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# Extracutaneous Manifestation of Ulcerative Colitis: Pyoderma Gangrenosum, A Case Report

**Rohan Sapkota,[1] Eliz Aryal,[1] Prashanna Raj Shrestha,[1] Jyoti Bhari,[1] Sunny Shrestha[1]**

[1]Department of Dermatology, Kathmandu Medical College, Sinamangal, Kathmandu, Nepal

## Abstract

Pyoderma gangrenosum (PG) is a rare, ulcerative skin condition often associated with systemic diseases such as inflammatory bowel disease (IBD), including ulcerative colitis (UC). This case report describes a 79-year-old male patient with a history of ulcerative colitis who presented with increased passages of blood-mixed stools and a solitary ulcer on the left lower shin following minor trauma. The ulcer, measuring 10 cm by 4 cm, exhibited a violaceous border with purulent necrotic tissue. Histopathological examination revealed neutrophilic exudate, dense inflammatory cell infiltrate, perivascular neutrophilic infiltration, extravasated red blood cells, congested blood vessels, and hemosiderin-laden macrophages. The clinical presentation, along with the biopsy findings, confirmed the diagnosis of PG associated with UC. The patient was managed with systemic corticosteroids and antineutrophilic therapy, emphasizing the importance of early diagnosis and treatment to prevent disease progression. This case underscores the need for vigilance in identifying PG in patients with IBD, as prompt intervention can significantly improve outcomes.

**Keywords:** Extracutaneous; Inflammatory Bowel Disease; Neutrophilic dermatoses; Pyoderma gangrenosum

## Introduction

Inflammatory bowel disease (IBD) encompasses several chronic inflammatory conditions, most significantly ulcerative colitis (UC) and Crohn's disease (CD). While these two conditions share many common features – diarrhea, bloody stools, weight loss, abdominal pain, fever, and fatigue – each has unique features.[1]

The current working hypothesis of disease pathogenesis is a dysregulated mucosal inflammatory response to intestinal microbes in genetically susceptible individuals. Notably, genes implicated in mucosal barrier function (ECM1, CDH1, HNF4a, and laminin B1) confer a risk of UC; furthermore, E-cadherin is the first genetic correlation between colorectal cancer and UC. The pathogenesis of EIM in IBD is not well understood. It is believed that the diseased gastrointestinal mucosa may trigger immune responses at the extraintestinal site due to shared epitopes.[2]

The different clinical types of PG are ulcerative, pustular, bullous, and vegetative. The legs are most affected, but other parts of the skin and mucous membranes may also be affected. In many cases, PG is associated with an underlying disease, most commonly inflammatory bowel disease, rheumatic or hematological disease, and malignancy.[3]

Here, we present a case of a 79-year-old male with a history of the passage of blood-mixed stools for 20 days and a solitary ulcer over the left lower shin since 15 days.

## Case Report

A 79-year-old male with a known case of ulcerative colitis for 4 years presented to Kathmandu Medical

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**Corresponding Author:**
Dr. Rohan Sapkota
Department of Dermatology,
Kathmandu Medical College,
Sinamangal, Kathmandu, Nepal
ORCID ID: 0009-0009-4604-0757
Email: rohan123.sapkota@gmail.com

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College Teaching Hospital, Emergency Department, with a chief complaint of increased passages of loose stools, initially 8-10 times later uncountable. The stool was mixed with blood and mucus, with an offensive smell, 1-2 teaspoonfuls per defecation. He had a similar history 4 years back. Two weeks later, the patient developed a lesion on the left shin, which was initially papular with mild peripheral swelling, which gradually progressed to form a tender ulcer of size 10 cm by 4 cm, which had a violaceous border with purulent or necrotic tissue (Figure 1). There were no similar lesions present in other body parts. Routine blood tests were done. A punch biopsy of the lesions was performed, and the tissue was sent for histopathological analysis

