associations and management of pyoderma gangrenosum: a single-center retrospective case series

Ilyas El Kassimi[1*], Adil Rkiouak[1], Nawal Sahel[1], Meryem Zaizaa[1], Zineb El Bougrini[1], Bilal Talamoussa[1], Youssef Sekkach[1]

European Journal of Medical Case Reports
Volume 10(7):218–222
DOI: 10.24911/ejmcr.9-2648

ABSTRACT

Background: Pyoderma gangrenosum (PG) is a rare neutrophilic dermatosis frequently associated with systemic inflammatory diseases. We aimed to describe systemic disease associations, management strategies, and outcomes of PG managed in internal medicine.

Settings: Single-center retrospective case series conducted in an internal medicine department between January 2021 and December 2025.

Methods: We retrospectively reviewed all eligible patients treated for PG in our department during the study period. Diagnosis was based on clinicopathological assessment after exclusion of major mimickers; the Delphi consensus criteria were not formally applied because their individual components were not consistently documented in the retrospective records. Demographic, clinical, histological, treatment, and outcome data were extracted from medical records.

Results: Ten patients (five women; median age 40 years, range 19-58) were included. Ulcerative PG predominated (6/10), while four patients had pustular PG; lesions mainly involved the lower limbs, and pathergy was observed in five patients. An associated systemic disease was identified in eight patients, including Behçet disease, inflammatory bowel disease, systemic sclerosis, anti-neutrophil cytoplasmic antibody-associated vasculitis, systemic lupus erythematosus, and Takayasu arteritis. All patients received systemic corticosteroids; additional immunosuppressants or anti-tumor necrosis factor-alpha therapy were used as steroid-sparing treatment and/or to control active associated systemic disease. Six patients achieved complete healing without relapse, whereas four experienced at least one relapse during chart-documented follow-up.

Conclusion: In internal medicine practice, PG is commonly linked to systemic inflammatory diseases, supporting a structured, clinically guided systemic work-up. Systemic corticosteroids remain first-line therapy, while immunosuppressants and targeted biologics are valuable steroid-sparing options when disease is refractory or coexists with active systemic inflammation.

Keywords: Pyoderma gangrenosum, neutrophilic dermatoses, systemic inflammatory diseases, autoimmune diseases, corticosteroids, biologic therapy.

Type of Article: CASE REPORT **Specialty:** Internal Medicine

Correspondence to: Ilyas El Kassimi
*Department of Internal Medicine, Mohammed V Military Teaching Hospital, Mohammed V University, Rabat, Morocco.
Email: dr.elkassimiilyas@gmail.com
Full list of author information is available at the end of the article.

Received: 18 February 2026
Revised (1): 08 March 2026
Revised (2): 10 March 2026
Accepted: 06 April 2026

Background

Pyoderma gangrenosum (PG) is a rare neutrophilic dermatosis characterised by painful inflammatory skin lesions, most often ulcerations with violaceous undermined borders [1-3]. Although primarily cutaneous, PG frequently occurs in a systemic context, with an associated disease reported in up to half of cases in published series, including inflammatory bowel disease, inflammatory arthritis, and hematologic disorders [4,5]. The management of PG remains challenging because of its heterogeneous presentations, the absence of a pathognomonic diagnostic test, and the need to exclude mimickers such as infection, vasculitis, and malignancy [1,6,7]. Diagnostic frameworks such as the Delphi consensus criteria are useful for standardization, but their retrospective application may be limited when some items are not systematically documented [6]. Therapeutic evidence is mainly derived from case series and observational studies; systemic corticosteroids and ciclosporin are commonly used, and biologic therapies, particularly anti-tumor necrosis factor (TNF)-alpha agents, are increasingly reported in refractory disease [2,8,9]. Internal medicine plays a key role in the diagnosis and comprehensive assessment of PG because of its systemic associations. We therefore report a case series of PG

managed in an internal medicine department, focusing on clinical features, associated systemic diseases, treatment strategy, and outcomes.

