International Medical Case Reports Journal

Dovepress
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CASE REPORT

Case Study of Fulminant Pyoderma Gangrenosum: Conservative Therapy—Cornerstone of Management

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Background: Pyoderma gangrenosum (PG) is a rare, immune-mediated neutrophilic dermatosis characterized by painful, rapidly progressive ulceration and a propensity for pathergy. Diagnosis is clinical and often complicated by nonspecific or evolving histopathologic findings that may overlap with other neutrophilic or granulomatous dermatoses.

Case Presentation: We report a 63-year-old woman with ulcerative colitis who developed extensive, multifocal ulcerations involving peristomal skin, trunk, breast, face, and genital region following colectomy. Early biopsies demonstrated a palisaded neutrophilic and granulomatous dermatitis–like pattern, while subsequent biopsies were nonspecific but compatible with PG in the appropriate clinical context. The patient was managed primarily with systemic biologic therapy, conservative wound care, and strict avoidance of surgical trauma. Limited surgical excision was performed only after prolonged disease control and resulted in durable healing.

Conclusion: This case highlights the diagnostic pitfalls of relying on histopathology alone in PG, emphasizes the rarity and non-ulcerative nature of palisaded neutrophilic and granulomatous dermatitis, and demonstrates that even extensive PG can heal without skin grafting when managed with appropriate systemic therapy and surgical restraint.

Keywords: pyoderma gangrenosum, palisaded neutrophilic and granulomatous dermatitis, pathergy, inflammatory wounds

Introduction

Pyoderma gangrenosum (PG) is a rare, inflammatory neutrophilic dermatosis characterized by painful, necrolytic ulcers with irregular, undermined, violaceous borders. It is frequently associated with systemic inflammatory conditions, most commonly inflammatory bowel disease, inflammatory arthritis, and hematologic malignancies.[1] Despite increasing recognition, PG remains diagnostically challenging due to its variable clinical presentation, lack of pathognomonic histopathologic features, and overlap with other inflammatory dermatoses.[2]

Histologic findings in PG are highly variable and depend on lesion age, biopsy depth, and sampling location. Early lesions may demonstrate dense neutrophilic infiltrates, whereas chronic lesions often show mixed inflammation, fibrosis, or granulation tissue.[3] Consequently, PG is regarded as a clinical diagnosis of exclusion that requires careful clinico-pathologic correlation. Misdiagnosis may lead to inappropriate surgical intervention, which may exacerbate disease through pathergy—an exaggerated inflammatory response to trauma.[4–6]

We present a case of extensive, multifocal PG complicated by histopathologic findings initially suggestive of palisaded neutrophilic and granulomatous dermatitis (PNGD). This case highlights the limitations of histology in isolation and emphasizes the importance of medical management and surgical restraint, particularly within the context of plastic and reconstructive surgery.

https://doi.org/10.2147/IMCRJ.S600540
Received: 1 February 2026
Accepted: 20 April 2026
Published: 12 May 2026

Case Presentation

A 63-year-old African American woman with a history of ulcerative colitis (UC), initially suspected based on colonoscopy findings in October 2019 and treated with corticosteroids and antibiotics, underwent emergent subtotal colectomy with retention of the rectal stump and creation of an end ileostomy for Clostridioides difficile colitis during a UC flare in July 2020. Approximately three months postoperatively, she developed painful peristomal skin erosions. Over the subsequent two months, multiple truncal ulcerations developed spontaneously and progressed rapidly, leading to hospitalization for suspected wound infection. Extensive multifocal ulcerations were present early in the disease course (Figures 1 and 2).

