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Case Study

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AN INHABITUEL OCCURRENCE OF CUTANEOUS PYODERMA GANGRENOSUM IN A PATIENT WITH RHEUMATOID ARTHRITIS ON METHOTREXATE

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ABSTRACT

Background: Pyoderma gangrenosum (PG) is a rare inflammatory pathology in the group of neutrophilic dermatoses, resulting in ulceration of the skin. Commonly, More than half of patients acquire PG in association with implicit inflammatory bowel illness, hematologic malignancy, hepatisis, polyarthritis, such as rheumatoid polyarthritis (PR).[1] Its pathophysiology is complicated, not amply comprehended. The diagnosis of PG can be challenging and is often one of exclusion. We here report a case of PG in a patient with rheumatoid polyarthritis on methotrexate. The purpose of this manuscript was to highlight the epidemiological, clinical, therapeutic and evolutionary features of this dermatosis.

KEYWORDS: Pyoderma Gangrenosum; Rheumatoid Arthritis, Neutrophilic Dermatoses.

CASE REPORT

A 56-year-old woman was evaluated in the outpatient dermatology clinic as a second opinion for an acutely worsening ulcer over her anterior shin. The patient had a history of rheumatoid arthritis who were successfully contained with oral corticosteroids 60 mg /day, and methotrexate 25 mg per week for tow years.

At presentation in our clinic, The start dates back to 4 months with gradual onset, without causing factors of a painful erythematous papule sits on the extended face of the left leg. it extended then gradually increase in a few days with a centrifugal extension and continues to develop into a necrotic and mucopurulent ulcer. There was no fever or deterioration of the general condition.

On examination, he wound on the patient's left leg showed extensive necrosis and ulceration of the skin with with atrophic center and polycyclic purplish edges undermined. It was solitary circular ulceration, the lesion was oozing ulcer axis measuring 5 4 cm with sharp edges surrounded by a bulge red located on the anterior aspect of the left leg. (figure1)(figure2) an origin arterial or venous was eliminated by Doppler ultrasound.

There was no biological inflammatory syndrome. the blood count was normal.

Results of microbiological examinations of swab samples and wound cultures shows tow pathogens : staphycoccus and mycoplasm.

Faced with the resistance of the leg ulceration to antibiotics and its progressive deterioration, the patient underwent a skin biopsy.

Histopathological examination of the skin biopsy taken from a border revealed massive neutrophilic inflammation often altered with exocytosis, without vasculitis,and without signs of malignancy. The diagnosis of PG was confirmed.

The patient received local treatment, the lesion was covered with daily dressings by acid fucidic; a partially pleasing evolution was notable. patient's severe pain quickly resolved and the ulcers healed within a few weeks.

DISCUSSION

PG is a rare condition, an infrequent chronic neutrophilic dermatosis, defined by extensive superficial cutaneous ulceration, recurrent with localizations multifocal cutaneo-mucosa.

It has been described at first in the United States in the 1930s by Brunsting, Goeckermann and O´Leary.[4]

PG is an ulceratingorder of the skin with sterile neutrophilic inflammation that may be seen in up to 5% of patient with chronic ulceration of the legs.[4]

Epidemiological data are insufficient precise, the general incidence is estimated between 3 and 10 cases per year,

per million inhabitants and the peak incidence is between 25 and 55 years old.[5]

Diagnosis of PG is above all clinical, confirmed by the examination histopathological. The initial lesion is a pustule type sterile or hot nodule, of spontaneous appearance which ulcerates and very quickly undergoes centrifugal extension superficial. PG develops predominantly on the lower limbs (75-80%) but can affect the whole mucocutaneous covering (upper limbs, thorax,hands, trunk, head, neck, upper aero-digestive tract, eyes, genital mucosa) Several conditions can be confused with a PG which can delay the Diagnosis; The association of P.G with another pathology is seen in 50% of case and therefore the age of onset is variable and depends on the associated pathology.