Patients and Methods

Study design and setting: Retrospective descriptive observational study conducted in an internal medicine department. Patient identification and selection: Cases were identified through review of departmental inpatient and outpatient records between January 2021 and December 2025. Patients were included if they had a final diagnosis of PG and sufficient clinical, histological, treatment, and outcome data for analysis; records lacking essential data were excluded. Diagnostic approach: PG was diagnosed on clinico-pathological grounds based on a compatible painful ulcerative or pustular presentation, exclusion of major alternative diagnoses (infection, vasculitis, malignancy), and supportive histology when available. Skin biopsy was used primarily to rule out infectious, vasculitic, or malignant causes and, when compatible, to support the diagnosis of neutrophilic dermatosis. Because of the retrospective design and incomplete documentation of all individual items, Delphi consensus criteria were not formally applied. Data collection: Data were extracted from medical records using a standardized form including demographics; past medical and surgical history; PG characteristics (onset, subtype, number, size, localization, pain, pathergy); laboratory and histological findings; associated systemic diseases; treatments (local and systemic); and outcomes (healing, relapse, follow-up documentation). Laboratory and systemic assessment: All patients underwent a basic laboratory work-up, including blood count, inflammatory markers, renal and liver function tests, serum protein electrophoresis, and viral serologies. Beyond routine laboratory tests, the systemic work-up was clinically guided according to the presentation and included, when indicated, autoimmune testing [for example ANA, anti-dsDNA and anti-neutrophil cytoplasmic antibody (ANCA)], evaluation for hematologic disease, gastrointestinal assessment in patients with digestive symptoms, and symptom-directed imaging or specialist referral. Outcome definitions: Complete healing was defined as full re-epithelialization of all active PG lesions. Relapse was defined as the recurrence of clinically compatible PG after complete healing. Outcomes were assessed from chart-documented follow-up visits. Because follow-up duration was not uniformly recorded across all files, a reliable cohort-level median and range of follow-up could not be calculated. Statistical analysis: Data are presented descriptively as *n* (%) or median (range), as appropriate.

Results

Demographics: Ten patients were included (five women and five men), with a median age of 40 years (range 19–58). Clinical presentation: Disease onset was progressive in most cases. Lesions were constantly painful, typically presenting as painful ulcerations with a fibrino-necrotic base and raised violaceous borders (Figure 1). Ulcerative PG was the most frequent subtype (6/10), while four patients presented with pustular PG (Table 1). Lesions mainly involved the lower limbs, followed by the upper limbs and, less commonly, the trunk. Pathergy was observed in five patients, including lesions arising on surgical scars or after minor trauma (Figure 2). Laboratory and histology: Several patients had inflammatory anaemia and/or neutrophilic leukocytosis. Inflammatory markers were variably elevated (Table 2). Histology most often showed a neutrophil-predominant inflammatory infiltrate without specific evidence of vasculitis; biopsy was mainly useful to exclude differential diagnoses. Associated systemic diseases: An associated systemic disease was identified in eight patients (80%): Behçet disease (n = 2), inflammatory bowel disease (Crohn's disease, n = 1), systemic sclerosis (n = 2), systemic lupus erythematosus (n = 1), ANCA-associated vasculitis (n = 1), and Takayasu arteritis (n = 1) (Table 1). Treatment and outcomes: All patients received systemic corticosteroids. Escalation to conventional immunosuppressants (methotrexate, azathioprine, cyclophosphamide) and/or anti-TNF-alpha therapy

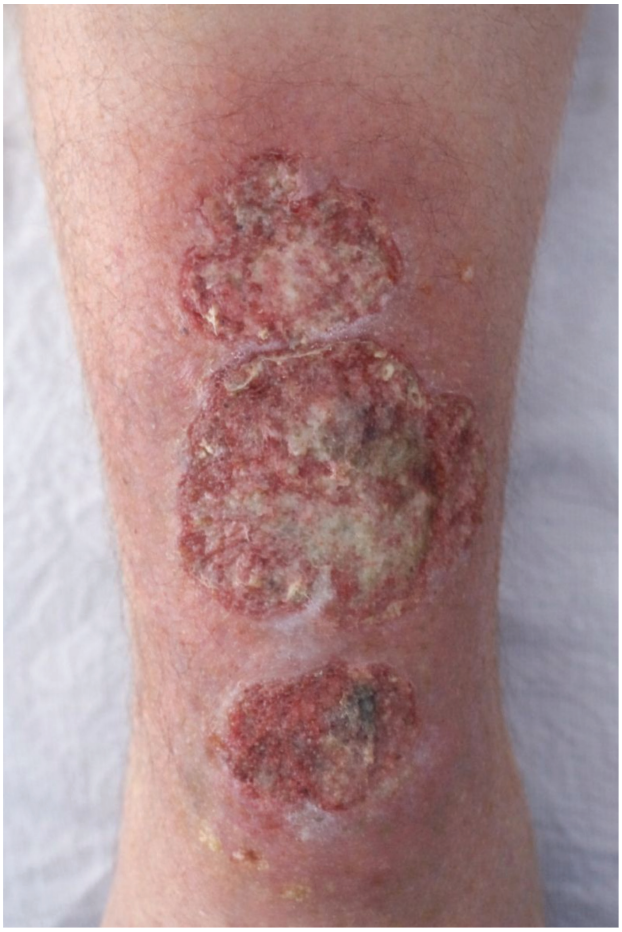

Figure 1. *Painful lower-limb ulcer with a fibrino-necrotic base and raised violaceous undermined borders, clinically typical of ulcerative PG.*

