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Case Report

Intersection of Diabetic Foot Infection and Pyoderma Gangrenosum: A Conflicting Treatment Approach

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Abstract

This article discusses the treatment course of an aggressive diabetic foot infection (DFI) complicated by the emergence of pyoderma gangrenosum (PG). A 67-year-old man with long-standing diabetes presented with a nonhealing DFI that prompted antibiotic treatment and surgical debridement. However, the coexistence of DFI and PG may create a conflict in the treatment approach, as treating one condition may exacerbate the other. The patient responded positively to systemic corticosteroids and infliximab, and, despite conflicting recommendations on debridement, surgical intervention proved necessary. This report advocates for early PG diagnosis using the Delphi model and stresses the need for ongoing research regarding surgical debridement in these complex scenarios.

Keywords: diabetic foot infection; diabetic foot ulcer; pyoderma gangrenosum; neutrophilic dermatoses

1. Introduction

We present the clinical course of a patient with an aggressive diabetic foot infection (DFI) intricately complicated by the emergence of pyoderma gangrenosum (PG), a rare inflammatory skin disorder. The coexistence of these pathologies is difficult to distinguish and treat, requiring a nuanced approach for accurate identification and tailored management, as patients suffering from PG may exhibit rapidly evolving and painful ulcers that could pose serious risks of limb loss and even death.

As a formidable healthcare challenge, DFI is associated with substantial morbidity [1]. While DFIs are often treated with antibiotics and surgical debridement, the rare emergence of PG within the context of a DFI adds an additional layer of complexity. Classified within the category of neutrophilic dermatoses, PG is a collection of inflammatory skin conditions characterized by abnormal neutrophilic infiltration. Patients with PG may go on to live most of their life not knowing they are affected by this rare disease until an exaggerated skin injury occurs from minor trauma that leads to a painful and rapidly progressing ulcer, described as a pathergy response. Any mechanical debridement may further exaggerate the wound.

The incidence rate of this autoimmune skin disorder is approximately 5.8 cases per 100,000 adults, affecting more women than men [2]. More commonly, PG is associated with inflammatory bowel disease (IBD), such as ulcerative colitis (UC). In most cases, treating UC may lead to improvement of the PG since the treatment involves systemic steroid or biological therapies. This case report's focal point is a patient who initially presented with a DFI with a worsening ulcer after treatment and was subsequently diagnosed with an acute flare of UC. The patient's clinical presentation intertwines the complexities of a DFI with the

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aggressiveness of PG, emphasizing the need for an early diagnostic and interdisciplinary approach to address this intricate convergence of pathologies.

2. Case Report

A 67-year-old man with a long-standing history of diabetes presented to the emergency department with a painful nonhealing ulcer on the posterior aspect of his left heel. A preceding trauma involving a dropped hammer on the forefoot led to swelling, prompting the patient to elevate his foot for a prolonged period of time on a hard wooden stool, inadvertently causing a pressure injury to the heel. Clinical examination revealed a large, ulcerative, fluctuant mass with purulent discharge, erythema, calor, edema, and ascending cellulitis. Radiograph and magnetic resonance imaging revealed an abscess without evidence of soft tissue gas or osteomyelitis. Broad-spectrum antibiotics were initiated, and the patient was taken to the operating room for source control.

Intraoperative cultures demonstrated a polymicrobial infection. Three days after the initial surgical debridement, the site showed extensive devitalized tissue and purulent drainage, which raised suspicion of continued infection and prompted further surgical debridement. After the second debridement, the wound appeared to be stable and clear of infection based on examination and repeat culture. The patient was discharged on outpatient antibiotics and underwent weekly follow-up for local wound care. Negative pressure wound therapy (NPWT) was initiated. Several weeks after the application of NPWT, new large areas of deep tissue injury occurred. There was erythema extending from the ulcer site, and the patient reported severe pain and diarrhea, which heightened the suspicion of an undiagnosed pathology superimposed with a diabetic foot infection.

