

LETTER TO THE EDITOR

Disseminated superficial granulomatous pyoderma

Dear Editors,

Staff of wound centres may not be familiar with superficial granulomatous pyoderma (SGP), which is a rare variant of pyoderma gangrenosum (PG). SGP tends to appear as a single, non-tender, well-defined superficial ulcer with exophytic or vegetating clean granulations.¹ We report a case of disseminated SGP in an otherwise healthy male.

A 61-year-old man with a history of arterial hypertension presented with a 6-year history of multiloculated, slowly growing, superficial ulcerated lesions. The skin condition had originally begun as a single erosive violaceous, partly apple-jelly plaque on his right ankle (Figure 1). Over the following years, several new ulcers developed over the arms, back, and legs. These superficial, oval, ulcerated violaceous lesions had a sharp border and brownish-yellowish crusts and formation of crypts. Multiple skin biopsies had been performed with consistent findings including acanthotic epidermis and formation dermal crypts and fistulas, with sheets of neutrophils surrounded by palisaded epithelioid histiocytes and foreign body-type multinucleated giant cells. There was also extensive dermal fibrosis and a mixed inflammatory cell infiltrate particularly including plasma cells and eosinophils extending into the deep dermis (Figure 2). Several tissue and pus cultures revealed negative results for bacteria, mycobacteria, parasites, or fungi. Moreover, direct polymerase chain reaction for leishmaniasis and *Mycobacterium tuberculosis* complex in tissue specimen was negative. The patient had never experienced any episode of unclear fever or other systemic constitutional symptoms. Blood investigations including anti-nuclear, anti-glycoprotein, anti-cardiolipin and anti-neutrophil cytoplasmic antibodies, extractable nuclear antigen, rheumatoid factor, serum protein electrophoresis and immunoglobulins, serum complement, interleukin 2 receptor, and angiotensin-converting enzyme levels were all within normal limits. Abdominal/thoracic computed tomography and lymph node ultrasound did not reveal relevant pathologies. The clinical and histopathological findings were consistent with a diagnosis of disseminated SGP. The patient was given a tapering course of oral prednisolone. This resulted in transient improvement but not complete resolution of the skin lesions.

SGP has first been described in 1988 by Wilson-Jones and Winkelmann.¹ Five major clinical and histopathological variants of PG have been suggested – ulcerative, pustular, bullous, peristomal, and vegetative (superficial

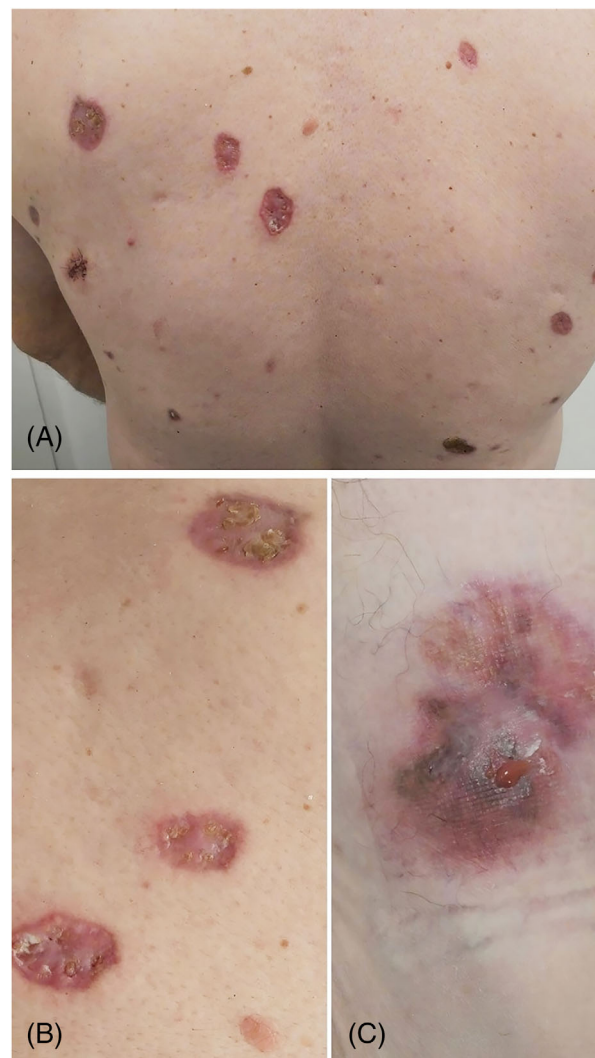


FIGURE 1 A patient with disseminated superficial granulomatous pyoderma showing oval, superficially ulcerated violaceous lesions with a sharp border and brownish-yellowish crusts and formation of crypts (A, B). Moreover, there was a single erosive violaceous, partly apple-jelly plaque on his right ankle (C)