Table 1. *Baseline clinical characteristics of patients with PG (n = 10).*

PATIENT	AGE (YEARS)	SEX	PG SUBTYPE	COMORBIDITIES	NUMBER OF LESIONS	MAIN LOCALIZATION
1	37	Female	Ulcerative	Raynaud phenomenon; systemic sclerosis; hypertension	1	Anterior aspect of right leg
2	19	Male	Ulcerative	Systemic lupus erythematosus	3	Anterior aspect of left leg
3	45	Female	Ulcerative	Systemic sclerosis; Raynaud phenomenon	3	Lateral aspect of left leg
4	58	Male	Pustular	Current smoker	12	Dorsum of hand; left elbow; knees; abdomen
5	43	Male	Ulcerative	Angio-Behçet disease	1	Medial aspect of left leg
6	24	Female	Ulcerative	None reported	2	Medial aspect of elbow
7	50	Female	Pustular	ANCA-associated vasculitis	Multiple (>3)	Right leg
8	28	Male	Pustular	Fistulising Crohn's disease; current smoker	1	Lateral aspect of right ankle
9	24	Female	Pustular	Takayasu arteritis	1	Left leg
10	44	Male	Ulcerative	Behçet disease; current smoker	1	Anterior aspect of right leg

Abbreviations: PG, pyoderma gangrenosum.

was undertaken in selected patients because of insufficient response to corticosteroids, slow healing or relapsing disease, and/or the need to control an active associated systemic disease (Table 3). Six patients achieved complete healing without relapse, whereas four experienced at least one relapse during chart-documented follow-up. Because follow-up duration was heterogeneous and incompletely documented in some charts, relapse rates should be interpreted cautiously.

Discussion

This study illustrates the polymorphic nature of PG and underscores the central role of internal medicine in recognising systemic associations and coordinating management [1,2]. The balanced sex distribution observed in our cohort differs from some reports suggesting a slight female predominance, but sex ratios vary widely across published series [4,5]. A key finding is the high frequency of associated systemic diseases (80%). This proportion appears higher than the approximately 50% association rate generally reported in population-based or dermatology-led series [4,5], and may reflect the case mix of an internal medicine referral setting, where patients with multisystem inflammatory disease are overrepresented. These findings support a structured, but clinically guided, search for inflammatory, autoimmune, and autoinflammatory disorders in patients with PG, particularly when the presentation is atypical, recurrent, or treatment-refractory [1,5]. Diagnosis remains challenging because no single clinical, histological, or laboratory feature is pathognomonic. In our practice, diagnosis relied on the combination of a typical painful ulcerative or pustular lesion, exclusion of infection, vasculitis, and malignancy, and supportive biopsy findings when

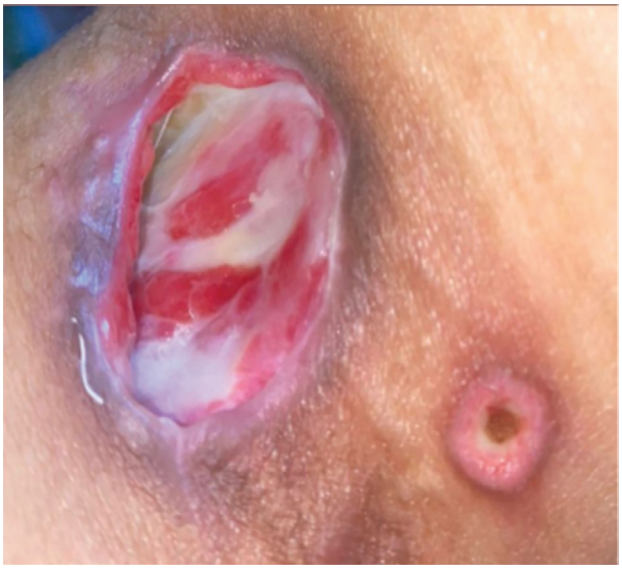

Figure 2. *Upper-limb PG occurring on a postsurgical scar and after an insect bite, illustrating pathergy and the need to avoid unnecessary trauma.*

available. Although the Delphi consensus criteria provide a useful standardized framework, they were not formally applied retrospectively because not all items were consistently documented in the medical records [6]. Pathergy, observed in half of our patients, is an important clinical clue and has practical implications for avoiding unnecessary surgical procedures and for careful wound care [7,9]. Therapeutically, systemic corticosteroids were used in all cases, consistent with their role as first-line therapy [8,9]. Escalation beyond corticosteroids was considered in cases with inadequate early response, slow healing, relapsing or multifocal disease, or when additional control of an active associated systemic disease was required. In this setting, the choice of adjuvant therapy was individualised according to