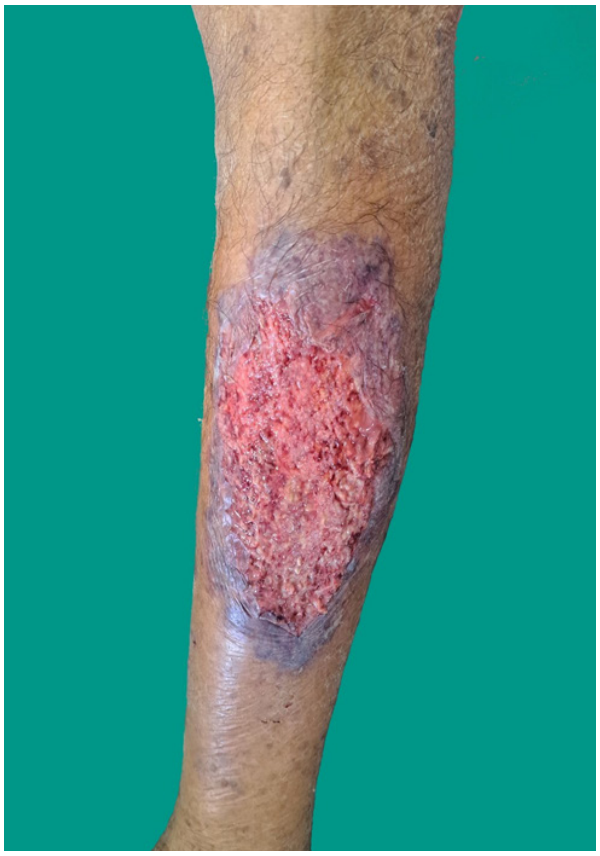

Figure 1: A large, irregularly shaped ulcer on the left lower shin, characterized by a red, raw wound bed with visible granulation tissue. The surrounding skin appears inflamed with a purplish border.

to confirm the clinical diagnosis. The dermis showed extensive inflammation on histopathological analysis, with a mixture of neutrophils, lymphocytes, and histiocytes. There was also fibrinoid necrosis of the vessel walls, consistent with the diagnosis of pyoderma gangrenosum, highlighting the vasculitis component of the disease (Figure 2).

Laboratory investigations: The patient's laboratory results were analyzed for abnormalities relevant to the diagnosis. Elevated markers of inflammation, including ESR (60) and CRP (79), were noted. Routine blood tests

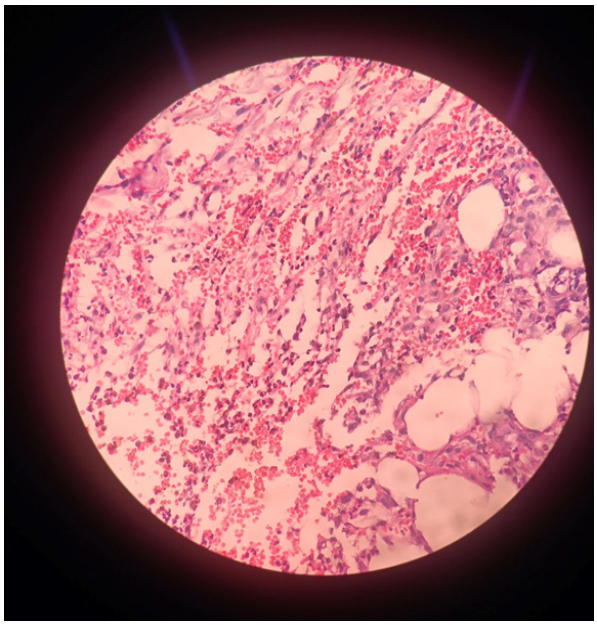

Figure 2: Dermis shows extensive inflammation, with a mixture of neutrophils, lymphocytes, and histiocytes. There is also fibrinoid necrosis of the vessel walls, consistent with the diagnosis of pyoderma gangrenosum, highlighting the vasculitis component of the disease. (H&E, ×40)

revealed anemia (hemoglobin: 10.1 g/dL), leukocytosis (WBC: 14,000/µL), and hypoalbuminemia (serum albumin: 2.8 g/dL). Sputum culture and sensitivity, and stool culture and sensitivity, did not show any pathogenic organism. Ultrasonography of the abdomen and liver function tests were within normal limits. These findings were consistent with active ulcerative colitis and systemic inflammation.

After a detailed history, clinical examination, laboratory investigations, and histopathological examination, we diagnosed pyoderma gangrenosum associated with ulcerative colitis. Our patient was initially treated with injectable corticosteroids (dexamethasone 8 mg per day) for 5 days, after which they were switched to oral prednisolone at a dose of 40 mg per day. Oral corticosteroids were continued for 4 weeks, with tapering initiated thereafter. Mesalamine (1200 mg/day) and dapsone (100 mg/day) were also introduced early in the treatment to address underlying inflammatory pathways. For wound management, a topical antibiotic-steroid combination (fusidic acid with hydrocortisone) was used to promote healing and minimize secondary infection. The corticosteroid tapering is still ongoing, and the patient remains under regular follow-up to monitor disease progression and identify potential flare-ups.

