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Case Report

A Challenging Case of Superficial Granulomatous Pyoderma in a 91-Year-Old Chinese Female

Simal A. Qureshi [1,*], Muhammad N. Mahmood [2] and Eunice Y. Chow [3]

[1] Faculty of Medicine, Memorial University of Newfoundland, St. John's, NL A1B 3X5, Canada
[2] Department of Laboratory Medicine and Pathology, University of Alberta Hospital, Edmonton, AB T6G 2B7, Canada
[3] Division of Dermatology, Department of Medicine, University of Alberta, Edmonton, AB T6G 2G3, Canada
* Correspondence: sq2152@mun.ca

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Abstract: Superficial granulomatous pyoderma (SGP), also known as vegetative pyoderma gangrenosum, is a rare, localized variant of pyoderma gangrenosum (PG). It typically presents as slowly progressive, superficial ulcerations with a vegetative border and clean base, most often on the trunk. Unlike classic PG, SGP is usually not associated with systemic disease, follows an indolent course, and may respond to topical or conservative systemic therapy. Histopathology can be variable; reported features include a zoned neutrophilic infiltrate in the upper dermis and occasional sinus tract formation. We report a diagnostically challenging case of SGP in a 91-year-old Chinese woman who presented with a three-year history of pruritic, superficial ulcerated plaques. Multiple biopsies and cultures initially yielded nonspecific results, with findings obscured by prior topical treatment and colonization of chronic wounds. A definitive diagnosis was achieved only after biopsy of a new, untreated lesion, which demonstrated ulceration with dermal neutrophilic infiltrate. The patient subsequently improved on topical clobetasol and low-dose doxycycline monohydrate. This case highlights the importance of clinicopathologic correlation, the value of obtaining biopsies from fresh, untreated lesions, and the potential role of conservative therapies in elderly patients with SGP.

Keywords: superficial pyoderma gangrenosum; superficial granulomatous pyoderma; topical corticosteroid; clobetasol; doxycycline monohydrate

1. Introduction

Superficial granulomatous pyoderma (SGP), also known as vegetative pyoderma gangrenosum, is an uncommon and localized form of pyoderma gangrenosum (PG) [1,2]. It typically presents as chronic, superficial ulcerations on the trunk, hip, or abdomen, manifesting as papules, nodules, or plaques that evolve into shallow ulcerations with a clean base and vegetative border [1,3]. Similar to classic PG, SGP may demonstrate pathergy [4]. In contrast to classic PG, SGP usually spares the lower extremities, lacks undermined borders, progresses slowly, is less painful, and is rarely associated with systemic diseases [2,5]. Negative microbiologic cultures are required to exclude infectious mimickers [1]. Histopathology can be variable but may show a three-layered inflammatory pattern formed by central neutrophils, a middle layer of giant cells and histiocytes, and an outer rim of plasma cells and eosinophils, with or without sinus tracts, and pseudoepitheliomatous hyperplasia [4,6]. Treatment is often successful with topical therapies such as corticosteroids or tacrolimus, or with intralesional steroid injections [7,8]. Some cases require systemic treatment, including corticosteroids, doxycycline, minocycline, dapsone, or sulfonamides [7–9].

Herein, we report a diagnostically challenging case of SGP in a 91-year-old Chinese woman whose ultimate diagnosis required multiple skin biopsies. Biopsy of a new, untreated lesion revealed dense dermal neutrophilic infiltrate, and the patient subsequently responded well to topical clobetasol and oral doxycycline monohydrate.

2. Case Report

A 91-year-old Chinese woman with Fitzpatrick Skin Type III presents with a three-year history of pruritic, enlarging plaques on the right gluteal region. Examination revealed roughly three-to-five-centimeter discoid erythematous plaques with central superficial ulceration and yellow fibrinous debris with slightly scaly edges (Figure 1). On the left lower back, three smaller eroded macules were noted. No other lesions were present aside from a large biopsy-proven lipoma in the right gluteal region. She denied systemic symptoms.