These conditions include digestive pathologies (Crohn's disease and ulcerative-hemorrhagic recto-colitis), hepatic (chronic active hepatitis, sclerosing cholangitis,primary biliary cirrhosis), rheumatic (polyarthritis rheumatoid as in our patient, spondyloartropathy, polychondritis) and hematological (dysglobulinemia, leukaemias, polycythemia myeloproliferative syndromes, thrombocystemia, myeloid splenomegaly, myelodysplasia).[5]

Maverakis et al contend that PG is defined by criteria, that represent an important step forward in the diagnosis of PG, no longer labeled as a diagnosis of exclusion. The sole major diagnostic criterion for ulcerative PG proceeds by the Delphi exercise was biopsy of ulcer border confirming a neutrophilic infiltrate. Biopsy is also precious to evaluate for PG imitators. Although at a minimum we Propose that patients with a suspected diagnosis of ulcerative PG undergo a thorough history and physical examination, a skin biopsy with tissue culture, a complete blood count with differential, and age-appropriate malignancy screening, with further evaluation guided by the patient's age, comorbidities, and symptoms according to the clinical judgment of the treating physician.

The original diagnostic criteria for PG proposed by Su et al listed rapid response to systemic steroid treatment as the fourth minor criterion.
From a historical perspective, systemic corticosteroids have been used as the therapeutic criterion standard against which novelsteroid-sparing therapies have been measured.

In the management of PG, cyclosporine and tumor necrosis, factor- inhibitors are now considered first-line therapies for PG along with systemic corticoste roids.[6]

An invasive procedure, like irrigation; lesion débridement or skin grafting, may raise progression of the malady and have to be performed only under adequate immunosuppressive therapy.[4]

Althouth, Treatment of PG is not well codified and remains empirical. For localized lesions, the topical treatment may be sufficient.[7]

The evolution of PG is associated with a rheumatic condition is generally less favorable and standard treatment with corticosteroids systemic associated with an immunomodulator can be insufficient. In a 2-year study, 23.4% of PG associated with arthritis heal while we observe 78.9% healing for other PGs.[7]

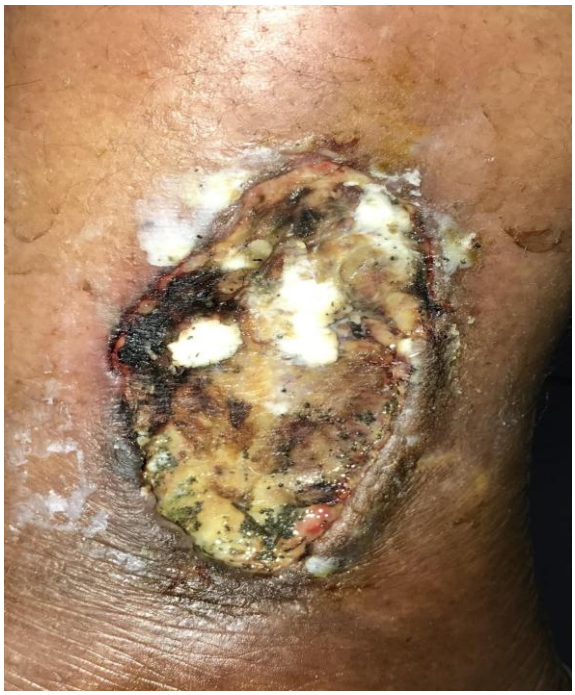

Figure 1: a necrotic and mucopurulent ulcer on the extended face of the left leg.