## Discussion

Pyoderma gangrenosum is a rare, ulcerative, cutaneous condition. First described in 1930, the pathogenesis of pyoderma gangrenosum remains

unknown, but it is probably related to a hyperallergic reaction. There are various clinical and histological variants of this disorder. Pyoderma gangrenosum often occurs associated with a systemic disease such as inflammatory bowel disease, rheumatologic disease, paraproteinaemia, or haematological malignancy. The diagnosis, mainly based on the clinical presentation and course, is confirmed by eliminating other causes of cutaneous ulcers. The differential diagnosis of ulcerative cutaneous lesions includes infectious disease, malignancy, vasculitis, insect bites, venous or arterial insufficiency (comprising antiphospholipid antibody-associated occlusive disease), and, above all, factitious (self-inflicted) ulcerations. Local treatment may be sufficient for mild disease, while systemic immunosuppressants are the mainstay for severe cases. Long-term treatment with these agents is often required, but this can expose patients to adverse side effects.[4]

The ulcerative form of PG tends to occur predominantly on the lower extremities or the trunk. These ulcerative lesions may arise de novo or as a pathergic response to trauma. Malignant pyoderma, as described above, is considered a form of ulcerative PG. The typical patient is in the age range 25-55 years, in whom lesions manifest as ulcers with a peripheral inflammatory halo, most often on the lower extremities or the trunk; other areas of involvement include the vulva and penis, the head and neck region, the breast, and ocular site.[5]

Our patient's histopathological examination of a skin lesion biopsy showed neutrophilic exudate and a dense inflammatory cell infiltrate comprising lymphocytes and plasma cells. Perivascular neutrophilic infiltration, and extravasated RBCs and cell debris, were noted in places. Congested blood vessels and hemosiderin-laden macrophages were also seen (Figure 2).

Biopsies from the erythematous border or necrotic edge of the pyoderma gangrenosum lesions usually demonstrated a characteristic pathogenic morphologic evolution. The early lesions revealed mild to moderate perivascular lymphocytic infiltrate associated with endothelial swelling. The fully developed lesions demonstrated necrosis and a dense lymphocytic infiltration surrounding and involving the blood vessels. Extravasation of erythrocytes and thrombosis sometimes were seen. Ulceration, infarction, and abscess formation were found in the later stages of evolution. Pyoderma gangrenosum appears to be a reactive process that is manifested as vasculitis. Biopsy material from the advancing active erythematous border has early characteristic dermatopathologic findings of lymphocytic vasculitis. Cutaneous vascular immune deposits suggest the immune pathogenesis of an immune complex disease or a lymphocytotoxic reaction.[6]

Wound care is an integral part of the treatment. The main principles involve proper cleansing with sterile saline or antiseptic and dressing changes. The dressing should adhere to the wound bed and promote a moist environment that is not overly dry or wet. Moreover, it should be easy to remove to prevent trauma and subsequent pain. Surgical procedures and surgical wound management should be limited to avoid pathergy. Gentle mechanical and autolytic debridement is recommended. Compression therapy reduces localized inflammation and tissue swelling, directly and indirectly promotion better mobility, resulting in increased blood circulation.[7]

First-line treatment options with the best evidence are systemic corticosteroids, cyclosporine, and tumor necrosis factor alpha inhibitors. Topical corticosteroids, intralesional corticosteroids, and calcineurin inhibitors are often used as adjuncts and are well tolerated.

Other steroid-sparing therapies include dapsone, mycophenolate mofetil, intravenous immunoglobulin, and targeted biologic or small molecule inhibitors. Patients without associated comorbidities responded better to treatment and suffered fewer adverse events. Wound care and management of underlying disorders are also critical parts of care.[8]

Biologics have shown efficacy in treating PG, including tumor necrosis factor-α inhibitors such as infliximab, adalimumab, etanercept, certolizumab pegol, and golimumab. Interleukin-1 inhibitors include anakinra (blocking IL-1α and IL-1β), canakinumab (IL-1β-specific inhibitor), and gevokizumab (IL-1β-specific inhibitor). Interleukin-23 inhibitors, such as ustekinumab (targeting the common p40 subunit of IL-12 and IL-23), tildrakizumab, guselkumab, and risankizumab (targeting the p19 subunit of IL-23 without affecting IL-12), have also been explored. Sparse reports describe successful PG treatment using IL-17 inhibitors, including secukinumab (anti-IL-17A), brodalumab (anti-IL-17 receptor), and ixekizumab (anti-IL-17A/F).[9]

