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A Case of Superficial Granulomatous Pyoderma Mimicking a Basal Cell Carcinoma

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SUMMARY Pyoderma gangrenosum (PG) is a rare neutrophilic dermatosis of unknown etiology with distinct clinical manifestations, frequently associated with systemic diseases. Four clinical and histological variants have been described: ulcerative, pustular, bullous, and vegetative. We report on a case of superficial granulomatous pyoderma (SGP), a vegetative form of PG, in a 40-year-old woman. Physical examination revealed an erythematous crusted plaque, measuring 2 cm in diameter, located on her left hip, which had appeared 18 months ago. Dermoscopy showed lack of pigment network, large gray-blue ovoid nests, irregular peripheral vessels, and ulceration. Laboratory examinations were normal; smears and cultures for bacteria and fungi were negative. Clinical and dermatoscopical presentation suggested basal cell carcinoma. The lesion was completely removed: histological examination showed pseudoepitheliomatous hyperplasia with intraepidermal micro-abscesses and prominent dermal inflammatory infiltrate with typical three-layered granulomas consisting of palisading suppurative granulomas surrounded by plasma cells and eosinophils (diffuse neutrophilic infiltration with dermal inflammatory infiltrates consisting of epithelioid histiocytes, lymphocytes, and multinucleated giant cells). Based on clinical and histological correlation, the diagnosis of SPG was definitively established.

KEY WORDS: superficial granulomatous pyoderma, pyoderma gangrenosum, dermoscopy.

INTRODUCTION

Pyoderma gangrenosum (PG) is a rare neutrophilic dermatosis of unknown etiology frequently associated with systemic morbidities such as inflammatory bowel diseases, rheumatoid arthritis**,** immunological dysfunctions, and myeloproliferative disorders (1,2). Four clinical and histological variants have been described: ulcerative, pustular, bullous, and vegetative. Vegetative PG is usually not associated with a systemic disease (3). We report on a case of superficial granulomatous pyoderma (SGP), a vegetative form of PG, first described by Wilson-Jones and Winkelmann in 1988 (4,5).

CASE REPORT

A 40-year-old healthy woman presented with a progressive onset of an erythematous, crusted plaque measuring 2 cm in diameter located on her left hip. The patient said the lesion had initially appeared 18 months ago as an erythematous, non symptomatic macule which was slowly evolving in to

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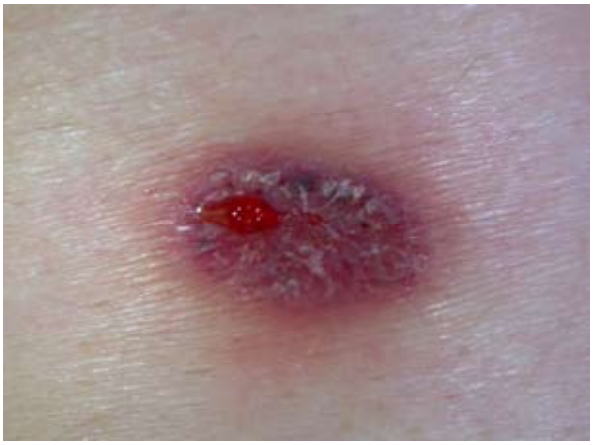

Figure 1. Erythematous crusted plaque with superficial ulceration, surrounded by purple borders.

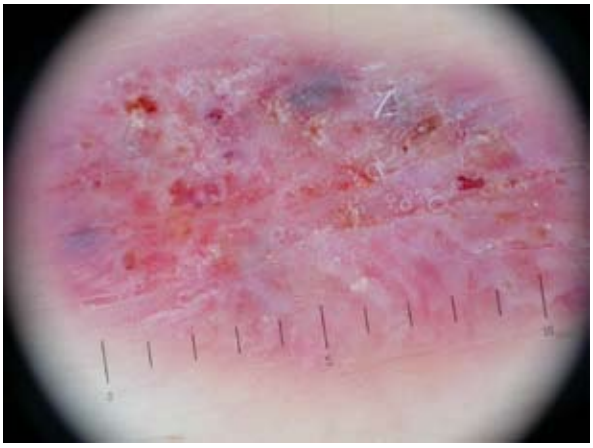

Figure 2. Dermoscopy showed lack of a pigment network, large gray-blue ovoid nests, peripheral linear and irregular vessels, and ulceration (original magnification ×20).

a plaque. It gradually increased in size and was surrounded with purple borders, as well as superficial ulceration (Figure 1). Dermoscopy with 20× magnification showed large gray-blue ovoid nests, peripheral linear and irregular vessels, and ulceration, but no pigment network (Figure 2). Laboratory findings were normal; smears and cultures for bacteria and fungi were negative. The lesion was completely ablated, and histological examination showed a florid pseudoepitheliomatous hyperplasia and a characteristic intraepithelial and superficial dermal three-layered granuloma with the inner layer consisting of sterile neutrophil micro-abscess and haemorrhage, surrounded by a granulomatous infiltrate and an outer rim of plasma cells and eosinophils (Figures 3, 4). Granulation tissue was found near to the cutaneus ulceration, and scarring was observed at base of the zoned inflammatory infiltrate. Based on the clinical and histological findings, our case was diagnosed as SGP, a localized vegetative variant of PG. Further tests, including a chest x-ray, full abdomen ultrasound, and fecal occult blood test, were all negative. Her lesion resolved after complete surgical excision, and there has been no relapse so far.

