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Disseminated Bullous Pyoderma Gangrenosum: A Clinical Case Report

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ABSTRACT

Pyoderma gangrenosum is a neutrophilic dermatosis that is difficult to diagnose, with various clinical manifestations. Its variants include ulcerous, pustulous, bullous, vegetative, peristomal, and drug-induced. We present a clinical case of bullous pyoderma gangrenosum in a male patient with generalized dermatosis, characterized by flaccid blisters of hematopurulent content, which are subsequently unroofed and evolve into painful ulcers with irregular and purplish borders. Associated diseases were ruled out. The diagnosis was made by histopathological study that showed perivascular and deep neutrophilic inflammatory infiltrate, with areas of necrosis and edema. Remission of lesions was obtained with combined topical corticosteroid therapy and systemic therapy with thalidomide.

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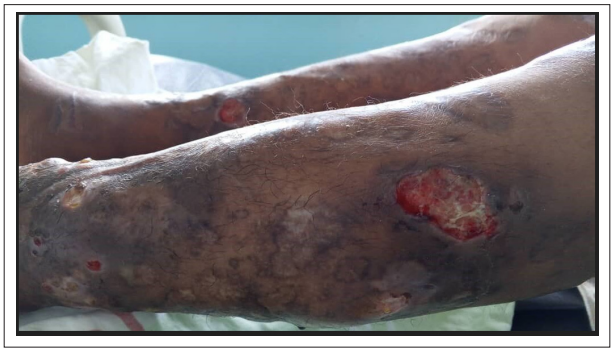
Clinical Case

A 19-year-old male patient from Puerto Cabello, Venezuela, had a cutaneous phototype IV according to the Fitzpatrick scale. She consulted because she presented with painful generalized ulcers of variable sizes, with an evolution of four months. The dermatosis began in the upper and lower limbs and face, presenting flaccid blisters, which progressed to painful ulcers with irregular borders with a purplish halo. He was treated with antibiotic regimens (ciprofloxacin and clindamycin) without improvement.

She attended the dermatology service of the Dr. Enrique Tejera Hospital City after two months. On physical examination, the patient was in stable clinical condition, without fever or lymphadenopathy. Painful ulcers in the lower limbs of variable size with a violet halo and fibrin bottoms are evident. Also, flaccid blister on the right arm with hematopurulent content, in addition to multiple cribriform scars and signs of patergia. (Figure 1 and 2).



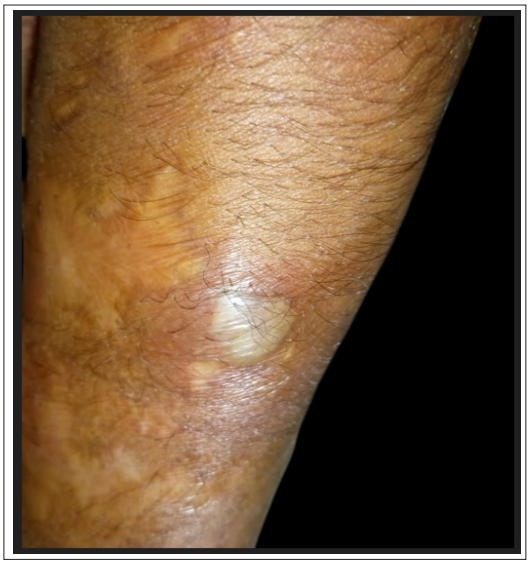


Figure 1 and 2: Ulcers in the Lower Limbs of Variable Size with a Purplish Halo and Bottoms with Fibrin. Also, Flaccid Blister on the Right Arm with Hematopurulent Content

The rest of the physical examination scalp, mucosa and nails showed no alterations. The diagnoses of bullous pyoderma gangrenosum, paraneoplastic pemphigus and Sweet's syndrome were raised.

Culture was requested (for typical and atypical germs and fungi), obtaining results without bacterial or fungal growth. Laboratory tests (complete hematology, ESR, kidney, thyroid and liver function, coagulation times, anti-Ana, anti-DNA, C3, C4, rheumatoid factor)

and imaging studies (chest x-ray and abdominal ultrasound) were within their normal range. The patient was evaluated by the Gastroenterology and Hematology services, with no clinical evidence of inflammatory bowel disease or acute myeloid leukemia. Complementary studies included endoscopy, colonoscopy and peripheral blood smears, which showed no abnormalities. In addition, POTATO, PASH AND PAPASH syndromes were ruled out.