Table 2. *Summary of laboratory findings.*

CASE	BLOOD COUNT	CRP / ESR	RENAL FUNCTION (UREA/ CREATININE)	LIVER TESTS	SERUM PROTEIN ELECTROPHORESIS	VIRAL SEROLOGIES
1	Normal	CRP normal; ESR normal	Normal	Normal	Normal	Negative
2	Normal	CRP normal; ESR normal	Normal	Normal	Normal	Negative
3	Normal	CRP normal; ESR normal	Normal	Normal	Normal	Negative
4	Normal	CRP 42 mg/l; ESR 35 mm/h	Normal	Normal	Moderate inflammatory pattern	Negative
5	Hb 9.6 g/dl	CRP normal; ESR normal	Normal	Normal	Inflammatory pattern	Negative
6	Neutrophilic leukocytosis	CRP 30 mg/l; ESR 56 mm/h	Normal	Normal	Normal	Negative
7	Neutrophilic leukocytosis; Hb 10.8 g/dl	CRP 30 mg/l; ESR 60 mm/h	Normal	Mild cholestasis	Inflammatory pattern	Negative
8	Neutrophilic leukocytosis; Hb 11.1 g/dl	CRP 70 mg/l; ESR 100 mm/h	Normal	Normal	Inflammatory pattern	Negative
9	Hb 9.2 g/dl (WBC normal)	CRP 60 mg/l; ESR 70 mm/h	Normal	Transaminases 1.5 × ULN	Inflammatory pattern	Negative
10	Neutrophilic leukocytosis (Hb normal)	CRP 10 mg/l; ESR 20 mm/h	Normal	Normal	Normal	Negative

Abbreviations: Hb, haemoglobin; WBC, white blood cell count; CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; ULN, upper limit of normal.

Table 3. *Systemic treatments and rationale for therapeutic escalation in patients with PG.*

PATIENT	SEX	AGE (YEARS)	COMORBIDITY	SYSTEMIC TREATMENT	RATIONALE FOR ADJUVANT THERAPY
1	Female	37	Raynaud phenomenon; systemic sclerosis; hypertension	Systemic corticosteroids + methotrexate + azathioprine + colchicine	No improvement with corticosteroids alone
2	Male	19	Systemic lupus erythematosus	Systemic corticosteroids	—
3	Female	45	Systemic sclerosis; Raynaud phenomenon	Systemic corticosteroids	—
4	Male	58	Current smoker	Systemic corticosteroids	—
5	Male	43	Behçet disease	Systemic corticosteroids + methotrexate + anti-TNF-alpha + colchicine	No improvement with corticosteroids alone; treat underlying Behçet disease
6	Female	24	None reported	Systemic corticosteroids	—
7	Female	50	ANCA-associated vasculitis	Systemic corticosteroids + cyclophosphamide	Treat ANCA-associated vasculitis
8	Male	28	Fistulising Crohn's disease	Systemic corticosteroids + azathioprine + anti-TNF-alpha	Slow healing; treat underlying Crohn's disease
9	Female	24	Takayasu arteritis	Systemic corticosteroids	—
10	Male	44	Behçet disease	Systemic corticosteroids + anti-TNF-alpha	No improvement with corticosteroids alone; treat underlying Behçet disease

Abbreviations: TNF-alpha, tumor necrosis factor alpha.

comorbidity profile and the need for steroid-sparing treatment. Anti-TNFa therapy was used in patients with Behçet disease or Crohn's disease, reflecting a pragmatic strategy that targeted both PG and the associated inflammatory disorder [2]. Overall outcomes were favorable, although relapses occurred in a substantial proportion of patients,

in line with previous reports [4]. Limitations include the retrospective single-center design, the small sample size, and the lack of uniformly documented follow-up duration, which limits the precise interpretation of relapse rates. Nevertheless, our findings provide practical insights relevant to internal medicine, where PG is often encountered in complex systemic contexts.

