

Feasibility of Conservative Medical Treatment for *Pyoderma Gangrenosum*

PAOLO FIORAMONTI, MARIA GIUSEPPINA ONESTI, PASQUALE FINO,
SILVIA DI RONZA, VALENTINA SORVILLO and PAOLO PERSICHETTI

*Department of Dermatology and Plastic, Reconstructive and Aesthetic Surgery,
University of Rome La Sapienza, Rome, Italy*

Abstract. *Pyoderma gangrenosum (PG) is a chronic skin disease with an incidence of 3-10 per million, and it is often associated with underlying systemic disease. A case of PG of the left leg successfully treated with local debridement and advanced and compression dressings, without systemic treatment is reported. Progression and healing of the ulcer was incredibly rapid. The treatment of PG usually consists of systemic administration of corticosteroids. But wherever the administration of systemic immunosuppressive therapy is impractical, a topical treatment can be used. This type of treatment does not represent a mere palliative, but could be significant for healing, as in the case of our patient.*

Pyoderma gangrenosum (PG) is a rare, chronic, frequently undiagnosed inflammatory skin disease, in which a painful nodule or pustule breaks down to form a progressively enlarging ulcer with a raised, tender and undermined border (1). The incidence of pyoderma gangrenosum is uncertain, but estimated to be 3-10 per million per year and has a slight predominance in females (2, 3). It is associated with underlying systemic diseases, such as arthritis and inflammatory bowel disease, in 50% of patients. Pulmonary and cardiac involvement are other possible underlying systemic diseases, although PG can also arise spontaneously (3).

The clinical course may present two patterns: an explosive and rapid spread of lesions or a spread that is indolent and gradual. Diagnosis of PG is mainly based on clinical findings because neither laboratory finding nor histological features show specific diagnostic features. Sometimes, however, a

biopsy can help exclude other conditions (4, 5). Differential diagnosis should take into account seven categories that can mimic the clinical appearance of PG (6): vascular occlusive or venous disease, vasculitis, cancer, infectious disease, exogenous tissue injury and factitious panniculitis.

There is no specific and uniformly effective therapy for *Pyoderma gangrenosum*. Treatment typically requires systemic suppression of the immune system, with corticosteroids and ciclosporin to regulate the immune response (7-10). However, a topical medical treatment for PG can be performed in patients who do not tolerate systemic treatment with corticosteroids. We report a case successfully treated by local dressing alone in which immunosuppression was not possible due to general medical condition (8-10).

Case Report

The patient was brought to our attention in April 2010, for a skin ulceration localized at the medial malleolus of the left leg. The patient had a serious underlying lung disease causing a strong immunosuppression and debilitation; he was listed for a transplant. In addition, the patient reported having undergone surgery in 1992 for ulcerative colitis. The patient was found to be positive for HbcAg.

The lesion was necrotic and highly painful. Initially, the lesion was limited to the ankle region, with an extension of 15×15 cm (Figure 1a). Within three weeks, it had spread to the whole left leg (35 cm in length and circularly extended) (Figure 1b). The progression had a peculiar feature characterized by healing of the first affected areas and the subsequent extension of the lesion in an ascending direction (Figure 1c). Furthermore, the progression of the lesion was very rapid, reaching complete extension in just three weeks. The same speed was evident in the healing phase which occurred in less than three months. This feature is even more unexpected when considering the general debilitated state of the patient, who died two days after complete wound closure (Figure 1d).

The treatment we performed consisted of washing with scrubs, wound debridement and application of silver

Correspondence to: Pasquale Fino, MD, Department of Dermatology and Plastic, Reconstructive and Aesthetic Surgery, University of Rome "La Sapienza", Policlinico Umberto I, Viale Pantelleria, 35, Scala B, Interno 1/A, 00141, Rome, Italy. Fax: +39 06491525, e-mail: pasquale.fino@gmail.com, pasquale.fino@uniroma1.it

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Figure 1. View of the left leg wound at the first dressing (a), after three weeks (maximal spread) (b), after six weeks showing initial healing (c), and at the end of treatment (two days before death) (d).

dressings alternating with absorbent dressings. Finally, a compression dressing was made. Treatment was performed three times a week for three months. The week after the first dressing, microbiological investigation demonstrated the presence of *Staphylococcus aureus*, and the patient was treated for two weeks with Ciprofloxacin (one tablet 500 mg taken twice a day), Piperacillin Sodium/Tazobactam Sodium (2 gr Piperacillin sodium and 0.250 gr Tazobactam Sodium injection solution taken three times a day), Amoxicillin/Clavulanic Acid (one tablet Amoxicillin 875 mg and Clavulanic Acid 125 mg taken three times a day). Patient received no corticosteroid therapy.