The histopathological study, carried out with special stains of Fite-Faraco and Grocott, yielded negative results for alcohol-fast acid bacillus and fungi, respectively. In addition, a neutrophilic inflammatory infiltrate with areas of fibrinoid necrosis and edema was observed with hematoxylin-eosin staining at 40X in the dermis. In conclusion, bullous pyoderma gangrenosum was diagnosed (Figure 3).

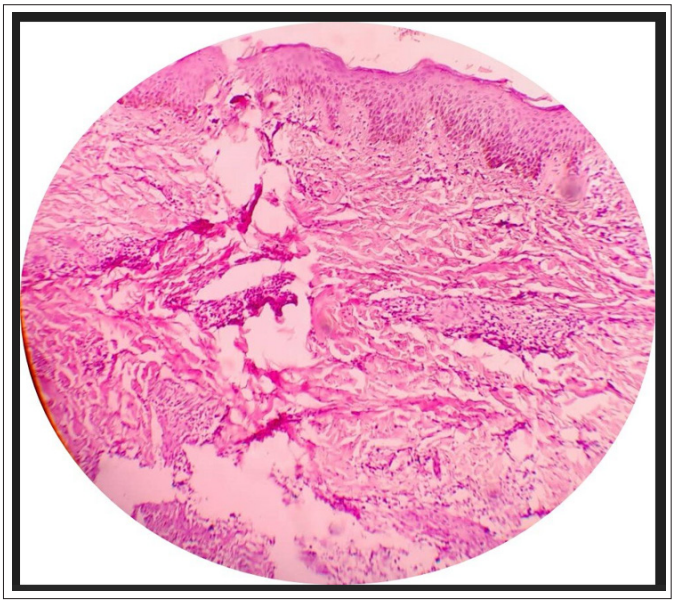


Figure 3: Hematoxylin-Eosin staining (40X) is observed in Neutrophilic Inflammatory Infiltrated Dermis with Areas of Fibrinoid Necrosis and Edema

The indicated therapy included topical clobetasol propionate 0.05% (cream) on the lesions, applied once daily for twenty-one days. Due to the extent of the dermatosis, systemic treatment was suggested with prednisone 0.5 mg/kg/day in a descending schedule for three months and cyclosporine 5 mg/kg/day for 20 weeks, with little reduction in lesions.

Two months later, the patient presented recurrence of the lesions, so it was decided to implement a combined treatment with clobetasol propionate 0.05% alternated with tacrolimus 0.1% for one month, followed by prednisone 1 mg/kg/day with gradual reduction of the steroid for four months and thalidomide 100 mg/day for six months, obtaining complete remission of the dermatosis after four weeks. Currently, the patient is untreated and under control, with no evidence of the disease after five months of treatment discontinuation (Figure 4).

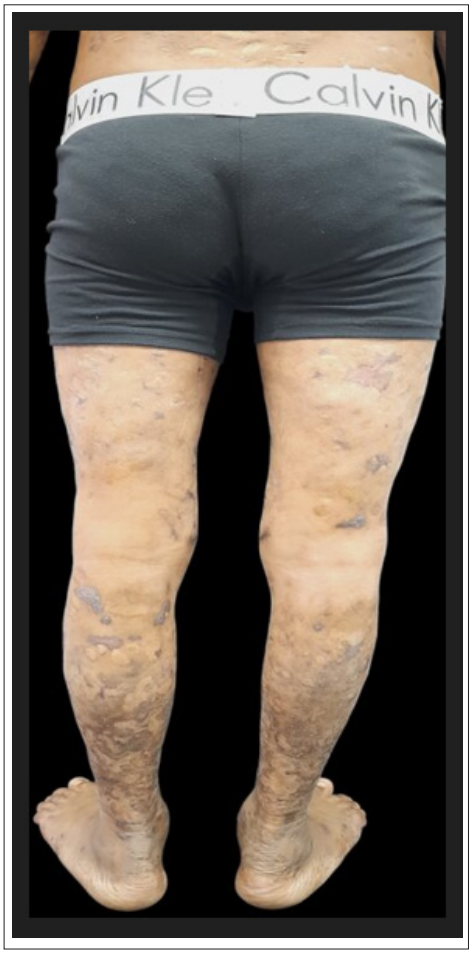


Figure 4: Complete Remission of the Lesions, Multiple Cribriform Scars are Observed

Discussion

Pyoderma gangrenosum is a complex neutrophilic dermatosis that is difficult to diagnose, belonging to a group of non-infectious entities. It is frequently associated with systemic pathologies such as inflammatory bowel disease, hematological disorders or autoimmune diseases [1].