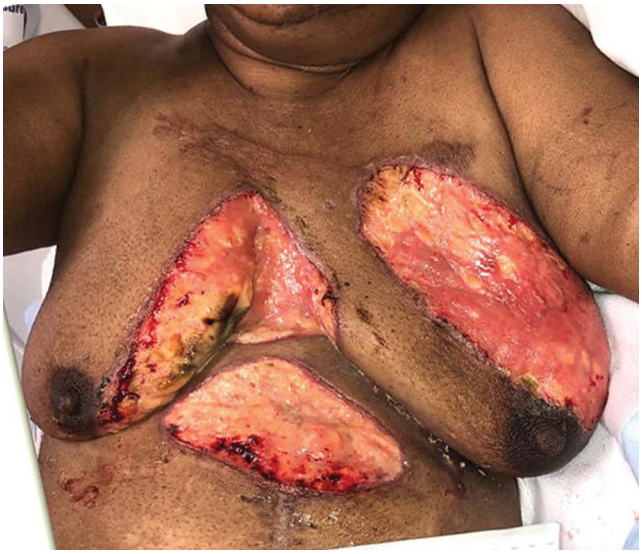

Figure 1 Clinical photograph obtained approximately six months after disease onset demonstrating extensive, multifocal ulcerations involving the anterior torso.

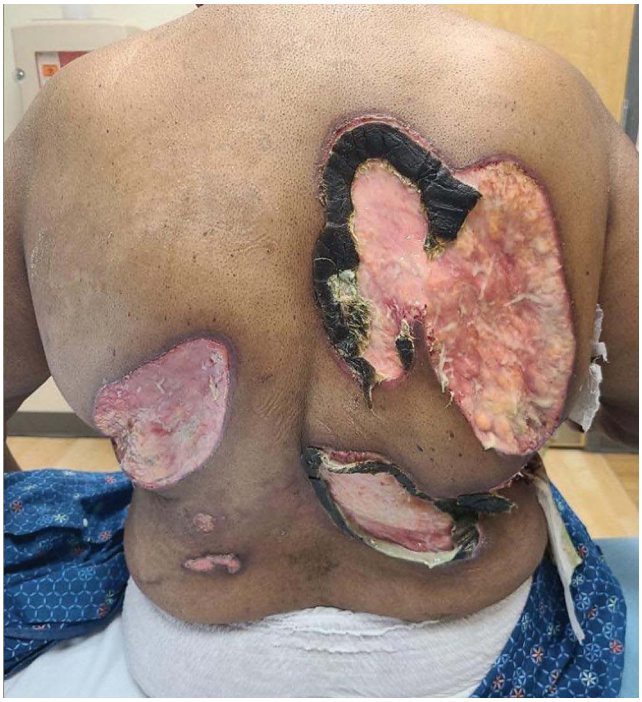

Figure 2 Clinical appearance six months after disease onset (posterior view) demonstrating extensive multifocal ulcerations involving the posterior trunk.

She was initially treated with high-dose systemic corticosteroids for suspected Sweet syndrome; however, her clinical course was complicated by poorly controlled diabetes mellitus. During this time, additional spontaneous ulcerations developed involving the eyebrow and forehead, which were contained. She was referred to a tertiary care center, where biopsy of a truncal lesion demonstrated a palisaded neutrophilic and granulomatous dermatitis–like pattern without