The patient was readmitted and brought back to the operating room. An extensive amount of devitalized and necrotic tissue with purulent abscess and tunneling was discovered intraoperatively, with the Achilles tendon now fully exposed without necrosis (Figure 1). We performed a final debridement, and the infectious disease service initiated broad spectrum antibiotics. Concomitantly, a gastroenterologist was consulted, and a sigmoidoscopy was performed. The result showed severe UC. Clinical evaluation suggested that the wound was demonstrating a pathergy response that was most consistent with pyoderma gangrenosum superimposed with recurrent diabetic foot infection. Other potential causes of this atypical lesion were excluded. Wound biopsies were obtained and found negative for fungal, yeast, and human immunodeficiency virus infections. Anaerobic, Gram periodic acid–Schiff, and acid-fast bacillus stains showed no organisms. The T-spot was negative, and no malignant cells were seen. However, tissue biopsy exhibited dense inflammatory cell infiltration that was predominately neutrophilic (Figures 2–5). The diagnosis of pyoderma gangrenosum was confirmed.

At this point, the patient's UC was severe enough that he was becoming significantly malnourished, and a multidisciplinary meeting was held to discuss a potential colectomy and below-the-knee amputation surgery. However, with adequate source control and no further signs of infection, the team decided to attempt limb salvage with conservative treatment of the UC, consisting of prednisone and infliximab. After several weekly clinic follow-ups with conservative debridement and systemic therapy, the wound rapidly improved. At the most recent follow-up, granulation tissue had filled the defect, the wound was in the process of epithelializing, and the patient's UC was fully controlled (Figure 6).

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Figure 1. Post-surgical debridement of posterior left heel.

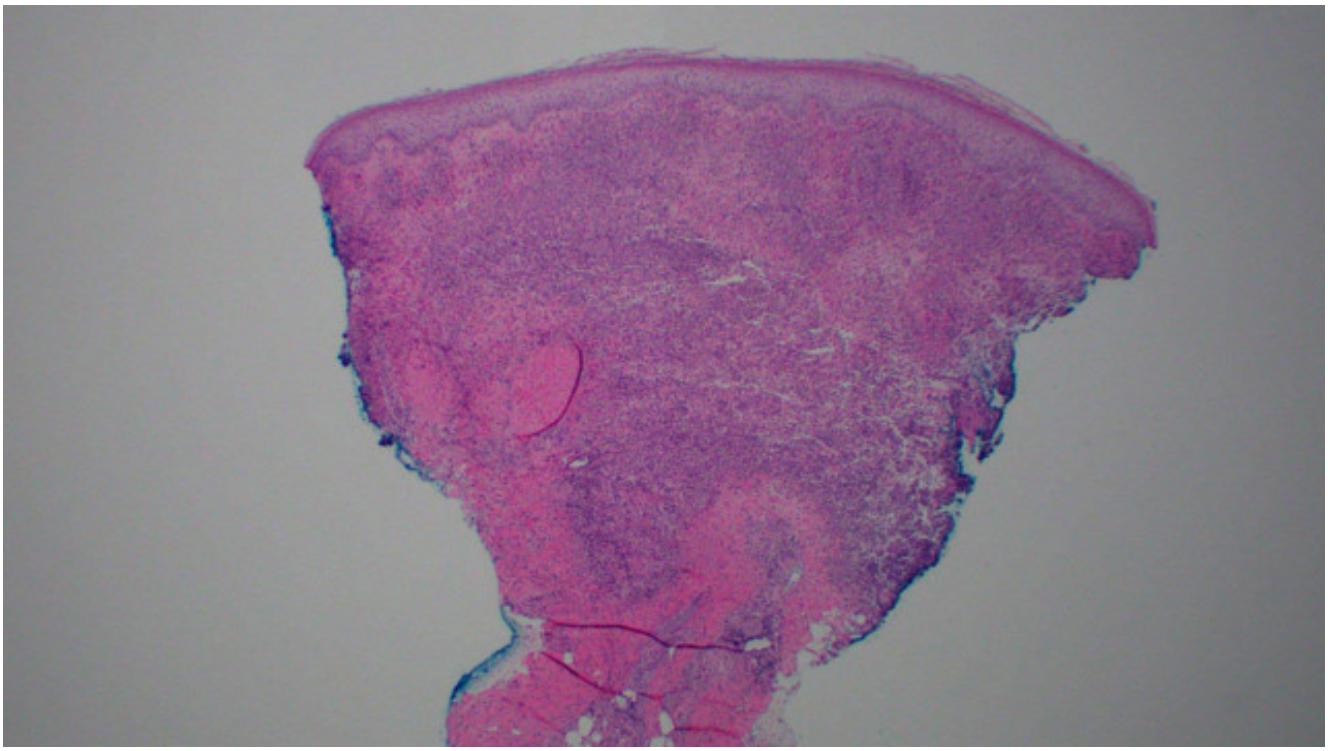


Figure 2. Low-power view showing a dense infiltrate of inflammatory cells within the superficial and deep dermis. The overlying epidermis shows no significant pathologic changes.