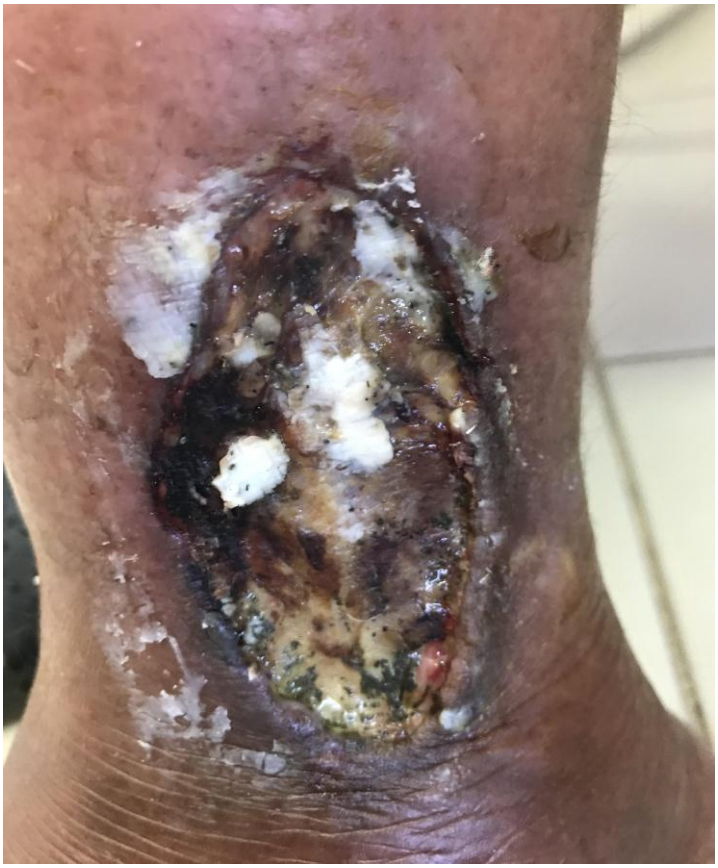

Figure 2: a necrotic and mucopurulent ulcer on the extended face of the left leg.

CONCLUSION

PG is an infrequent pathology Its diagnosis is often difficult, and commonly misdiagnosed. A thorough evaluation is required in all patients suspected of having PG to rule out alternative diagnoses. multiple explorationsis required to eliminate an infectious cause and a malignant lesion. Its management requires excellent coordination between the attending physician and specialists such us internists, dermatologists, infectious diseases.

REFERENCES

1. Paul A. Leiphart, BA, a Charlene C. Lam, MD, b and Galen T. Foulke, MDb Hershey, Pennsylvania; Suppression of pathergy in pyoderma gangrenosum with infliximab allowing for successful tendon debridement. JAAD CASE REPORTS, January 2018; 4(1).
2. Brittney DeClerck, MD*; Paul Jhun, MD; Aaron Bright, MD; Mel Herbert, MDthe American College of Emergency Physicians. Pyoderma Gangrenosum: An Inside Job, 2015.
3. Newell LM, Malkinson FD. "Pyoderma (ecthyma) gangrenosum" by Brunsting, Goeckerman and O'Leary, October 1930, Commentary: pyoderma gangrenosum. Arch Dermatol, 1982; 118(10): 743-73.
4. Norman-Philipp Hoff MD, Peter Arne Gerber MD,Pyoderma gangrenosum associated with chronic polyarthritis, CMAJ, 2011 ; DOI:10.1503 /cmaj.110679.
5. Faten Frikha1, &, Chifa Damak1, Yosra Bouattour1, Mayeda Ben Hamad1, Ranya Ghariani1, Mouna Snoussi1, Raida Ben Salah1, Zouhir Bahloul; Pyoderma gangrenosum associated with rheumatoid arthritis: a diagnosis that should be suspected. Case report | Volume 1, Article 53, 10 Dec 2019 | 10.11604/pamj-cm.2019.1.53.21048.
6. Hovik J. Ashchyan, MD, a Caroline A. Nelson, MD, b, Sasha Stephen, MD, c William D. James, MD, cRobert G. Micheletti, MD, c and Misha; Rosenbach, MDc4.‘‘New validated diagnostic criteria for pyoderma gangrenosum’’https://doi.org/10.1016/j.jaad.2018.12 .013.J AM ACAD DERMATOL APRIL, 2019.
7. Charles CA, Bialy TL, Falabella AF, Eaglstein WH, Kerdel FA, Kirsner RS. Poor prognosis of arthritis-associated pyoderma gangrenosum. Arch Dermatol, 2004 Jul; 140(7): 861-4.