

A Case of Gangrenous Pyoderma Treated with Ozone Therapy

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SUMMARY - *Pyoderma gangrenosum* is a skin lesion classified among the necrotizing lesions, having an imprecise etiology and an evolution that does not tend to cure spontaneously. The commonly accepted treatment considered useful is very aggressive and has well-known side-effects (corticosteroids and antimitotic immunosuppressants). This paper describes the case of a patient cured through the application of ozone and the patient's own platelet growth factors, all of which brings hope to understanding the etiology of *pyoderma gangrenosum* and giving us a means of treating it effectively.

Introduction

Necrotizing lesions are usually the result of infection by a variety of aerobic and anaerobic microorganisms. There are also similar acknowledged infections caused by fungi, zygomycetes (previously known as ficomycetes). Meleney (REF) described necrotizing infections due to beta-hemolytic streptococci and bacterian synergy, which nowadays many still call *Meleney's gangrene* or *Meleney's synergic infection*.

Necrotizing syndromes tend to appear in immunocompromised hosts with degenerative and debilitating diseases, diabetes or vascular diseases, or neoplasia, or in patients who have undergone trauma or been subjected to operations involving the gastrointestinal and genital tracts. However, they also arise in people with no pathological history, with a minimum of open trauma or traumatism brought on by contusion, after hypodermic injections and even following clean operations.

Only early diagnosis and urgent radical surgical intervention combined with excellent follow-up of organic function in a good intensive care unit can save the life of the patient.

Types of Necrotizing Lesions

Depending on where the germ causing the infection settles, we will have either necrotizing cellulitis or fasciitis, or myonecrosis. Equally, depending on the germ involved we will be able to "classify" the different kinds of necrotizing lesions. Therefore we have:

1. Synergistic bacterial gangrene or Meleney's gan-

grene (Microaerophilic streptococcus + *S. aureus* or *S. Proteus*).

2. Necrotizing synergistic cellulitis.
3. Non-clostridial crepitant cellulitis.
4. Necrotizing fasciitis.
5. Staphylococcal pyoderma, staphylococcal cellulitis and wound infections.
6. Streptococcal pyoderma or impetigo, erysipela, cellulitis, ulcers and gangrenes, and streptococcal wound infections.
7. Pyoderma gangrenosum (due to polymicrobial flora).
8. Gas gangrene (clostridial).
9. Necrotizing cutaneous mucormycosis.

In general terms, we may summarise by saying that they are usually produced by aerobic streptococci, other Gram positive cocci, coliform aerobic bacilli and clostridiums. Although cellutic lesions are usually monomicrobial, in general they are of a polymicrobial etiology.

The term "gangrene" means necrosis, i.e. destruction of the tissue, but not all the blame belongs to the "causal" agent found in the lesion.

The one thing that is clear is that the survival of these patients depends on immediate and aggressive surgical treatment, which consists of wide-scale and total resection of all affected tissue. At the same time, the underlying infection must be fought with antibiotics and antifungals.

Pyoderma Gangrenosum

Among the previously described gangrenous lesions, we have pyoderma gangrenosum the etiology of which is uncertain. It was first described



Figure 1



Figure 2



Figure 3



Figure 4

in 1930 by Brunsting, Goeckerman and O'Leary (REF). Pyoderma gangrenosum is a rapid spreading ulcer, with raised erythematous-violaceous edges. It is painful, with a poor response to topical treatments and rapid improvement under therapy with systemic steroids or with sulfones. In 90.9% of cases pyoderma gangrenosum appears in women

and the remainder in men (9.1%). The average age at disease onset is 47.5 years. The most common place for the cutaneous lesion is in the lower extremities (eight patients, 72.7%), lesions have also been detected on face, upper extremities and genital area (REF). The importance of diagnosis resides in the fact that pyoderma gangrenosum appears

concomitantly with systemic ailments such as colitis ulcerosa, Crohn's disease, arthritis and hematological neoplasm (REF).

Due to its low rate of incidence, there are few studies on large case series, and few precedents based on the long-term course of the illness (REF).

