J. Pharm Pract Comm Med, 2026; 12(4):317-320.
www.jppcm.org

Case Report

A Case Report on *Pyoderma gangrenosum*

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ABSTRACT

Pyoderma gangrenosum is a rare dermatological condition characterised by ulceration of the skin. It is more frequently observed in adults than in children. It is associated with inflammatory bowel disease, rheumatoid arthritis and lymphoproliferative disorders. A 12-year-old female reported a lesion on the fore arm 1 year back and was on treatment. Now, she has complaints of lesions over arms and thighs covering almost 30% area associated with burning sensation and pain and was prescribed tablet prednisolone, tablet dapsone and tacrolimus ointment. Although *Pyoderma gangrenosum* is a rare disease, clinicians should suspect it in the presence of purplish wound edges with a necrotic centre. Surgical debridement, while necessary in other ulcers, can worsen the condition and is only applicable to self-detachable necrotic plaques.

Keywords: Chronic Inflammatory Dermatosis, Corticosteroid Therapy, Erythematous Lesions, Immunosuppressive therapy, Neutrophilic Dermatosis, Pathergy Phenomenon, *Pyoderma gangrenosum*.

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Received: 23-02-2026;
Revised: 03-04-2026;
Accepted: 16-06-2026.

INTRODUCTION

Pyoderma gangrenosum (PG) is a rare dermatological condition that was first described by Brocq, a French dermatologist, in 1916 (Brocq, 1916). 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 (Weenig *et al*., 2002). *Pyoderma gangrenosum* is characterised by one or a few chronic ulcerations with typical violaceous, undermined borders. Rare cases with acute onset of multiple ulcerating or chronic vegetating and granulomatous lesions, as well as bullous PG in association with leukaemia, have been described (Driesch, 1997). It is characterised by rapidly progressing ulceration of the skin with an ill-defined border and can occur at any age, but is more frequently observed in adults than in children. It has a gender predilection for females and commonly affects the lower extremities, in particular the pretibial area (Ye and Ye, 2014). Although of non-infectious etiology, the pathogenesis of the disease is unclear, but evidence suggests an underlying defective neutrophilic function (Androutsakos *et al*., 2015).

DOI: 10.5530/jppcm.20260878

Etiology

After excluding more common causes of cutaneous ulcerations, the diagnosis of PG is suggested by the presence of a painful, necrolytic cutaneous ulcer, with an irregular, undermined border, showing rapid progression. Histologically, there is lymphocytic infiltration in the early stages, followed by neutrophilic infiltration and haemorrhage. *Pyoderma gangrenosum* is related to a concomitant disease in 50 to 70% of cases, including Inflammatory Bowel Disease (IBD) in 10 to 15%, atrophic arthritis, and lymphoproliferative disorders. Although PG has been encountered with hepatitis, only a small number of cases are reported with Autoimmune Hepatitis (AIH), one related to concurrent primary sclerosing cholangitis and ulcerative colitis (Androutsakos *et al*., 2015).

Clinical Presentation

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. The ulcer is exquisitely painful. Pustular, bullous, and vegetative or superficial granulomatous variants have also been described (Su *et al*., 2004).

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 necrotising 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 (Burgess and Lari, 1991; Al-Rimawi *et al.*, 1996; Graham *et al.*, 1994).

PG occurs most ordinarily on the lower legs with a preference for the pretibial area. PG has been reported on other sites of the body also, including breast, hand, trunk, head, neck, and peristomal skin. Extracutaneous manifestations include involvement of the upper airway mucosa, eye, genital mucosa, sterile pulmonary neutrophilic infiltrates or spleen infiltrates, and neutrophilic myositis. Sterile cortical osteolysis has been observed adjacent to PG ulcers as another extracutaneous manifestation of the disease (Wollina, 2007).

Diagnosis

Diagnosis of PG requires clinicopathologic correlation and is often a diagnosis of exclusion after common causes of skin ulceration, such as infection, malignant neoplasms, and vasculitic syndrome. Histopathological findings of PG are not specific. Early lesions may reveal dermal neutrophilia centred on follicles, while severe skin lesions may show tissue necrosis with surrounding mononuclear cell infiltrates (Ye and Ye, 2014).

Proposed diagnostic criteria of classic, ulcerative *Pyoderma gangrenosum* (PG) requires both major criteria and a minimum of two minor criteria.

Major Criteria

- Rapid progression of a painful, necrolytic cutaneous ulcer with an irregular, violaceous, and undermined border.
- Other causes of cutaneous ulceration are excluded.