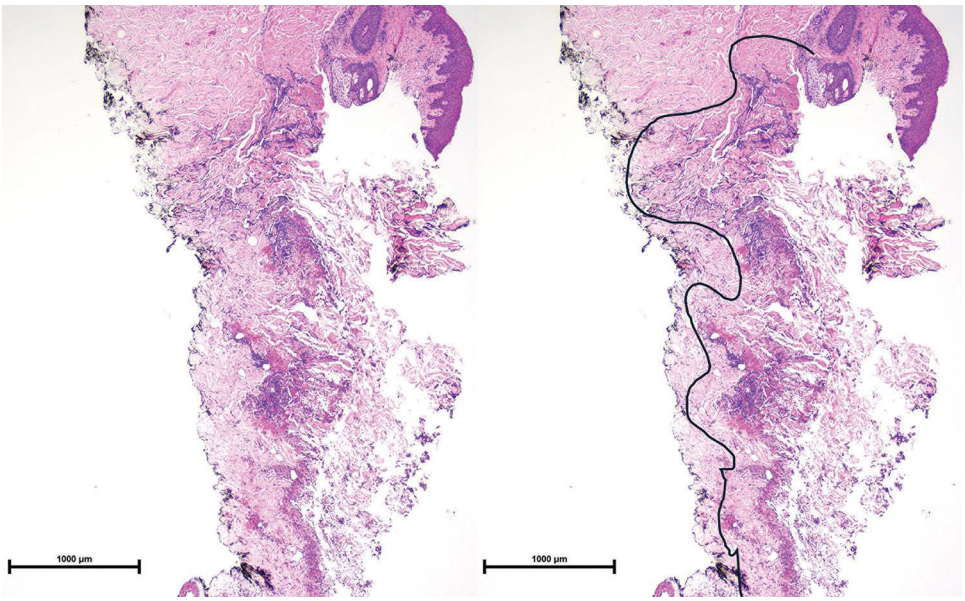

Figure 3 Initial biopsy demonstrated palisaded neutrophilic and granulomatous dermatitis (PNGD-like pattern). Hematoxylin and eosin (H & E) stained section, 20×. Outline shows palisading pattern of inflammation.

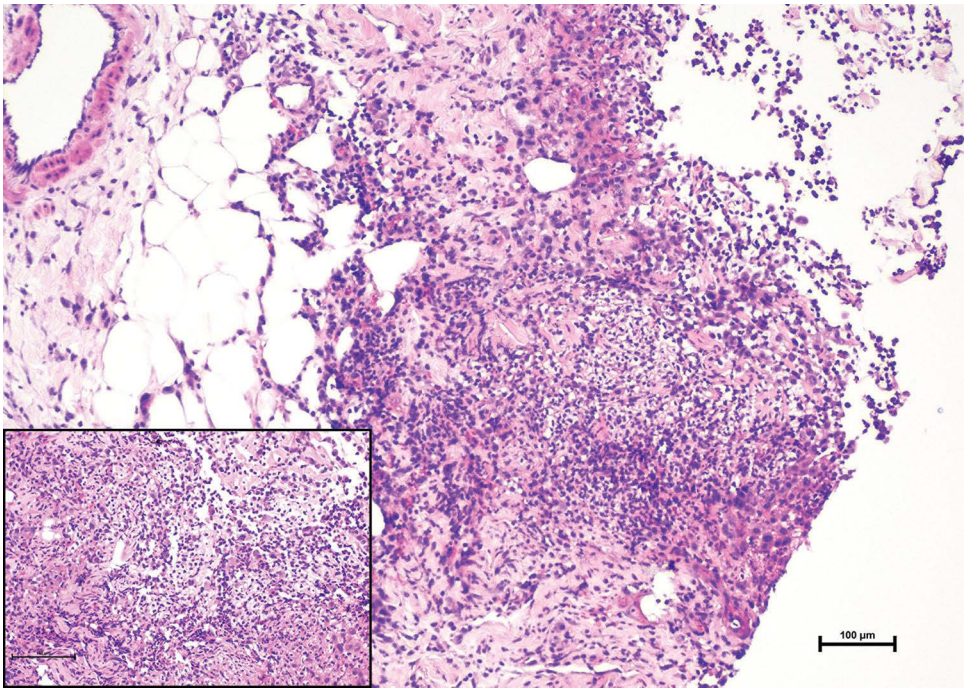

Figure 4 Higher-magnification (100×) H & E section of the initial biopsy: Neutrophilic microabscesses with surrounding histiocytes and lymphocytes from the initial biopsy; in the presence of granulomas with associated neutrophils, infectious etiologies represent an important diagnostic consideration and must be excluded. The inset is taken at 200×.

https://doi.org/10.2147/IMCRJ.S600540

evidence of vasculitis (Figures 3 and 4). Serologic evaluation for rheumatoid arthritis and systemic lupus erythematosus-conditions which can be associated with palisaded neutrophilic and granulomatous dermatitis- was negative.

Wound care was managed intermittently at multiple centers until a private general surgeon raised the suspicion of PG, prompting consolidation of dermatological care at our academic institution. A repeat biopsy of her back lesion performed two months later demonstrated ulceration with scar formation and a more diffuse, interstitial mixed inflammatory infiltrate (lymphocytes, neutrophils, and plasma cells) with rare histiocytes and poorly formed granulomatous zones (Figure 5). While nonspecific, these findings were deemed compatible with PG in the context of UC.