DISCUSSION

SGP is characterized by sterile abscesses, which form sinuses in the skin surface, as well as vegetative ulcers, mainly at the sites of trauma. It clinically presents with papules, nodules, or plaques, which can discharge pus. The base of the ulcer is clean and granulating, with borders that are not undermined (6). The predilection site is on the trunk: SGP is most frequently found on the back, followed by the hip, arm, and abdomen; only rarely it presents on the face (7). Dermoscopy may lead to the false diagnosis of a basal cell carcinoma (BCC), since the characteristics of such a tumor are present: in our case they included lack of a pigment network, large gray-blue ovoid nests, peripheral linear and irregular vessels, and ulceration (8-11). In SGP, culture for bacteria, fungi, and mycobacterium is negative. Healing varies from 3 months to several years; spontaneous healing is infrequent. Histologicaly, SGP is characterized by a three-layered granuloma: inner neutrophils and necrosis, a surrounding layer of histiocytes and giant cells, and an outer layer of plasma cells with eosinophils. As opposed to to PG, SGP is not associated with

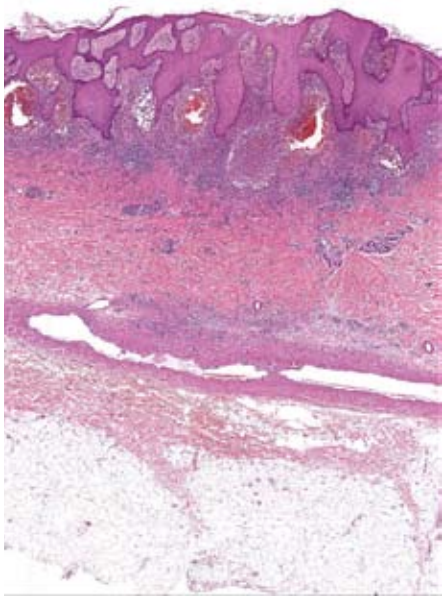

Figure 3. Low-power view showing pseudoepitheliomatous hyperplasia, subepithelial suppurative granulomata, and normal deep reticular dermis (H&E; x40).

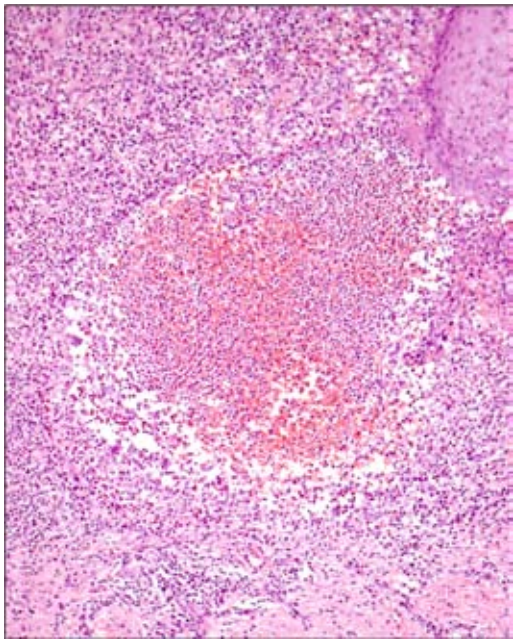

Figure 4. Typical subepidermal granuloma composed of a central zone of neutrophils and hemorrhage, a surrounding layer with histiocytes and giant cells, and an outer zone of plasma cells with eosinophils (H&E; x40).

systemic diseases (3,12). Differential diagnosis includes deep chronic infections and classic PG (3,6,12). The etiopathogenesis of SGP and PG is not clear. The association of PG with systemic diseases (ulcerative colitis, Crohn's disease, hematological malignancies, and others) suggests an underling autoimmune condition. T-lymphocyte helper/suppressor imbalance, defective neutrophil chemotaxis, impaired phagocytosis, and deranged monocyte function have been postulated in the pathogenesis of PG. Acute vascular insufficiencies of the skin, as a consequence of immunocomplexes deposition, and lymphocytotoxicity leading to infarcts, have also been proposed (3,6,12). The histological finding of vasculitis and the occasional demonstration of immunoglobulin complement through immunofluorescence, as well as fibrin deposits, support this hypothesis. Furthermore, in SGP the histological findings, especially the three-layered granuloma with eosinophils, are suggestive of an immunological granuloma, which is a delayed type of hypersensivity to an otherwise non-pathogenic organism. The antigen has not yet been documented, but it could be either endogenous or exogenous (6).

SGP shares several features with PG, such as a similar clinical appearance, pyodermatous histology, and pathergy. However, there are differences between the two forms: SPG has a slower progression of the lesion, less tendency for the undermining of skin edges, and a lack of regular association with other diseases (3,6). Although there are no pathognomonic histological features of SGP, foreign-type granulomas, sinus tract formation, and pseudoepitheliomatous hyperplasia are regularly present in SGP, but by far less so in PG. SGP is a diagnosis by exclusion: suppurative granulomatous infections, such as those caused by mycobacterium or deep seated fungi, should be considered the main histological differential diagnosis of SGP (7). Pyoderma-like lesions can also be observed in deep fungal infections, mycobacterium disease, foreign body granuloma, and other conditions. Treatment response in SGP is variable and unpredictable. Therapeutic options include oral tetracyclines, sulphapyridine, dapsone, clofazimine, immunosuppressants, and topical steroids; systemic steroids are usually unnecessary (6). Recalcitrant cases have been resolved with cyclosporine, minocycline, intravenous immunoglobulin, and anti-TNF-α agents (7,13-17). Lachapelle *et al.* have suggested that cyclosporine should be a first-line treatment for this condition (18); however, in our opinion, since this SGP progresses slowly, treatments with less significant complications should be tried first (7).

CONCLUSION

We present this case because of the rarity of SPG, but mostly because we would like to emphasize the clinical and dermatoscopical resemblance with BCC that might lead to a false diagnosis. Progressive onset, slow evolution, superficial ulceration, lack of pigmented network, and no association with a systemic disease suggest that BCC should also be added as a differential diagnosis of SPG.

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