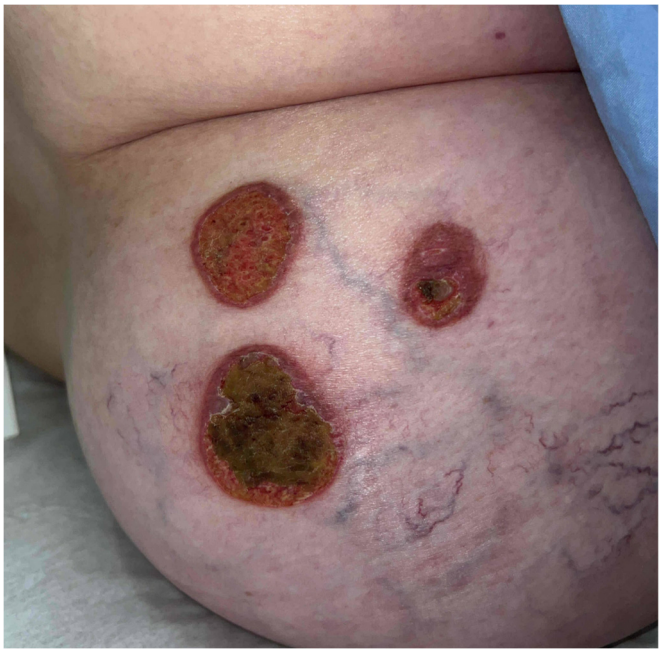


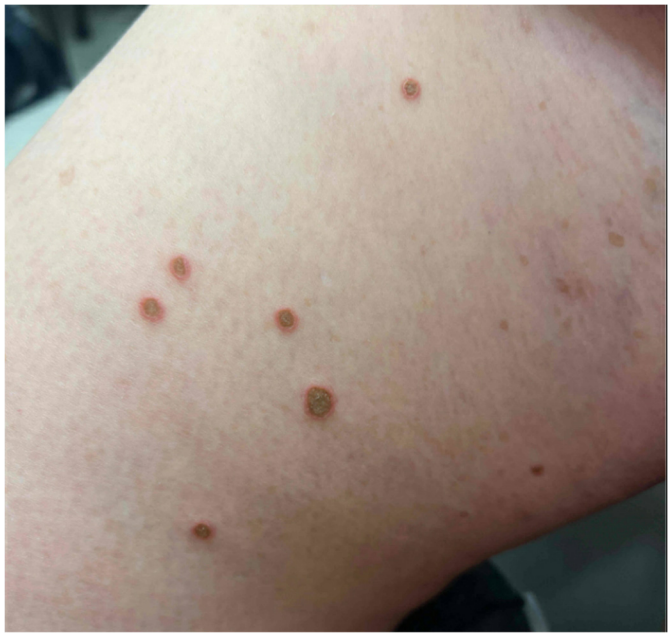
Figure 1. Well-demarcated, discoid, eroded erythematous plaques on the right gluteal region.

Her medical history included hypertension, gastroesophageal reflux disease, constipation, vertigo, and cataracts. Her medications included betahistine, calcium/vitamin D, diclofenac gel, domperidone, hydrochlorothiazide/valsartan, lansoprazole, multivitamin, nifedipine, and simvastatin. Past therapies for the lesions included liquid nitrogen cryotherapy, mometasone 0.1% cream, and betamethasone dipropionate 0.05%/clotrimazole 1% cream, none of which reduced lesion size.