Infliximab was initiated during an inpatient admission but was discontinued following hospital discharge due to insurance denial, because authorization was submitted for a dermatologic rather than gastrointestinal indication. The patient subsequently received conservative wound care through home health services and outpatient wound clinics. UC management included initiation (August of 2022) of vedolizumab (300 mg) infusions every 6–8 weeks. Figure 6 demonstrates the appearance of the breasts and abdomen at the peak of disease progression (approximately 20 months after disease onset), photographed two months prior to initiation of vedolizumab. Ten months later, due to persistent active proctitis in the rectal stump, adalimumab 40 mg subcutaneously every 14 days was initiated in June 2023. Adalimumab was previously denied by insurance for dermatologic reasons.

Over the following year, the patient demonstrated marked improvement in wound burden with progressive healing. Initially wound care included twice per day dressing changes with silver-impregnated alginate dressing, which was Silvercel™ (Solventum, St. Paul, MN) covered with abdominal pad where needed and topical corticosteroids (triamcinolone 0.1% followed by clobetasol 0.05%). For secondary dressing, a polyurethane foam dressing with soft silicone adhesive border was used. The foam core also handled exudate management. Three years into her disease, dermatology added topical timolol 0.5% drops applied twice daily and alternating dilute bleach baths and Epsom salt baths.

At approximately 3.5 years after disease onset, a recalcitrant, painful ulcer in the left inframammary crease failed to resolve despite quiescent systemic disease (Figure 7). Given prolonged stability on biologic therapy, the patient underwent limited surgical excision and primary closure. Healing was durable with no recurrence at the 18-month follow-up (Figure 8).

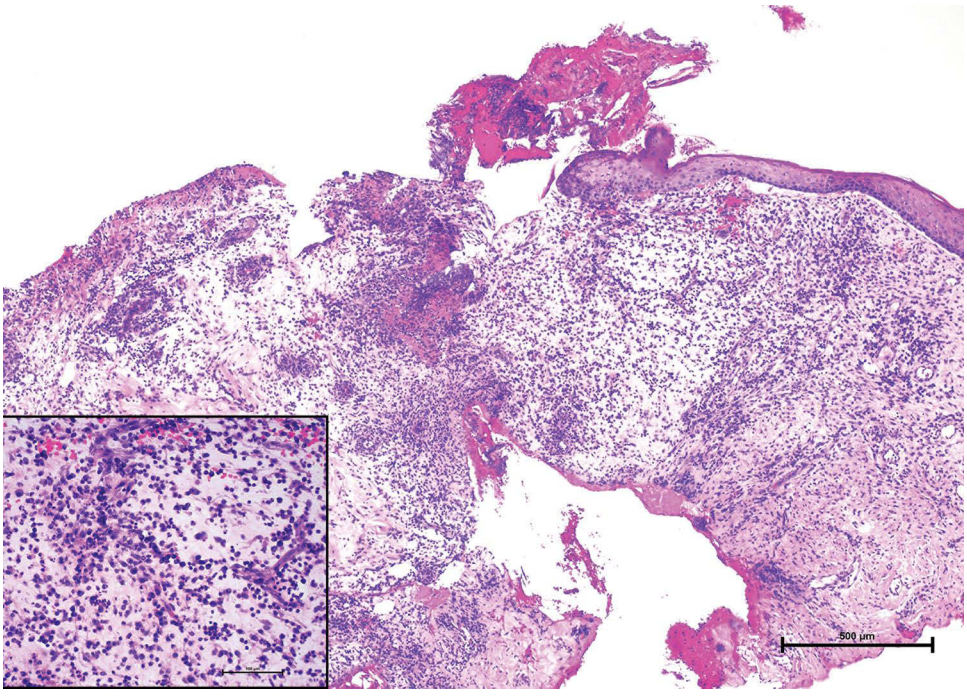

Figure 5 H & E stained 40× section of subsequent biopsy demonstrated nonspecific inflammatory features. Biopsy obtained approximately two months later demonstrating ulceration with associated scar formation and a more interstitial and diffuse mixed inflammatory infiltrate composed predominantly of lymphocytes, neutrophils, and plasma cells. Compared with the initial biopsy, there are fewer histiocytes and less well-defined granulomatous zones. These features are not supportive of palisaded neutrophilic and granulomatous dermatitis. While nonspecific, the findings may be compatible with pyoderma gangrenosum in the appropriate clinical context. The inset is taken at 200×.