Our patient was treated with injectable dexamethasone (8 mg/day), mesalamine (1200 mg/day), and dapsone (100 mg/day). A topical fusidic acid-hydrocortisone combination was used for wound care. Injectable steroids were switched to oral prednisolone (40 mg/day), with tapering planned alongside the introduction of steroid-sparing agents. Dapsone will be continued, and close monitoring is essential to manage UC and prevent PG flare-ups. Regular follow-up visits will be necessary to monitor both conditions and ensure effective disease control. Continuous management of UC will be critical. The patient will be closely monitored for gastrointestinal symptoms, with regular assessments for new ulcerative lesions. Maintenance therapy with mesalamine will be key to sustaining remission. If disease activity increases, biologic therapies may be considered. Proper UC control reduces systemic inflammation and lowers the risk of

PG recurrence. Wound care will be vital for preventing infection and promoting healing. The patient will be educated on proper skin hygiene, avoiding trauma to the affected areas, and recognizing early signs of disease. Topical fusidic acid with hydrocortisone will help manage local inflammation and prevent bacterial infection. Preventive measures to avoid trauma and skin breakdown will be important in reducing the risk of PG flare-ups.

A collaborative approach involving dermatologists, gastroenterologists, and primary care physicians will be crucial for managing UC and PG. Coordination between specialists will ensure comprehensive care and allow for timely adjustments to therapy based on disease progression and response.

## Conclusion

This case highlights the intricate relationship between pyoderma gangrenosum and inflammatory bowel disease, particularly ulcerative colitis. The patient's history of UC and the characteristic histopathological findings strongly suggest an underlying immune-mediated process. Effective PG management requires systemic therapy and careful wound care, with corticosteroids playing a central role in controlling inflammation. Early recognition and treatment are crucial to prevent complications and improve the patient's quality of life. This report adds to the growing body of evidence supporting the association between PG and IBD and the necessity for a multidisciplinary approach to management.

## References


1. Head KA, Jurenka JS. Inflammatory bowel disease part 1: ulcerative colitis--pathophysiology and conventional and alternative treatment options. Altern Med Rev. 2003; 8(3): 247–83. PMID: 12946238
2. Thompson AI, Lees CW. Genetics of ulcerative colitis. Inflamm Bowel Dis. 2011; 17(3): 831–48. https://doi.org/10.1002/ibd.21375
3. Wollina U. Pyoderma gangrenosum--a review. Orphanet J Rare Dis. 2007; 2: 19. https://doi.org/10.1186/1750-1172-2-19
4. Ruocco E, Sangiuliano S, Gravina AG, Miranda A, Nicoletti G. Pyoderma gangrenosum: an updated review. J Eur Acad Dermatol Venereol. 2009; 23(9): 1008–17. https://doi.org/10.1111/j.1468-3083.2009.03199.x
5. Crowson AN, Mihm MC Jr, Magro C. Pyoderma gangrenosum: a review. J. Cutan. Pathol. 2003; 30(2): 97–107. https://doi.org/10.1034/j.1600-0560.2003.00024.x
6. Su WP, Sctiroeter AL, Perry HO, Powell FC. Histopathologic and immunopathologic study of pyoderma gangrenosum. J Cutan Pathol. 1986;13(5):323–30. https://doi.org/10.1111/j.1600-0560.1986.tb00466.x
7. Łyko M, Ryguła A, Kowalski M, Karska J, Jankowska-Konsur A. The Pathophysiology and Treatment of Pyoderma Gangrenosum-Current Options and New Perspectives. Int J Mol Sci. 2024; 25(4): 2440. https://doi.org/10.3390/ijms25042440
8. Tan MG, Tolkachjov SN. Treatment of Pyoderma Gangrenosum. Dermatol Clin. 2024; 42(2): 183–92. https://doi.org/10.1016/j.det.2023.12.002
9. Maronese CA, Pimentel MA, Li MM, Genovese G, Ortega-Loayza AG, Marzano AV. Pyoderma Gangrenosum: An Updated Literature Review on Established and Emerging Pharmacological Treatments. Am J Clin Dermatol. 2022; 23(5): 615–34. https://doi.org/10.1007/s40257-022-00699-8