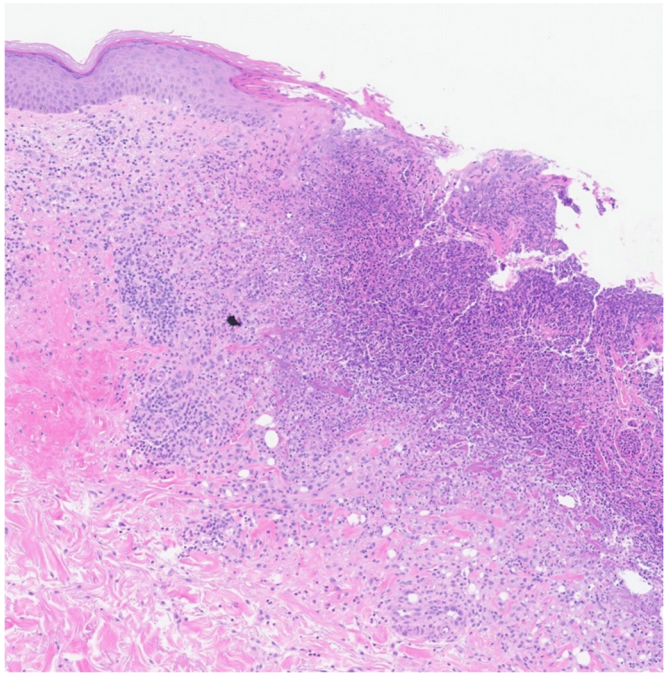
The first punch biopsy from the edge of the right gluteal plaque revealed cutaneous ulceration with dermal lymphoplasmacytic infiltrate. No neutrophilic or granulomatous infiltrate was present. Direct immunofluorescence studies on perilesional skin were negative. Bacterial culture via swab was positive for lactose-fermenting gram-negative bacilli, which was deemed colonization. Herpes simplex virus, varicella zoster virus, and syphilis testing were negative. The working diagnosis was excoriated nummular dermatitis, and she was treated with mupirocin and fluocinonide 0.05% ointment, twice daily. One month later, one lesion had resolved, but others persisted. Home nursing wound care was arranged. Repeat biopsies from smaller macules showed superficial perivascular lymphocytic infiltrate with eosinophils. No ulceration, neutrophilic infiltrate, or granulomas were present in this biopsy. The histopathologic differential diagnosis included arthropod assault, drug eruption, papular dermatitis, and chronic excoriated spongiotic dermatitis. T-cell receptor gene rearrangement testing was negative for monoclonality. Tissue culture grew mixed anaerobes, considered colonizers. The patient reported mild improvement with the addition of wound care dressings (silicone foam dressing); however, lesions persisted, and new ones appeared.

Over subsequent months, older plaques partially re-epithelialized, but new papules and erosions developed on the thighs and abdomen. Phototherapy and systemic immunosuppressants were discussed but declined due to her age and logistical issues. A biopsy from a new, untreated thigh lesion (Figure 2A) revealed a cutaneous ulceration with dense neutrophilic infiltrate consistent with pyoderma gangrenosum (Figure 2B). Although neutrophilic infiltrate was present in the upper dermis, underlying zoned granulomatous inflammation was not

present. Tissue cultures and mycobacterial cultures were negative aside from Gordonia species, deemed a contaminant (after Infectious Disease consultation). With clinicopathologic correlation, the diagnosis of SGP was made.



(**A**)



(**B**)

Figure 2. (**A**) Clinical: New, untreated lesions from the thigh. (**B**) Histopathology: Punch biopsy of skin from the thigh showing cutaneous ulceration with dense neutrophilic infiltration (H & E, ×50).

The patient was started on clobetasol 0.05% ointment twice daily, five days per week. To reduce new lesion formation, oral doxycycline hyclate 100 mg twice daily was initiated but switched to doxycycline monohydrate 40 mg once daily due to gastrointestinal intolerance. This regimen was well-tolerated, alleviated pruritus, and prevented new lesions.

At the one-month follow-up visit after initiating the clobetasol and doxycycline monohydrate, significant improvement was observed (Figure 3). Doxycycline was continued for a total of three months. Tacrolimus 0.1% ointment was added as a steroid-sparing agent. Malignancy workup was unremarkable. At the seven-month follow-up, five months after discontinuing doxycycline, no recurrences were observed.