The pathophysiology of this disease is not yet well elucidated, but it is considered to involve dysfunctions in chemotaxis and neutrophilic activity, along with alterations in the immune response. Immune abnormalities have been identified in some patients, including elevated levels of IL-8, IL-16, IL-23, and TNF-α. The close relationship with inflammatory bowel disease suggests the possible involvement of cross-reaction mechanisms between intestinal and cutaneous antigens, which reinforces the hypothesis of a shared immunological link [1,2].

Clinically, it has several variants, including classic (ulcerative), bullous, vegetative, pustulous, peristomal and drug-induced. The ulcerative form is the most common and is usually located in the lower limbs, starting with pustular lesions that rapidly evolve into necrotic ulcers with punch-shaped edges. These rapidly progressive lesions generate a significant clinical impact due to their painful and destructive nature [3].

The bullous variant of pyoderma gangrenosum usually manifests itself in less common areas, such as the face, extensor surfaces, and upper limbs. It is characterized by the appearance of hemorrhagic

blisters, accompanied by erythematous plaques or nodules that progressively develop phlyctenas on their surface. These lesions may lose their covering, giving rise to superficial ulcerations with purplish edges, around which new blisters emerge that rapidly expand in concentric and centrifugal patterns [4,5].

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Atypical pyoderma gangrenosum represents a real diagnostic challenge due to its clinical variable, overlap with other dermatoses and its frequent association with systemic diseases. The absence of pathognomonic findings in histopathology and laboratory studies requires that other entities be ruled out before confirming the diagnosis. The differentials to be considered depend on the evolution of the disease and include typical and atypical pathogen infections, paraneoplastic pemphigus, erythema multiforme, and Sweet's syndrome [5].

In clinical cases, the bullous variant stands out, without association with systemic diseases and its appearance in the lower extremities, which is not frequent in bullous pyoderma gangrenosum. This manifestation, although less common, is equally challenging to deal with. It is common to observe the phenomenon of pathergy at sites of recent trauma, as in the case described.

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erythrocytes and epidermal fibrinoid necrosis [4,5]. The biopsy result of the presented case reported neutrophilic inflammatory infiltrate with areas of fibrinoid necrosis and edema, as described in previous studies. In dermatology, it is crucial to know the clinical characteristics of each variant and the diseases associated with this pathology. It is also essential to perform laboratory tests and histopathological studies to confirm the diagnosis and provide timely management of the disease.

The management of pyoderma gangrenosum requires a multidisciplinary and individualized approach, given the variability in response to treatment. There are various immunosuppressive and immunomodulatory therapies for the treatment of affected patients. In addition, concomitant topical drug treatment, wound management, and pain management should always be addressed. Corticosteroids and/or cyclosporine remain the systemic therapies of choice for most patients. The use of immunomodulators such as dapsone, thalidomide, and intravenous immunoglobulin may also be considered [6]. In the literature, cases of recalcitrant, refractory, or resistant PG are reported that initially did not respond to various schemes. Becker, in his series of 148 cases, reports that most of them improved with second- or third-line combination therapy [7].

The use of thalidomide in 1965 reported improvement in the treatment of erythema nodosum due to leprosy and since then good results have been seen in multiple skin pathologies of an inflammatory nature. Its immunomodulatory action inhibits neutrophil chemotaxis and phagocytosis and the production of pro-inflammatory mediators such as tumor necrosis factor, interleukin 12 and gamma interferon, as well as its antiangiogenic effect. Currently, it is approved for use in PG, Sweet's syndrome, Behcet's disease, discoid lupus erythematosus, rheumatoid arthritis, prurigo, erythema multiforme, Kaposi's sarcoma, lymphomas, and multiple myeloma [8,9].

There are about a dozen reports that weigh the excellent response obtained with thalidomide alone or in combination in initially refractory cases. Responses vary with doses of 100 to 400 mg daily, obtained in ranges of 5 days to 9 months, without recurrence [9,10].

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Conclusion

There is currently no standardized therapeutic protocol for this pathology. Although the bullous variant is rare, it represents a diagnostic and therapeutic challenge. The case presented showed a favorable response to the combined treatment, which highlights the importance of an individualized and multidisciplinary approach in the approach to this complex dermatosis. More studies and clinical trials are required to evaluate the efficacy and safety of combined therapeutic schemes.

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