Conclusion

In this single-center internal medicine case series, PG was frequently associated with systemic inflammatory diseases and required multidisciplinary management. Diagnosis relied on careful exclusion of mimickers and clinico-pathological correlation. Systemic corticosteroids were used in all patients, while additional immunosuppressants and anti-TNF-alpha agents were valuable steroid-sparing options, particularly when PG coexisted with active systemic disease. These findings support a structured, clinically guided systemic work-up in patients with PG.

What is new?

In a single-centre retrospective case series managed in an internal medicine department, the authors found systemic disease associations in 80% of patients, spanning a broad spectrum of inflammatory and autoimmune disorders. They describe practical diagnostic clues (including pathergy) and real-world management patterns showing that treatment choices were guided by both PG severity and the underlying systemic disease, including the use of anti-TNFα agents as steroid-sparing therapy in selected patients.

Acknowledgment

We thank all healthcare professionals involved in the care of the patients. We also thank Professor A.R and Professor Y.S for their support and supervision during the preparation of this work.

List of Abbreviations

ANCA	Anti-neutrophil cytoplasmic antibodies
CRP	C-reactive protein
ESR	Erythrocyte sedimentation rate
IBD	Inflammatory bowel disease
PG	Pyoderma gangrenosum
TNF-alpha	Tumor necrosis factor alpha

Conflict of interest

The authors declare that there is no conflict of interest regarding the publication of this article.

Funding

None.

Consent for publication

Written informed consent for publication of clinical images was obtained from the relevant patients; consent forms are archived by the authors. For this retrospective analysis of de-identified routine-care data, additional individual informed consent was not required according to institutional policy.

Ethical approval

This study was conducted in accordance with the Declaration of Helsinki. Because this retrospective study analyzed anonymized routine-care data, formal ethics committee approval was not required under institutional policy and applicable regulations.

Data sharing

No additional data are available.

Author details

Ilyas El Kassimi[1], Adil Rkiouak[1], Nawal Sahel[1], Meryem Zaizaa[1], Zineb El Bougrini[1], Bilal Talamoussa[1], Youssef Sekkach[1]

1. Department of Internal Medicine, Mohammed V Military Teaching Hospital, Mohammed V University, Rabat, Morocco

References

1. Alavi A, French LE, Davis MD, Brassard A, Kirsner RS. Pyoderma gangrenosum: an update on pathophysiology, diagnosis and treatment. Am J Clin Dermatol. 2017;18(3):355–72. https://doi.org/10.1007/s40257-017-0251-7
2. Fletcher J, Alhusayen R, Alavi A. Recent advances in managing and understanding pyoderma gangrenosum. F1000 Res. 2019;8:F1–19. https://doi.org/10.12688/f1000research.19909.1
3. Braswell SF, Kostopoulos TC, Ortega-Loayza AG. Pathophysiology, diagnosis, and treatment of pyoderma gangrenosum. Dermatol Ther. 2015;28:348–55.
4. Binus AM, Qureshi AA, Li VW, Winterfield LS. Pyoderma gangrenosum: a retrospective review of patient characteristics, comorbidities and therapy in 103 patients. Br J Dermatol. 2011;165(6):1244–50. https://doi.org/10.1111/j.1365-2133.2011.10565.x
5. Langan SM, Groves RW, Card TR, Gulliford MC. Incidence, mortality, and disease associations of pyoderma gangrenosum in the United Kingdom: a retrospective cohort study. J Invest Dermatol. 2012;132(9):2166–70. https://doi.org/10.1038/jid.2012.130
6. Maverakis E, Ma C, Shinkai K, Fiorentino D, Callen JP, Wollina U, et al. Diagnostic criteria of ulcerative pyoderma gangrenosum: a Delphi consensus of international experts. JAMA Dermatol. 2018;154(4):461–6. https://doi.org/10.1001/jamadermatol.2017.5980
7. Su WPD, Davis MDP, Weenig RH, Powell FC, Perry HO. Pyoderma gangrenosum: clinicopathologic correlation and proposeddiagnosticcriteria.IntJDermatol.2004;43(11):790–800. https://doi.org/10.1111/j.1365-4632.2004.02128.x
8. Reichrath J, Bens G, Bonowitz A, Tilgen W. Treatment recommendations for pyoderma gangrenosum: an evidence-based review of the literature based on more than 350 patients. J Am Acad Dermatol. 2005;53(2):273–83. https://doi.org/10.1016/j.jaad.2004.10.006
9. Brooklyn T, Dunnill G, Probert C. Diagnosis and treatment of pyoderma gangrenosum. BMJ. 2006;333(7560):181–4. https://doi.org/10.1136/bmj.333.7560.181