Minor Criteria

- History suggestive of pathergy or clinical finding of cribriform scarring.
- Systemic diseases associated with PG.
- Histopathologic findings (sterile dermal neutrophilia, ± mixed inflammation, ± lymphocytic vasculitis).
- Treatment response (rapid response to systemic steroid treatment).
- Characteristic margin expansion of 1 to 2 cm per day, or a 50% increase in ulcer size within 1 month.
- Pain is typically out of proportion to the dimensions of the ulceration.
- Typically preceded by a papule, pustule, or bulla.
- Usually necessitates skin biopsy and other investigations to rule out causes.
- Ulcer development at sites of minor cutaneous trauma.
- Inflammatory bowel disease, arthritis, IgA gammopathy, or underlying malignancy.
- Generally, responds to a dosage of Prednisolone 1 mg/kg to 2 mg/kg per day, with a 50% decrease in size within 1 month (Shahid *et al.*, 2014).

Differential Diagnosis

Six disease categories may imitate the clinical appearance of PG (Wollina, 2007):

a) Vascular occlusive or venous disease, including calciphylaxis, which is particularly painful and rapidly evolving.

b) Vasculitis - This is a particular challenge when PG develops in a patient with vasculitic rheumatoid arthritis. Differential diagnoses include Wegener's disease, phospholipid syndrome, or Adamantiades-Behcet's disease.

c) Cancer - The diagnosis is often most intricate in patients with lymphomas or leukaemia, where the cutaneous lesions may present as suppurative ulcers.

d) Infectious disease - Ecthymata and deep tropical mycoses such as sporotrichosis may resemble PG. The rapid onset of post-surgical PG often resembles acute deep skin infection such as erysipelas or gangrene. Late syphilis may also produce suppurative ulcerations. Deep viral herpetic infections can resemble PG.

e) Exogenous tissue injury - Factitious panniculitis as a part of Münchhausen syndrome may masquerade as PG. Insect or spider bites can cause necrotising skin ulcers.

f) Drug reactions - Pustular drug reactions may masquerade as pustular PG (Wollina, 2007).

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. 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. The factors inciting or maintaining these abnormalities are unclear but likely multifactorial, including genetic predisposition, undefined infectious agents, or paraneoplastic or para-immune phenomena. Rare familial forms of PG have been reported. The recently described "PAPA syndrome" (pyogenic sterile arthritis, PG, and acne) is an autosomal dominant disorder that maps to chromosome 15q. The IL-16 gene maps to 15q and is perhaps overexpressed in this

disorder because the IL-16 protein is chemotactic to neutrophils (Weenig *et al.*, 2002).

Treatment

Treatment for PG can involve a combination of wound care, topical therapy, and systemic therapy. Wound care is important for all lesions and is tailored to the type of lesion and extent of associated exudate. A moist environment is typically recommended to promote wound healing. It is also important to monitor for signs of superimposed infection. Topical treatment as monotherapy can be applied for localised and mild disease, but can also be used in combination with systemic treatment for more severe lesions. Topical therapies that have been tried with some success include corticosteroids, tacrolimus, ciclosporin, 5-aminosalicylic acid, and dapsone. More extensive disease is usually treated with systemic steroids. Immunosuppressive agents, most commonly ciclosporin, and others such as azathioprine, can also be used. Cyclophosphamide, mycophenolate, and tacrolimus are also used. Systemic dapsone has shown good results in PG. Recently, biologic therapy, particularly infliximab, has shown very good results in patients with pyoderma gangrenosum associated with inflammatory bowel disease (IBD) (Shahid *et al.*, 2014).

CASE REPORT

A female patient of age 12 years presented with a new lesion on the forearm. She reported a small rash on her left arm 1 year back and was on medication. There was a sudden appearance of erythematous lesions on both arms and thighs covering almost 30% area associated with severe burning sensation and pain. The patient denied having fever, weight loss, or other signs and symptoms of systemic illness. On further questioning, the patient's representative reported loss of appetite. On examination, lesions were large, spreading over arms and limbs as seen in Figure 1.

Investigations

The following investigations were carried out to evaluate disease activity and to rule out associated systemic conditions, and monitor drug safety.

- Hemogram- within normal limits except mild inflammatory changes
- Erythrocyte Sedimentation Rate (ESR) and C-Reactive Protein (CRP) - mildly elevated, suggestive of ongoing inflammation
- Liver Function, Renal Function Tests - within normal limits
- Blood glucose levels - monitored during corticosteroid therapy and found within normal range
- Peripheral smear examination - no significant abnormalities detected

Figure 1: Showing a lesion on the fore arm.