Case Report

During the holidays, a female patient called me and told me of her worries: she had been bitten by a mosquito and become "infected". She said that the bite gave off stabbing pains, had blue edges, looked horrible... On inspection the lesion had a horrible appearance: raised reddish-purplish edges, the appearance of the base similar to a mass of necrotic raw hamburger-like flesh which disintegrated on dressing the wound. The perilesional appearance was edematous, pink and very sensitive to the touch (the patient told me she could tolerate the bandage too tight). Given the extreme sensitivity, I decide to wash the lesion with ozonized water and apply ozone oil with paraffin and a loose bandage. When she got home, the patient removed the dressing as it was unbearable.

Antecedents

Suffering from ulcerative colitis for 20 years, after emotional upheavals in her family, the patient has had three or four crises a year with mucosanguinous deposits. These were associated with marked physical exhaustion to the point of presenting biochemical manifestations (low protein levels, high gammaglobulin, anemia, etc. see below). The patient referred a similar episode some five years earlier and an oncological dermatologist had cured the lesion.

18th August: (figure 1)

Treatment begins as follows:

- Cleaning of the ulcer with ozonized water for 10 minutes at an ozone concentration of 20 microgrammes/ml.
- *AHT Major (110 cc de blood+100 cc of ozone at 50 mcgr/ml concentration).
- Infiltrations around the lesion with the patient's own ozonized platelet-rich plasma supplemented with procaine.

22nd August: (table 1)

No improvement, the ulcerated surface area is spreading. Blood tests are orders. The Table shows the results before and after treatment.

Table 1 Contrasted biochemical serum levels (one month apart)

Month	Albumin	Platelets	Leucos/ Neutro	Gamma- globulins	IGE	Cholesterol	Iron	Ferritine	Hcrt
22-8-8	45	142	13.28/8.74	19.4	807	230 (65/130)	40	53	34.5
19-9-8	50	165	10.83/6.71	17.9	484	208 (57/124)	28	14	34.9

25th August: (figure 2)

In view of the lack of improvement, I refer the patient to the dermatologist who had treated her six years previously for the same disease (pyoderma gangrenosum in the other leg).

1st September:

The dermatologist is adamant: he prescribes topical cortisone in cream (Diprogenta) and per os systemic corticosteroids:

- Zamene (= Deflazacort) 30 mgr over three days reducing to one tablet daily, thus: 3-3-3-----2-2-2-----1-1-1----0.

Undaunted, I continue the ozone treatment and after talking it over with her, the patient gives her consent to go on "alone" with my treatment, desisting from the specialist's cortisone treatment.

4th September: (figure 3)

Granulating tissue? I notice less inflammatory reaction, less pain and the foot lymphangitis is reducing.

11th September: (figure 4)

The periulcer inflammatory reaction seems to have subsided and the lesion is less painful. The patient is in better spirits. I increase the interval till her next visit.

26th September: (figure 5, 6, 7)

Curing from the top down. I decide to vary the treatment slightly:

- Cleaning of the ulcer with ozonized water (10 minutes at 20 mcgr/ml concentration).

*AHT Major: Autohemotherapy Major.



Figure 5



Figure 6



Figure 7

– AHA major (110 cc of blood+100 cc of ozone at 50 mcgr/ml concentration).

5. Application of the patient's own platelet-rich ozonized plasma directly onto the ulcer (especially on the periphery).

Outcome

The lesion the patient had six years ago took six months to cure with a very aggressive treatment on the part of the dermatologist:

- Infiltration of cyclosporin (alternating with perilesional soluble cortisone).
- Application of hydrocortisone cream (Diprogenta).
- Administration "per os" of 30 mg of cortisone (Zamene) over a prolonged time span.

The present treatment carried out under ozone therapy protocols plus FRAP (=FEPA: fraction enriched with platelet agents) took two months to achieve a satisfactory cure.

I attribute the initial worsening of the lesion to "immunological shock" due to the treatment with the patient's own plasma. This may be due to her immune weakness following the crisis of ulcerative colitis as well as her drop in plasmatic proteins to be taken into account.

Lastly, according to present-day dermatology, pyoderma gangrenosum does not tend to cure spontaneously! The lesion is not caused at its outset by germs but is rather a case of tissue self-aggression which manifests itself as "vasculitis" with consequent necrosis. This is why it may be stated that, in this clinical case, ozone and the patient's own growth factors were crucial in her cure.

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