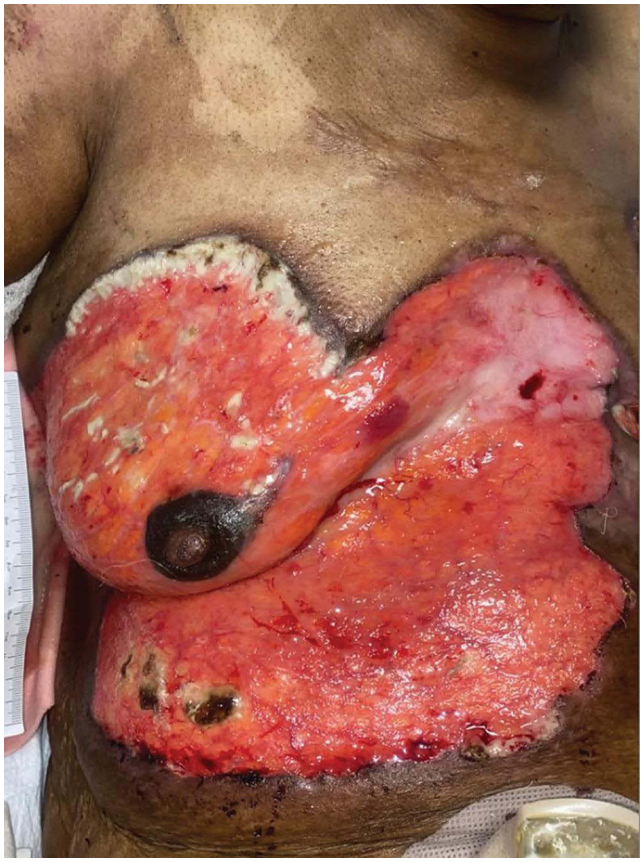

Figure 6 Clinical appearance of breast and abdomen at peak of disease progression (approximately 20 months after disease onset) demonstrating extensive ulceration on her entire right breast and right upper quadrant of abdomen. Photo was taken 2 months before the patient was started on vedolizumab 300 mg infusion.

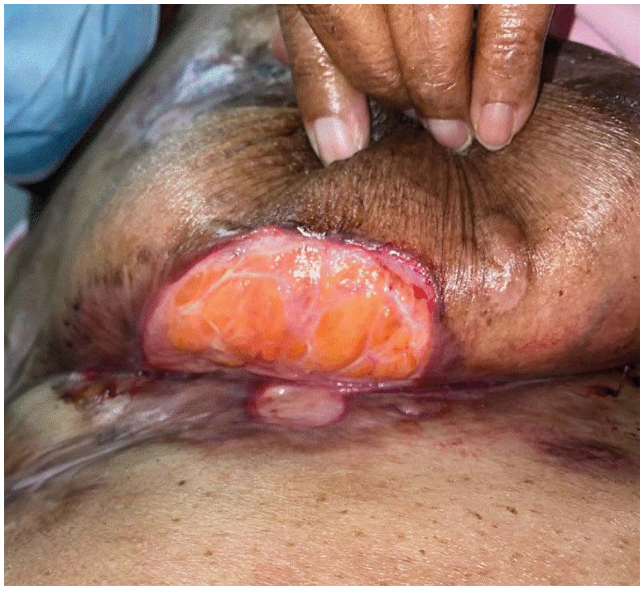

Figure 7 Chronic nonhealing inframammary wound prior to surgery: Clinical photograph demonstrating a persistent, painful, nonhealing ulcer involving the left inframammary crease approximately 3.5 years after disease onset, despite otherwise well-controlled systemic disease.