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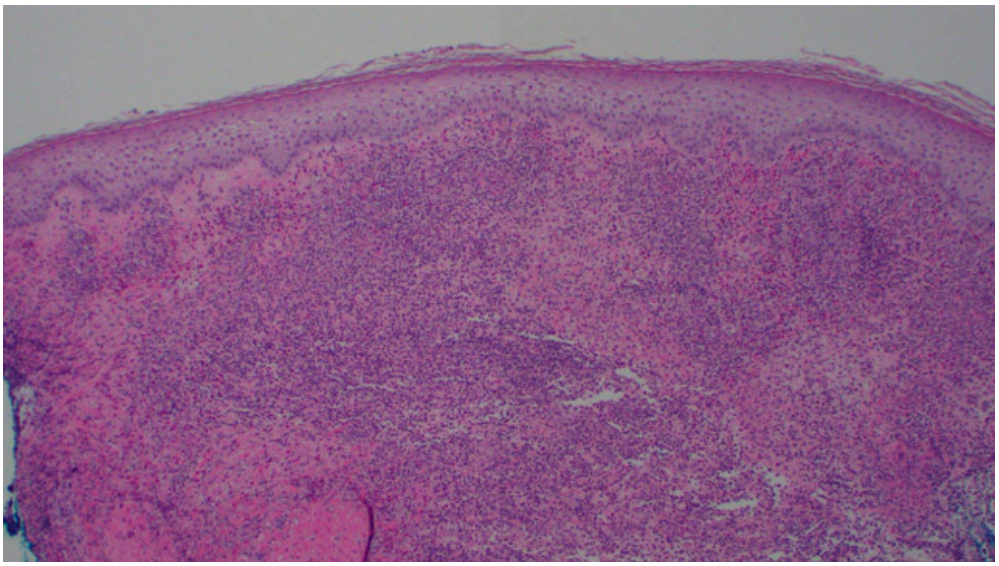


Figure 3. Intermediate-power view showing a dense, mixed inflammatory cell infiltrate.

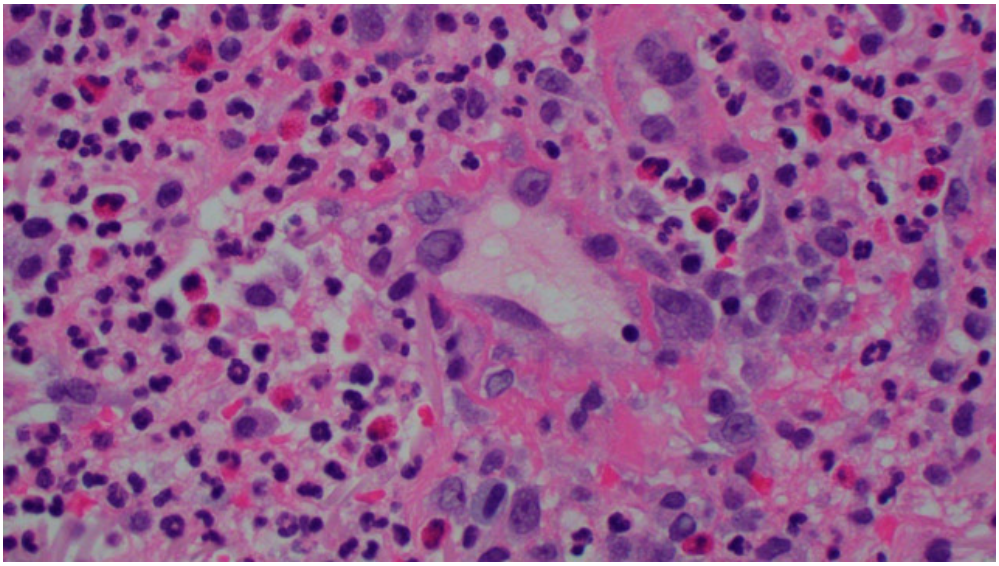


Figure 4. High-power view showing neutrophils.