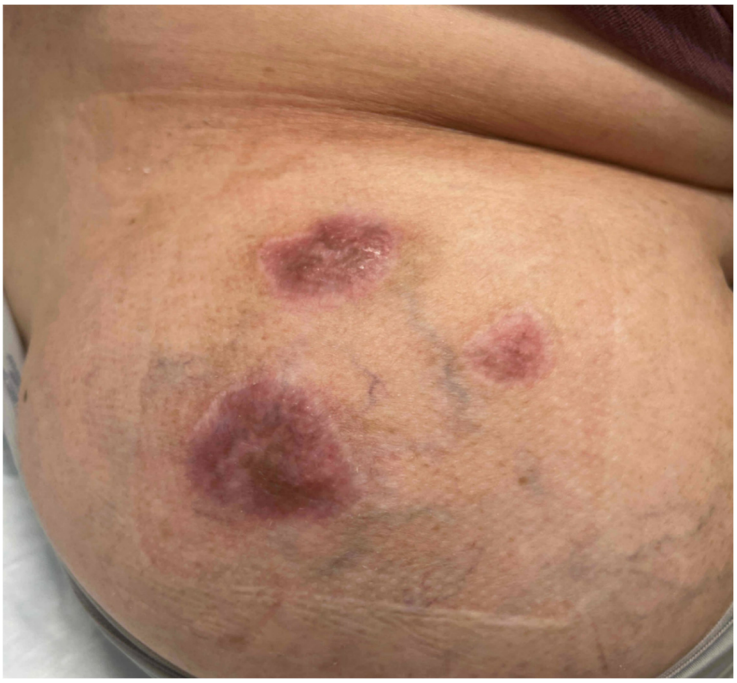


Figure 3. Resolving annular hyperpigmented and slightly purpuric patches.

3. Discussion

This case illustrates the diagnostic challenges of SGP. Unlike classic PG, SGP presents as superficial, relatively painless lesions without systemic associations. Repeated non-specific biopsies and polymicrobial cultures delayed diagnosis, with earlier findings potentially influenced by chronicity, prior treatment, and colonization. While topical corticosteroids are often thought to obscure neutrophilic infiltrates [10], direct evidence is limited. Differences in lesion chronicity likely play a larger role: acute, untreated lesions typically display prominent neutrophilic infiltrates, whereas chronic or partially treated lesions may show attenuated or mixed inflammatory patterns [11]. In addition, percutaneous absorption of corticosteroids increases when the epidermal barrier is disrupted or inflamed [12]. In eroded or ulcerated skin, this may result in high local concentrations and deeper penetration of corticosteroids, potentially exerting a more pronounced effect on neutrophils. Secondary changes, such as fibrosis or colonization by microorganisms, can further complicate interpretation. In our patient, this distinction proved diagnostically decisive: earlier biopsies from partially treated or older lesions were nonspecific, whereas targeted biopsy of a new, untreated lesion revealed a dense neutrophilic infiltrate and allowed definitive diagnosis. This underscores a key clinical principle: when SGP is suspected, biopsy should be directed toward early, clinically active, and previously untreated lesions to maximize diagnostic yield.

The initial lesions overlying a large lipoma may reflect pathergy, defined as an exaggerated neutrophilic response to local trauma [13]. The chronic friction or pressure from sitting may have contributed to lesion formation.

It is important to recognize that, unlike classic PG, SGP does not have universally accepted diagnostic criteria [2]. However, Su et al. proposed a diagnostic criteria for “vegetative PG” where the diagnosis requires both major criteria of “chronic erythematous plaques with sinus tract formation, shallow ulcerations or erosions and discomfort” and exclusion of other causes, and at least two of the three minor criteria of “histopathology compatible [dermal and histiocytic dermal infiltrate, granuloma formation]”, “no associated disease”, or “response to minor treatment measures” [2]. This proposed diagnostic criteria for SGP highlights that SGP lacks the more commonly recognized signs and symptoms of classic ulcerative PG, such as the association with systemic diseases, undermined border of the ulcer, tenderness, and cribriform scarring, making the clinical diagnosis of SGP difficult [2,6,14,15]. Pain out of proportion to the size of the lesion is a well-known characteristic feature of classic ulcerative PG, but it is not typical for SGP, wherein the proposed diagnostic criteria only include “discomfort” within the lesions of SGP [2]. In this patient, she met the two major criteria (chronic erythematous plaques with shallow ulcerations or erosions, other causes excluded) and two of the three minor criteria (no associated disease, response to minor treatment measures). The histopathologic finding of neutrophilic infiltrate is also supportive of this diagnosis.