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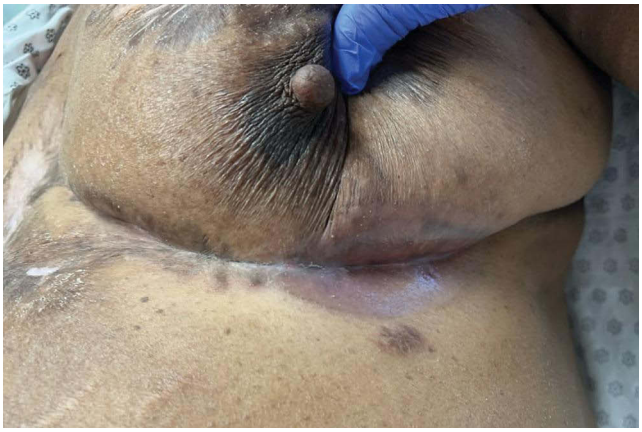

Figure 8 Clinical photograph obtained 1.5 years after limited surgical excision and layered closure of the left inframammary wound, demonstrating durable healing without recurrence.

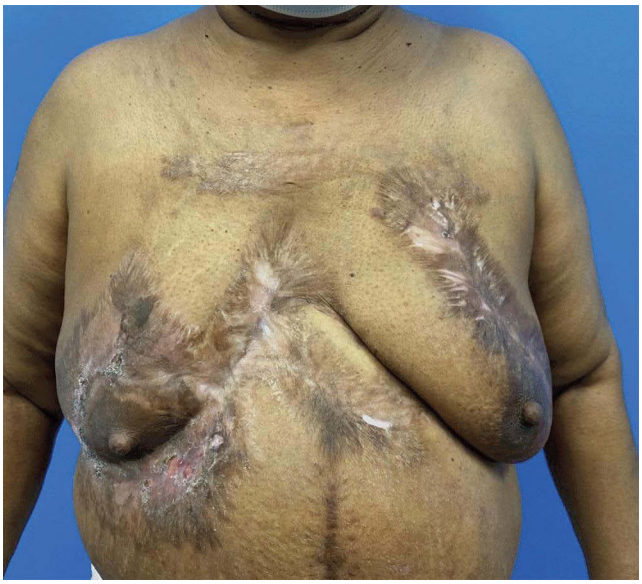

Figure 9 Clinical appearance five years after disease onset (anterior view) demonstrating near-complete resolution of anterior truncal lesions following prolonged biologic therapy and conservative wound management. Cosmesis was never a concern for this patient.

Subsequently, dapsone therapy was initiated for minor recurrent lesions, including one on her labia majora, but was discontinued after several months due to noncompliance. At five years following disease onset, the patient demonstrated near-complete resolution of all cutaneous lesions, including sustained healing of the left breast surgical site. Following prolonged biologic therapy and conservative management, near-complete resolution was observed at five years (Figures 9 and 10). Table 1 summarizes the timeline of disease course, treatments, and outcomes.

Discussion

This case illustrates critical principles in the management of complex inflammatory wounds, particularly the importance of clinicopathologic correlation and the values of non-surgical management.

The primary diagnostic hurdle was the discrepancy between early histology (PNGD-like pattern) and the evolving clinical phenotype. Subsequent biopsies showed nonspecific ulceration with fibrosis and mixed inflammation. Such variability is well documented in PG and reflects differences in lesion age and sampling.[7,8] The absence of classic neutrophilic predominance does not preclude a diagnosis of PG when clinical features, such as association with UC and

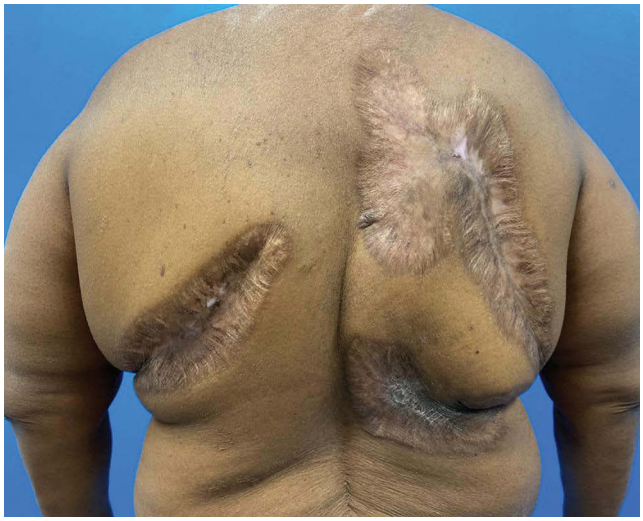

Figure 10 Clinical appearance five years after disease onset (posterior view) demonstrating near-complete resolution of posterior truncal lesions.