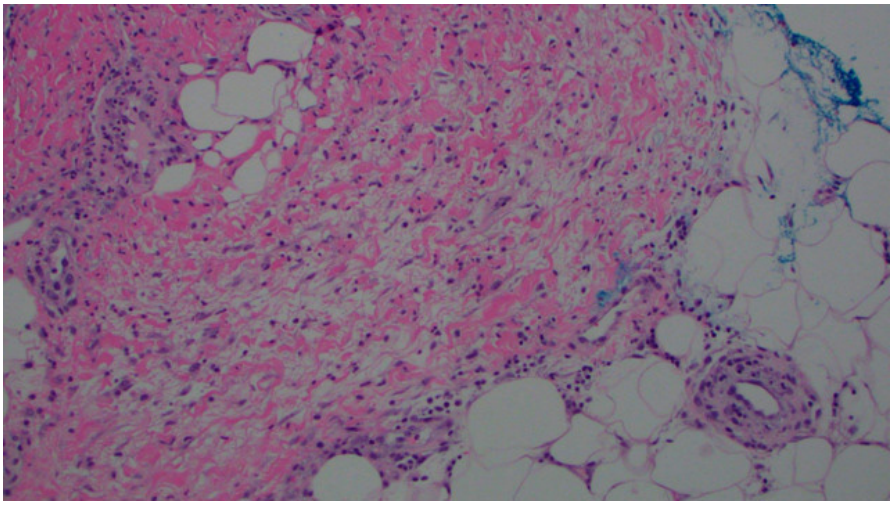


Figure 5. Inflammatory cells.



Figure 6. Clinical follow-up.

3. Discussion

As a rare inflammatory skin disorder categorized as neutrophilic dermatoses, PG has subtypes including pustular, bullous, vegetative, and the predominant ulcerative form, which commonly affects the lower extremities [3]. Since there is no definite answers as to what causes PG or to which treatment has a higher efficacy in managing these progressive lesions, PG is a challenging disorder to treat.

Various theories propose dysfunction, and the genetic mutation of neutrophil infiltration involving innate immune cells remains the culprit of this etiology; but, the pathophysiology remains elusive. One hypothesis involves the T cells and Th-17 cells of the adaptive immune system targeting the skin tissue's follicular units, indirectly activating neutrophils [4]. The same study identified active PG lesions as containing interleukin-8 and tumor necrosis factor-alpha in the wound bed, acting chemoattractants for neutrophils [4].

Historically, PG diagnosis relied on exclusion criteria, but recent advancements propose a diagnostic model involving major and minor criteria, as determined through the Delphi model [5]. The major criterion involves tissue biopsy confirming dense neutrophilic infiltration. The study also expresses the importance of obtaining a tissue biopsy from the ulcer edge, as it is superior to other ulcer sites. As with the patient in this case report or any other patient with suspected PG, tissue biopsy should reveal the dense infiltration of neutrophils. The minor criteria of the Delphi model involve the clinical assessment of the ulceration, as it is an important factor for diagnosing PG (Table 1): the lesion is extremely painful, can progressively increase in size or cause damage, appears as a necrotic lesion with a violaceous border and undermining edges, can be pretibial, and is associated with pathergy (Figure 7). Meeting four of the eight minor criteria in addition to having positive neutrophilic infiltration on tissue biopsy can have a sensitivity rate of 86% and specificity rate of 90% in diagnosing PG. While the Delphi model lists confirmed IBS as

a minor criterion, it is important to note that numerous patients with PG lesions are also confirmed to have one type of IBS, commonly UC. The patient in this case report met the Delphi criteria.

Table 1. Delphi model.

Delphi Criteria	
Major Criterion	Only 1 required
• Biopsy of ulcer edge demonstrating neutrophilic infiltrate	
Minor Criteria	Need a minimum of 4 to be met
• Exclusion of infection on histology • Pathergy • History of inflammatory bowel disease or inflammatory arthritis • History of papule, pustule, or vesicle that rapidly ulcerates • Peripheral erythema, undermining border, and tenderness at ulceration site • Multiple ulceration (at least 1 on an anterior lower leg) • Cribriform or wrinkled paper scars at healed ulcer sites • Decreased ulcer size after immunosuppressive treatment	