- G6PD screening prior to initiation of Dapsone therapy was found to be normal
- Wound swab culture and sensitivity - no significant pathogenic bacterial growth detected
- Autoimmune profile, including ANA, was negative
- Screening for associated systemic diseases, such as inflammatory bowel disease and rheumatoid arthritis, revealed no significant findings
- Skin biopsy from the lesion showed dense neutrophilic dermal infiltrate with ulceration, consistent with *Pyoderma gangrenosum*
- Viral screening tests, including HBsAg, HIV, and HCV, were non-reactive

Monitoring of haematological parameters, liver function and renal function was advised during continued therapy with corticosteroids and dapsone.

The patient was previously diagnosed with *Pyoderma gangrenosum* and was on medication for 1 year. At the first visit, the patient was having a small lesion on the fore arm and was prescribed Tablet prednisolone and Fusidic acid ointment for 3 months. In the next follow-up, the patient was advised with Tablet Prednisolone, Tab Naproxen 250 mg and silver colloid gel for 2 months. In the third follow-up, she was advised to use Tablet Dapsone 50 mg and Tacrolimus 0.03% ointment for 4 months. Now the patient came with complaints of erythematous lesions on arms and thighs with burning sensation and pain. So, the following treatment plan was prescribed to the patient is Tablet Prednisolone 10 mg once

daily orally for 5 days following 5 mg once daily orally for the next 5 days, Tablet Dapsone 50 mg orally at night and Tacrolimus ointment 0.1% twice daily to be applied over lesions.

The patient was advised to undergo regular follow-up for monitoring of lesion progression, treatment response, and potential adverse drug reactions. Proper wound care, avoidance of trauma to lesions, and adherence to medications were emphasised.

CONCLUSION

Clinical Conclusion

The patient is a known case of *Pyoderma gangrenosum* with recurrent inflammatory cutaneous lesions requiring long-term immunomodulatory therapy. Despite initial improvement with systemic corticosteroids and topical therapy, the patient developed recurrent erythematous painful lesions involving the arms and thighs, suggestive of disease relapse/reactivation. Stepwise management with corticosteroids, anti-inflammatory agents, and steroid-sparing therapy such as Dapsone and Tacrolimus was instituted to control disease activity and reduce long-term steroid exposure. Continuous follow-up and monitoring for therapeutic response, adverse drug reactions, and recurrence are essential for successful disease management and prevention of complications.

Learning Points

- *Pyoderma gangrenosum* is a chronic neutrophilic dermatosis characterised by painful inflammatory skin lesions with recurrent episodes.
- Early diagnosis and prompt initiation of immunosuppressive therapy help reduce disease progression and tissue damage.
- Systemic corticosteroids remain the mainstay of treatment during acute exacerbations.
- Steroid-sparing agents such as Dapsone and topical Tacrolimus are useful in long-term management to minimise prolonged corticosteroid use.
- Regular follow-up is important to assess treatment response, monitor adverse effects, and identify relapses early.
- Patient education regarding medication adherence and early reporting of new lesions or worsening symptoms plays a vital role in disease control.
- Management of *Pyoderma gangrenosum* often requires an individualised and multidisciplinary approach depending on disease severity and recurrence pattern.

Surgical debridement should generally be avoided in active *Pyoderma gangrenosum* lesions, as trauma may precipitate worsening of lesions due to the phenomenon of pathergy; conservative wound care and medical management are preferred unless clearly indicated.

ABBREVIATIONS

PG: *Pyoderma gangrenosum*; **IBD:** Inflammatory Bowel Disease; **AIH:** Autoimmune Hepatitis; **IL-8:** Interleukin-8; **IL-16:** Interleukin-16; **PAPA Syndrome:** Pyogenic Arthritis, *Pyoderma gangrenosum*, and Acne Syndrome; **ESR:** Erythrocyte Sedimentation Rate; **CRP:** C-Reactive Protein; **G6PD:** Glucose-6-Phosphate Dehydrogenase; **ANA:** Antinuclear Antibody; **HBsAg:** Hepatitis B Surface Antigen; **HIV:** Human Immunodeficiency Virus; **HCV:** Hepatitis C Virus.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

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Cite this article: Gupta A, Vyshnavi K, Soumya M, Puranam Y, Mounika P, Vanjari NK. A Case Report on *Pyoderma gangrenosum*. J Pharm Pract Comm Med. 2026;12(4):317-20.