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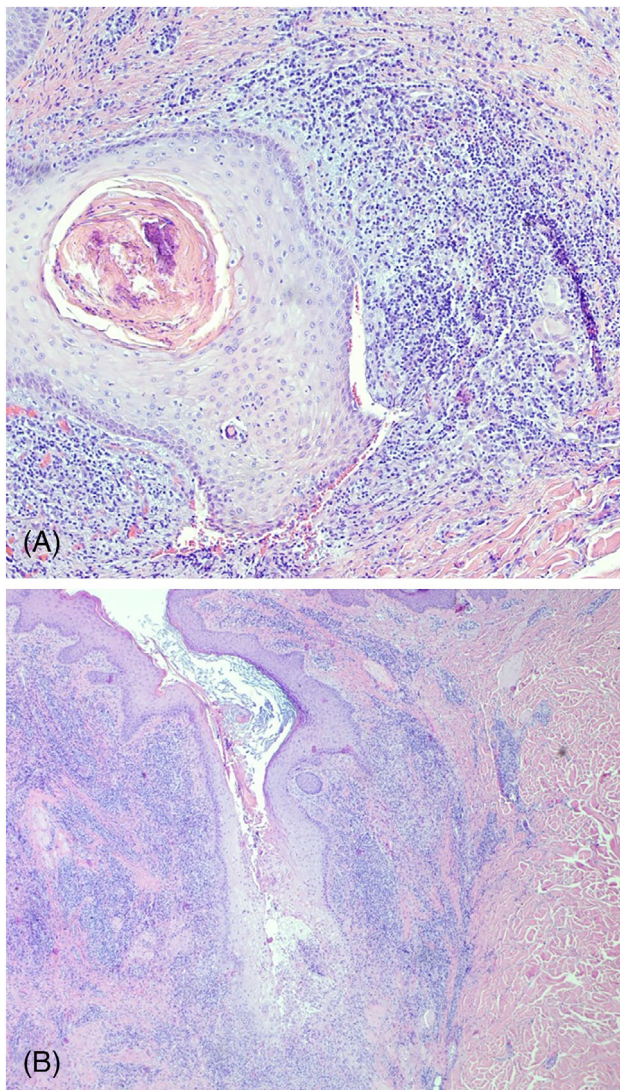


FIGURE 2 Histopathology of a patient with disseminated superficial granulomatous pyoderma showing acanthotic epidermis with some dyskeratotic keratinocytes (A) and formation dermal crypts and fistulas (B), with sheets of neutrophils partly surrounded by palisaded epithelioid histiocytes and foreign body-type multinucleated giant cells. In the deep dermis, there was also fibrosis and sclerosis a mixed inflammatory cell infiltrate particularly including plasma cells and eosinophils extending

granulomatous). For diagnosis of SGP, clinical and histopathological features are the mainstay after excluding other causes such as infections, autoimmune/autoinflammatory diseases, granulomatous conditions, or cutaneous neoplasms.¹⁻⁵ Clinically, the predilection site of SGP is the trunk, but may also rarely affect the face. Unlike classic PG, SGP is usually not associated with underlying systemic conditions. In most cases, SGP tends to appear as a single, non-tender, sharply bordered superficial ulcerated plaque with vegetating clean granulations. Histopathological findings consist of three-layered central zone of neutrophils surrounding with granuloma and outer layer of numerous plasma cells and

eosinophils, whereby sinus tract formations are typically observed in SGP.¹⁻⁴ The pathogenesis of SGP remains unclear. Similar to sarcoidosis, however, it has been proposed that SGP may be a localised delayed-type hypersensitivity reaction of the skin to a yet unidentified endogenous or exogenous organism or antigen. In the present case, clinical differential diagnosis particularly included verrucous sarcoidosis. However, the histopathological findings clearly favoured SGP. Other differential diagnoses of SGP include classic PG, infections, foreign-body granuloma, granulomatous vasculitides, ulcers associated with autoinflammatory syndromes, and halogenodermas. Systemic corticosteroids are first line in the treatment of PG but are usually not necessary to control localised SGP lesions. The latter may also responsive to conservative treatment with topical anti-bacterial/anti-inflammatory agents. In disseminated SGP, however, systemic corticosteroids may be necessary.²⁻⁴

The present case of disseminated SGP concisely highlights the clinicopathological characteristics of this rare superficial variant of PG.

ACKNOWLEDGEMENTS

We thank the patient for providing informed consent for publication of his case details and images.

CONFLICT OF INTEREST

The authors declare no conflicts of interest.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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