Discussion

Brunsting and Underwood (10) in 1949 first described five patients with rapidly progressive and painful suppurative skin ulceration with necrotic and undermined borders that were called *Pyoderma gangrenosum*. PG is a primarily sterile, inflammatory neutrophilic dermatosis. It occurs most commonly on the lower legs, with prevalence at the pretibial area, but PG has also been reported on other sites of the body

as well, including breast, hand, trunk, head and neck, and peristomal skin (10). The ulcer starts as a follicular pustule with rapid growth, tissue necrosis and enlargement of the area. The surrounding skin is erythematous with infiltration and oedema; in addition, ulcer borders are typically undermined and violaceous or bluish. The lesion develops a purulent cover, which rapidly becomes malodorous due to secondary infection.

The aetiology of PG is unknown and the pathogenesis is poorly understood. Although 25–50% of patients develop an idiopathic form of PG, an underlying immunological abnormality may exist, as suggested by its frequent association with systemic diseases with a suspected autoimmune pathogenesis.

Because the histopathological findings are non-specific, PG diagnosis rests primarily on clinical features. The primary objective of biopsy is to rule out other causes of ulceration (*e.g.* infection, vasculitis, malignancy). Skin biopsy specimens reveal oedema and massive neutrophilic inflammation; it can also show engorgement and thrombosis of small and medium-sized vessels, necrosis, and haemorrhage. The dense infiltrate of polymorphonuclear

leucocytes leads to abscess formation and liquefaction necrosis. Lesions further evolve into suppurative granulomatous dermatitis and regress with prominent fibroplasia. The neutrophil is the cytological hallmark of PG. In fully developed ulcers, there is marked tissue necrosis with surrounding mononuclear cell infiltrates.

The primary target of treatment is to solve the underlying disease. Most patients need systemic corticosteroid treatment to induce remission, which halts the progression of existing ulceration and prevents the development of new lesions. Prednisolone is the drug of choice and high doses such as 100-200 mg/day may be necessary initially.

However, in the case presented, local or general immunosuppression therapy was not feasible due to the general poor health condition of the patient. A local wound treatment was started for the sole aim to control the disease progression; however, the enlargement of the ulcer as well as its reduction up to the complete closure of the wound, was extremely rapid, within three months from the onset of the wound.

In conclusion, PG is a disease with an extremely variable presentation, progression and prognosis. It usually requires a whole spectrum of treatment modalities, starting with the control of the immune system with corticosteroids up to local, medical and surgical treatment. In selected cases, however, only the use of advanced dressings can allow a complete recovery in a short time.

References

- 1 Wolff K and Stingl G: *Pyoderma gangrenosum*. Fitzpatrick's Dermatology in General Medicine, 5th edn. Volume 2. In: Freedberg IM, Eisen AZ, Wolff K, Austen KF, Goldsmith LA, Katz SI and Fitzpatrick TB (eds.). New York, NY: Mc Graw-Hill, pp. 2207-2213, 1999.
- 2 Bologna JL, Jorizzo JL and Rapini RP (eds). *Pyoderma gangrenosum*. Dermatology. London, UK: Mosby 1: 415-418, 2003.
- 3 Ruocco E, Sangiuliano S, Gravina AG, Miranda A and Nicoletti G: *Pyoderma gangrenosum*: an updated review. J Eur Acad Dermatol Venereol 23(9): 1008-1017, 2009.
- 4 Brooklyn T, Dunnill G and Probert C: Diagnosis and treatment of *Pyoderma gangrenosum*. BMJ 333(7560): 181-184, 2006.
- 5 Aseni P, Di Sandro S, Mihaylov P, Lamperti L and De Carlis LG: Atypical presentation of *Pyoderma gangrenosum* complicating ulcerative colitis: rapid disappearance with methylprednisolone. World J Gastroenterol 14(35): 5471-5473, 2008.
- 6 Weenig RH, Davis MD, Dahl PR and Su WP: Skin ulcers misdiagnosed as *Pyoderma gangrenosum*. N Engl J Med 347(18): 1412-1418, 2002.
- 7 Knock K and Butcher M: Treatment of an atypical leg ulcer: *Pyoderma gangrenosum*. Br J Community Nurs 8(6 Suppl): 19-25, 2003.
- 8 Onesti MG, Bitonti A, Fino P, Ciotti M and Scuderi N: New therapeutic strategies for the treatment of difficult wounds. G Chir 29(5): 212-220, 2008.
- 9 Fioramonti P, Onesti MG, Tempesta M, Bitonti A, Fino P, Palumbo F and Scuderi N: Electronic sheet in the management of difficult wounds. G Chir 28(10): 394-398, 2007.
- 10 Brunsting LA and Underwood LJ: *Pyoderma vegetans* in association with chronic ulcerative colitis. Arch Derm Syphilol 60(2): 161-172, 1949.

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