

Superficial Granulomatous Pyoderma

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We report the case of a 30-year-old man who, from the age of 16, presented with abscess-type lesions that turned into ulcers with a torpid course and with histopathologic characteristics of superficial granulomatous pyoderma. The patient has been followed for 6 years, during which time the only treatment necessary to prevent recurrence of lesions has been the administration of different doses of minocycline.

Several clinical variants of pyoderma gangrenosum have been described, including ulcerative, pustular, bullous, and vegetative.¹ This last variant, characterized by its limited, nonaggressive, chronic course and a superficial vegetative clinical appearance, was termed *superficial granulomatous pyoderma* (SGP).²

Case Report

A 30-year-old man was admitted to the gastroenterology department 9 years ago with a duodenal ulcer. We were consulted during his admission because, from the age of 16, he had experienced outbreaks of superficial ulcerated lesions with purplish borders and a torpid course. Since that time, he had never been completely free from lesions, with 1 to 5 lesions being present at all times. They were located most commonly on his back and buttocks.

On admission, the patient had 2 ulcerated lesions on his back, both of which had clean bases and slightly erythematous borders (Figure 1). A year earlier, he had been operated on for phimosis and, since then, a torpid ulcer had appeared, affecting practically all the balanopreputial sulcus. The ulcer had a clean base and an erythematous-edematous border with a vegetative appearance (Figure 2).

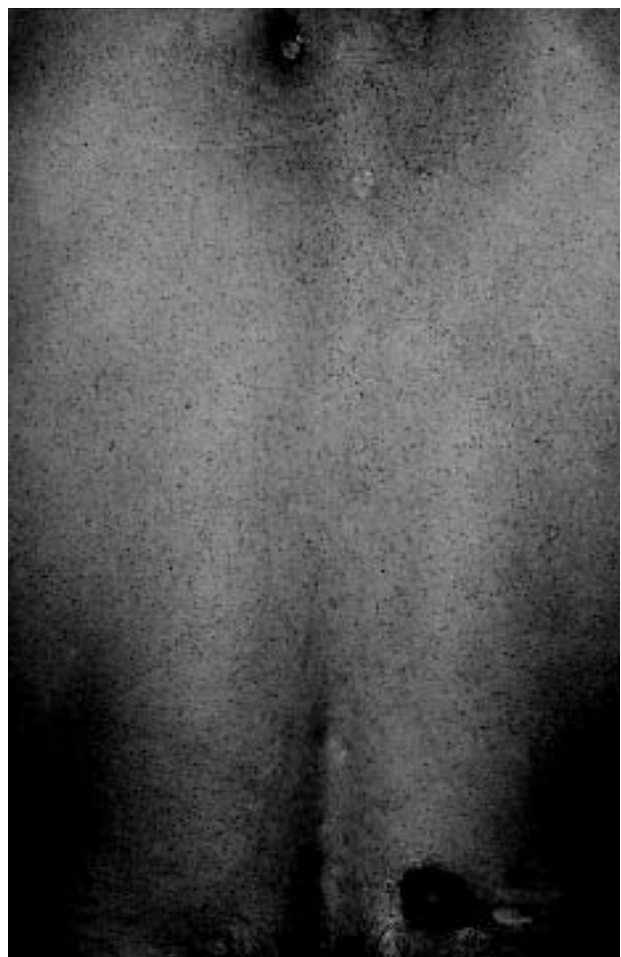


FIGURE 1. Chronic ulcers on the back.

Skin biopsy specimens were taken from the ulcers of both the balanopreputial sulcus and the back. Findings were similar in both cases; in the biopsy from the back, there was acanthosis and pseudoepitheliomatous hyperplasia of the epidermis with sinus tract formation (Figure 3). In the middle and papillary dermis, a neutrophilic abscess, surrounded by a mixed granulomatous infiltrate consisting of numerous plasma

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cells, histiocytes, lymphocytes, and some multinucleated giant cells and eosinophils, was observed. Sinus tract formation was seen near the adjacent adnexal epithelium (Figure 4). No fungal elements were seen on periodic acid-Schiff stain. Cultures of tissue for bacteria, fungi, and mycobacteria gave negative results.

The family and personal histories of the patient were uneventful. General medical examination was normal. The following investigations revealed negative or normal findings: complete blood cell count, erythrocyte sedimentation rate, serum electrolytes, liver function test, creatinine, cholesterol, triglycerides, urinalysis, fluorescent treponemal antibody absorption, immunoglobulins, serum protein electrophoresis, hepatitis B and C virus serology, antinuclear antibody, rheumatoid factor, chest roentgenography, and colonic and upper gastrointestinal x-ray studies with use of a contrast agent.



FIGURE 2. Ulcer of the glans penis.

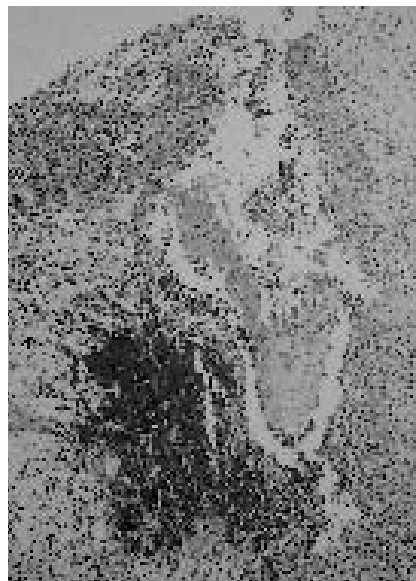


FIGURE 3. Biopsy specimen shows sinus tract formation surrounded by granulation tissue and histiocytes (H&E, original magnification $\times 100$).