rapid, painful ulcerations, are compelling, and the histopathologic features of PG are notoriously nonspecific, overlapping with infectious and other inflammatory etiologies.[9]

PNGD is a rare histopathologic reaction pattern typically associated with systemic autoimmune disease,[10] usually presenting as papules, plaques, or nodules rather than large, necrolytic ulcers.[11] We performed a structured literature search using OVID MEDLINE with the combined terms "palisading", "neutrophilic", "granulomatous", and "dermatitis" yielded 80 publications, most of which were single case reports or small case series. Across this literature, PNGD is not described as a cause of non-healing open wounds. Therefore, clinicians must avoid over-indexing on PNGD histology when the clinical presentation is aggressively ulcerative. Indeed, while currently underemphasized in dermatopathology teaching, early histopathologic descriptions of PG included the presence of granulomatous infiltrates, and therefore a PNGD-like pattern on biopsy in the setting of clinical ulcers may more accurately represent PG.[8]

Regarding the role of biologic therapy, TNF-α inhibitors remain the gold standard for refractory PG. While insurance barriers delayed optimal treatment in this case, the eventual initiation of adalimumab was the catalyst for progressive and sustained healing in our patient. TNF-α inhibitors, including infliximab and adalimumab, are among the most effective therapies for PG, with infliximab supported by randomized controlled trial data.[7,12] Although infliximab could not be continued due to insurance barriers, initiation of adalimumab was associated with sustained improvement and near-complete healing.

When treating PG, clinicians should recognize that anti–TNF agents such as infliximab can paradoxically induce neutrophilic dermatoses, including pyoderma gangrenosum.[13] However, in this case, the patient's cutaneous lesions preceded initiation of biologic therapy, making a drug-induced etiology unlikely. Clinicians should remain cognizant of this potential paradoxical effect if an existing lesion worsens or a new lesion develops.

Looking ahead, several targeted and adjunctive therapies may expand future treatment options for pyoderma gangrenosum,[14] including complement C5a inhibition (vilobelimab), IL-12/23, IL-17 receptor A, and IL-23 inhibitors, as well as intravenous immunoglobulin. At present, however, these therapies are supported primarily by case reports, retrospective data, or early-phase studies rather than robust randomized evidence.

Avoidance of pathergy is a cornerstone of PG management. Surgical debridement or aggressive local intervention may precipitate lesion expansion and clinical deterioration.[5,9] In this case, even extensive multifocal wounds healed through medical optimization and conservative, atraumatic wound care. Surgical intervention was employed only for an inframammary crease wound that remained recalcitrant due to chronic intertrigo with persistent moisture and maceration. Limited surgical excision was performed only after prolonged disease quiescence and resulted in durable healing, underscoring the importance of surgical restraint.

Table 1 Timeline of Disease Course, Treatments, and Outcomes

Time from Onset	Key Events & Findings	Treatment	Outcome
0 months	Fulminant Clostridioides difficile colitis	Emergent subtotal colectomy	
3–5 months	Peristomal erosions → rapidly progressive truncal ulcerations.	Admission- High-dose corticosteroids (suspected Sweet syndrome)	Wound progression; pre-existing IDDM→ poorly controlled hyperglycemia
6–7 months	New lesions (eyebrow, forehead) with general wound progression and severe pain.	Transfer care to tertiary medical center recommended	Treated for wound sepsis with IV antibiotics
7 months	Admitted to tertiary medical center	Skin biopsy (back): Palisaded, necrotizing granulomatous inflammation	Comprehensive work-up and wound care. Serologies were negative. Treated for pulmonary and wound infection.
9 months	Readmitted for progression of wounds- bilateral breasts, abdomen, and back.	Repeat biopsy suspicious for PG. Infliximab initiated but discontinued by insurance	Discharged to skilled nursing facility. Limited response in wound healing.
15–20 months	Peak progression of ulcerative wounds	Conservative wound care	Severe disease persists
25 months	Active disease found on rectal biopsy; UC-directed therapy	Vedolizumab initiated	Gradual improvement; epithelialization
35 months	Persistent proctitis	Adalimumab approved for UC and initiated; Vedolizumab discontinued	Continued healing
42–47 months	Recalcitrant left inframammary ulcer	Surgical excision and closure (~4 years from disease onset)	Durable healing
60 months	Resolution of wounds	Ongoing biologic + conservative care	Sustained remission

Abbreviations: UC, ulcerative colitis; PG, pyoderma gangrenosum; IDDM, insulin-dependent diabetes mellitus.