At present, there is no universally accepted standard for treating PG lesions. However, the recommended approach involves systemic therapy, aiming to address both the underlying symptoms of IBD and the associated skin lesions. In a systematic review, Partridge et al. [6] found that treatment of PG in individuals with IBD varies in effectiveness based on the different interventions and found a nearly 100% healing rate with biological agents such as infliximab and adalimumab for patients with both PG and IBD. In contrast, the commonly used systemic corticosteroids appear to have lower efficacy, with healing rates ranging from 33% to 50%. However, Partridge et al. noted the timing and dosage of corticosteroids impact their effectiveness, with higher doses and early initiation leading to faster healing times. In another literature review article, Maronese et al. [7] found that TNF-a inhibitors, such as infliximab and adalimumab, have shown efficacy as first-line treatments. Our patient was treated with corticosteroids early and treated with infliximab afterwards.

Regarding the physical wound management of PG lesions, this rare abnormal inflammatory skin disorder typically affects the outside layer of the skin and presents without infection. Additionally, any sort of sharp debridement may elicit a pathergy response. The convergence of a rapid DFI with PG required a personalized treatment strategy. In a case report by Lee et al. [8], the authors discussed PG mimicking a DFI and the complication of misdiagnosis. Our patient presented a similar unique clinical scenario involving a nonhealing diabetic ulcer. The treatment for DFI consists of antibiotics, surgical incision and drainage, and debridement, or all of the aforementioned. The challenge arose when the clinical course suggested PG, making debridement a nonviable option for this type of ulcer. However, a recent literature review supported this unconventional approach to the management of PG lesions. Taheri and colleagues discussed two types of debridement: aggressive and conservative. Aggressive debridement involves removing part of the healthy tissue to retrigger the inflammatory process of wound healing. Thus, it triggers abnormal inflammatory cells, leading the healthcare provider to become hesitant to debride PG lesion out of concern of triggering a pathergy response. Conservative debridement consists of removing the slough and necrotic tissue in the wound bed, as this will help control inflammation. Taheri et al.'s review consisted of 38 case reports and case series on sharp surgical debridement of PG lesions, and it found aggressive debridement can worsen PG lesions, but it provided no data on conservative debridement [9].



Figure 7. Violaceous border with undermining edges and cribriform wound base.

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Our patient had extensive abscesses invading fascial planes that prompted a deviation from the aggressive debridement approach. The patient's PG lesions were rapidly infected, requiring conservative debridement to eradicate the necrotic tissue for source control, and the operative findings of multiple sinus tracts into the deep fascia affirmed the team's decision. The decision to conservatively debride the necrotic and infected tissue was aimed at preventing bacteremia, and the overall clinical benefit was deemed necessary.

Another treatment for a PG lesion would be NPWT. A PG lesion is typically confirmed after several attempts at debridement, and, by that time, huge tissue defects are present, making the use of NPWT a viable option. While a systematic review demonstrated improvement in PG lesions with the use of NPWT [10], the patient in this case report did not respond well to this modality due to the pathergy response. There were positive outcomes such as faster wound bed preparation, granulation tissue improvement, and reduction in wound size when NPWT was applied before starting immunosuppressive therapy. This demonstrates that NPWT alone may not be curative and stresses the need for the immunomodulation or control of the inflammatory response. It would be worth researching the benefits of using NPWT with and without immunosuppression treatment before or during the treatment course.

In isolation, PG and DFI can pose a serious threat to a vulnerable patient, but the coexistence of these pathologies together can create conflicts in the treatment approach, as treating for one can further exacerbate the other condition. The management of such cases requires astute clinical acumen, ongoing collaboration between specialists, and a personalized treatment strategy tailored to unique challenges. With our case report, we aim to share an experience of these two pathologies coexisting and hope to provide insight for further studies to create a treatment strategy.

4. Conclusions

The use of the Delphi model is vital in the early diagnosis of PG in patients with DFI. Obtaining a thorough medical history is also important, as conditions such as IBD show a strong correlation with PG. Our case report highlights the need for ongoing research and a deeper understanding of the interplay between DFI and PG, as one of the pathologies requires surgical debridement, which is contraindicated in the other. The treatment in our case report deviatedf rom the traditional method.

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Informed Consent Statement: Verbal informed consent was obtained from the patient for publication of this case report and the accompanying clinical images. All identifying information has been removed to ensure patient confidentiality.

Data Availability Statement: The original contributions presented in this study are included in this article. Further inquiries can be directed to the corresponding author(s).

Conflicts of Interest: The authors declare no conflicts of interest.

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