There are no pathognomonic histopathologic features of SGP; however, there are several typical findings reported, including a three-layered granuloma consisting of central neutrophilic infiltrate, a middle layer of giant cells and histiocytes, an outer layer of plasma cells and eosinophils, and possible sinus tract formation, in addition to pseudoepitheliomatous hyperplasia [4,6]. It is important to note that some reported cases lack such a typical

pattern. For instance, Kumar et al. reported a case of SGP in which the patient's skin histopathology did not show the characteristic three-layer granulomatous pattern, but instead the diagnosis was confirmed by the presence of a neutrophilic infiltrate and few scattered multinucleate giant cells [16]. Shavit et al. also reported a case series focused on five patients with SGP involving the face [17]. They reported variable histopathological findings, including some cases exhibiting the classic zoned pattern while others lacked such a pattern, instead displaying mixed or neutrophilic dermal inflammation [17]. The authors have emphasized that SGP is a clinicopathological diagnosis [17]. Similar to classic PG, to effectively diagnose SGP, one must exclude other possible causes such as infections, autoimmune or autoinflammatory conditions, granulomatous diseases, and cutaneous neoplasms, and then correlate clinical presentation and histopathological findings to come to the correct diagnosis [9].

Treatment of SGP is individualized. While systemic corticosteroids are effective, elderly patients may benefit from conservative approaches. Various studies have reported success with topicals such as clobetasol or tacrolimus. For instance, a prospective cohort study by Thomas et al. evaluated topical therapy for PG in 66 patients and found that clobetasol propionate 0.05% (vehicle not specified) resulted in ulcer healing in 43% of patients by six months [18]. The study supports topical corticosteroids as an effective first-line therapy for PG, potentially avoiding the side effects systemic therapy may produce. Additionally, Grech et al. published a case of a 68-year-old woman with a three-year history of multiple, slow-growing, superficial ulcers on her arms, upper back, and abdomen, with histopathology in keeping with SGP [3]. Although the patient was treated with intravenous immunoglobulin for persistent lesions, new lesions responded well to oral prednisolone and topical tacrolimus ointment [3]. The patient was maintained successfully on doxycycline 100 mg daily and topical tacrolimus [3].

Doxycycline has anti-chemotactic effects on neutrophils, making it useful in the treatment of neutrophilic dermatoses [19]. Our patient's lesions responded to topical clobetasol, and the initiation of low-dose doxycycline monohydrate prevented new lesions from forming. To our knowledge, this may be among the first reports of successful management of SGP with low-dose doxycycline monohydrate (40 mg/day) combined with topical clobetasol.

4. Conclusions

SGP is a rare variant of PG that poses significant diagnostic challenges due to its atypical clinical presentation, variable histopathology, and potential mimicry of infectious or neoplastic processes. A critical insight from this case is that biopsy timing and site selection are essential: earlier biopsies from chronic or partially treated lesions yielded non-specific findings, whereas biopsy of a fresh, untreated lesion revealed the diagnostic neutrophilic infiltrate. This case reinforces that SGP is fundamentally a clinicopathological diagnosis. Histopathological findings must be interpreted alongside the clinical context, including lesion morphology, chronicity, and prior treatment, to avoid diagnostic error. Finally, this case supports the utility of conservative treatment strategies, particularly topical corticosteroids and low-dose doxycycline, in elderly patients for whom systemic immunosuppression carries meaningful risk. Individualized, stepwise management can achieve disease control while minimizing treatment burden in this population.

Author Contributions

E.Y.C.: conceptualization, investigation, patient management, patient consent acquisition, supervision, clinical photography, image description, writing, review and editing. S.A.Q.: writing, original draft preparation, literature review, writing, review and editing, submission coordination. M.N.M.: dermatopathology investigation, histopathological analysis, pathology reporting, histopathology image acquisition, histopathology image description, writing of pathology description, writing, review and editing. All authors have read and agreed to the published version of the manuscript.

Institutional Review Board Statement

IRB approval is not applicable—this is a single case report and ethics approval was not required for this case report according to institutional policy. All efforts were made to protect patient confidentiality.

Informed Consent Statement

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Data Availability Statement

All relevant data supporting the findings of this case report are included within the article.

Conflicts of Interest

The authors declare no conflict of interest.

Use of AI and AI-Assisted Technologies

No AI tools were utilized for this paper.

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