Adjunctive Topical Therapies

Topical timolol has been increasingly reported as an adjunctive therapy for chronic wounds, including PG, owing to its effects on angiogenesis and keratinocyte migration.[10] Dilute bleach baths are commonly used in inflammatory dermatoses to reduce bacterial colonization and secondary infection.[15,16] Although evidence specific to PG is lacking, both interventions were well tolerated in our patient and may have provided supportive benefit.

Additional topical agents reported with variable success in the literature include 2% Phenytoin sodium solution, tacrolimus ointment, sucralfate powder, corticosteroids, and combination tacrolimus/clobetasol therapy.[14]

Haroon et al identified antimicrobial dressings and hyperabsorbent dressings to be the most frequently used dressings for managing PG ulcers.[17] Conservative wound care focuses on addressing devitalized tissue, exudate, and infection. Fibrin and necrotic tissue can be reduced by enzymatic debridement or autolytic methods such as hydrogel application. Nonadherent and absorbent dressing (foam, alginate, and hydrofiber) are commonly employed, as in our case. In general, dressings should be nonadherent and easily removable to minimize trauma, pain, and pathergy.[18]

Negative pressure wound therapy (NPWT) has also gained increasing clinical use in this context to prepare the wound bed for definitive treatment or healing by the second intention, and to assist the integration of skin grafts.[19,20] NPWT utilize sub atmospheric pressure applied to the wound surface leading to reduction of local edema, increased perfusion, and enhance cellular proliferation. A systematic review of 57 articles on the use of NPWT, improvement in wound healing with the use of NPWT was observed in 74 patients (85.1%), while 10 patients (11.5%) did not improve; unspecified in 3 patients (3.4%).[20]

The role of surgical treatment in PG remains controversial as 25–50% of PG lesions demonstrate pathergy, which could worsen with surgical intervention.[21,22] Even with remission, the pathergy phenomenon persists. In a small cohort of 12 patients with known PG but had no active disease for an average 2.15 years, 6 (50%) experienced pathergy following routine surgical procedure.[23]

From a plastic surgery standpoint, this case illustrates that even extensive, multifocal PG wounds can heal without skin grafting or major reconstruction when managed appropriately. Early recognition of PG and avoidance of unnecessary surgical intervention are critical to preventing disease exacerbation and improving outcomes. In cases of severe wound infection, such as necrotic ulcers causing systemic sepsis, surgical debridement or even amputation may be unavoidable.[24] Without wound sepsis, procedures such as skin grafting may be considered but should be performed under adequate immunosuppression in a patient with stable or quiescent disease.[25]

Conclusion

This case underscores the diagnostic complexity of pyoderma gangrenosum, the limited utility of histopathology in isolation, and the critical role of multidisciplinary management. With appropriate and timely systemic therapy, meticulous conservative wound care, and careful avoidance of pathergy, even extensive PG wounds can heal without major reconstructive surgery. These observations may offer valuable insight for clinicians involved in the management of PG.

Ethical Approval and Consent

Ethical approval is not required to publish the case details in accordance with local or national guidelines. The hospital does not require ethical clearance for case reports. Written informed consent (Taylor & Francis Consent Form) for publication of their details, which includes photographs, was obtained from the patient presented. At our institution, no IRB is required for case series of 3 or fewer patients. Refer to Institutional Policy on Case Reports.

Funding

The authors received no financial support for the research, authorship, and/or publication of this article.

Disclosure

Sara Shalin reports being a committee member of American Board of Pathology Test Development Committee (dermatopathology), Editorial Board- Modern Pathology, and Editorial Board- Journal of Cutaneous Pathology. The authors report no other conflicts of Interest in this work.

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