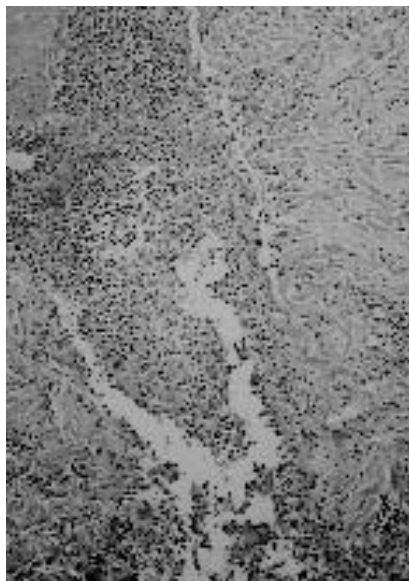


FIGURE 4. Initial stage of formation of sinus tract in the proximity of adnexal epithelium (H&E, original magnification $\times 200$).

The patient was treated initially with oral minocycline (100 mg twice daily) and topical corticosteroids, but the lesion in the glans did not clear up completely. Because improvement was slow and the patient requested prompt healing, oral corticosteroids (40 mg/day) were also administered. The dose was gradually tapered down and discontinued in 4 months. With this treatment the lesions healed up completely.

During a 6-year follow-up, the patient had been treated only with doses of minocycline ranging from 100 to 200 mg/day to 100 mg every third day. The maximum period without treatment and without lesions was 40 days. For the last year, the patient had been taking 100 mg of minocycline every third day and no lesions have been observed.

The only adverse effect from chronic treatment with minocycline was a slight increase in the transaminases (glutamic-oxaloacetic transaminase, 56 [normal, up to 39] and glutamic-pyruvic transaminase, 83 [normal, up to 35]) 2 years after commencing treatment. For this reason, he was referred to the gastroenterology department where a hepatic biopsy produced normal results. Hepatitis B and C markers were negative. The transaminase levels returned to normal after 4 months without having to suspend treatment with minocycline.

Comments

In 1988, Wilson-Jones and Winkelmann² reported 25 patients who had superficial ulcerative and vegetative pyoderma with granulomatous histologic findings. This type of pyoderma gangrenosum (PG) was SGP. To date, several cases have been reported.³⁻¹⁰

Various characteristics distinguish PG from SGP (Table I). The lesions of this chronic and superficial form of PG appear most commonly on the trunk and usually at sites of previous surgical treatment or other pathergic stimuli. Clinically, the ulcers show a relatively clean base and vegetative borders.

Histopathologic findings are not typical of ordinary PG. SGP is characterized by a 3-layer inflammatory reaction^{2,9,11}: a central abscess; a middle layer of histiocytes and giant cells; and an outer shell of a mixed inflammatory infiltrate with lymphocytes, numerous plasma cells, neutrophils, and

Table I.

Differences Between Pyoderma Gangrenosum and Superficial Granulomatous Pyoderma

Features	Pyoderma Gangrenosum	Superficial Granulomatous Pyoderma
Course	Acute	Chronic
Location	Extremities	Trunk
Pathergy	Yes	Yes
Associated diseases	Yes	No
Histologic findings	Deep neutrophils	Superficial granulomatous
Treatment	Anti-inflammatory agents Systemic corticosteroids	Anti-inflammatory agents Systemic corticosteroids

eosinophils. Acanthosis and pseudoepitheliomatous hyperplasia of the epidermis may be present. An interesting feature of this case, seen on biopsy, was the formation of sinus tracts near the adnexal epithelium.

The clinical and histologic appearance suggests the diagnosis of infectious and noninfectious granulomatous skin diseases (eg, deep fungus, atypical mycobacterial infection, blastomycosis, tuberculosis verrucosa cutis, amebic abscess, *Serratia granuloma*, halogenoderma). However, the major differential diagnosis is with classic PG.

Generally, SGP is not associated with other underlying systemic diseases (eg, chronic ulcerative colitis, Crohn's disease, rheumatoid arthritis, ankylosing spondylitis, chronic active hepatitis, monoclonal gammopathies, myeloma, blood dyscrasias, sarcoidosis), which are seen in approximately 50% of cases of classic PG.¹

It is important for physicians to recognize this variant of PG because it has a better prognosis, a benign course, and responds to more conservative treatments, such as intralesional corticosteroids, antibiotics (tetracycline, minocycline, sulfapyridine), and anti-inflammatory agents (dapsone). Systemic corticosteroids are usually not necessary. Also, an accurate diagnosis of SGP is essential because surgical intervention may lead to new lesions, as happened in our patient.

Our case is noteworthy in that the patient has been followed for more than 6 years, during which time he has remained free of lesions and has required only variable doses of minocycline. This demonstrates both the benign chronic course of this superficial variant of PG and how well it can respond to nonaggressive treatment.

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