

Efficacy of Etanercept in the Treatment of Acne Conglobata

Eficacia de etanercept en el tratamiento del acné conglobata

To the Editor:

Acne conglobata is a severe form of nodulocystic acne that often represents a therapeutic challenge for dermatologists due to its resistance to the usual therapies. The condition is chronic and disfiguring and has a significant impact on the patient's quality of life. It presents with numerous comedones, papules, pustules, nodules, and abscesses on the chest, back, face, and neck. Histology usually reveals a perifollicular inflammatory infiltrate, which extends to affect the adjacent dermis.

The patient was a 14-year-old adolescent (weight, 60 kg) referred by his primary care physician for management of severe acne that had not responded to topical erythromycin or oral minocycline (100 mg/d for 3 months). Physical examination revealed multiple inflammatory papules, as well as cysts and closed comedones on the face, neck, and upper chest (Figures 1 and 2). The patient had no relevant personal or family history (including laboratory abnormalities such as anemia or hypozincemia or relatives with severe acne or other inflammatory diseases). Blood tests including complete blood count, biochemistry, liver enzymes, and lipid profile were normal, and isotretinoin therapy was therefore started at the doses established for the patient's weight. After 6 months of treatment, the patient's condition continued to worsen; consequently, the isotretinoin dose was increased to 60 mg/d. While on this therapy, the patient experienced an outbreak of acne fulminans: the isotretinoin dose was decreased and oral prednisone (30 mg/d) was added. The condition improved partially, and treatment was discontinued. The total cumulative dose of isotretinoin was 10500 mg. Laboratory



Figure 1 Polymorphic acne with papules, pustules, and cysts on the neck.



Figure 2 Multiple cysts and inflammatory lesions on the back.

follow-up was within normal limits, and an axial radiograph of the vertebral column showed no abnormalities.

Five months later, the patient still presented many active acne lesions, and isotretinoin (30 mg/d) and prednisone (15 mg/d) were resumed. Following 4 months of therapy, the patient continued to experience severe flares of inflammatory and ulcerated acne, particularly on the neck and back. With a view to starting etanercept therapy we requested a work-up consisting of blood and urine tests (lipids, renal and liver profile, serology for hepatitis B virus, hepatitis C virus, and human immunodeficiency virus, antinuclear antibodies, and anti-double-stranded DNA), tuberculin test, and chest radiograph. In addition, we requested etanercept for off-label use and included the patient's informed consent in the request. All tests were normal or negative, and treatment was started with twice-weekly injections of etanercept 50 mg combined with oral isotretinoin 40 mg/d. After 2 months of treatment, there was a clear improvement (Figure 3). The patient completed the isotretinoin cycle of 9600 mg over 8 months. Etanercept (50 mg/wk) was administered an additional 3 months to prevent recurrence of the inflammatory flares. One year after the treatment was completed, the patient had experienced no relapse.

In Europe the only approved dermatologic indication for tumor necrosis factor (TNF) antagonists is psoriasis. However, these drugs have been successfully used in numerous inflammatory skin conditions.¹ With regard to the management of acne conglobata, only 1 report of etanercept treatment was found in the literature.² Another case of acne conglobata was reported in a patient with rheumatoid arthritis who improved significantly after starting treatment with infliximab.³ We also found a report of acne fulminans in a patient with synovitis, acne, pustulosis, hyperostosis, and osteitis syndrome who responded well to infliximab.⁴ There is greater experience with these drugs in hidradenitis suppurativa⁵⁻⁷ (also known as acne inversa); however, we believe that to be a distinct disorder despite the marked inflammatory response of the affected glands observed in both conditions.³



Figure 3 Residual scarring on the back after achieving therapeutic control of the condition.

Multiple factors are implicated in the pathogenesis of acne, including the proliferation of *Propionibacterium acnes*, which stimulates the production of proinflammatory cytokines such as interleukin (IL) 8, IL-1- β , and TNF- α . It would therefore seem obvious that these mediators play a role in the pathogenesis of the inflammatory lesions of acne.² The most common treatment for severe acne with a marked inflammatory component is isotretinoin at a dose of 0.5-1.0 mg/kg/d for at least 5 to 6 months, often in association with oral prednisone. However, the response is sometimes minimal and can be slow to develop, and there may be frequent flares of the acne during treatment, as occurred in our patient. Moreover, the adverse reactions of these drugs may limit their use in certain patients.³ Under these circumstances, we believe that TNF antagonists may be an effective alternative.

We present the second case in the literature of severe acne conglobata resistant to conventional treatments. In our patient, rapid, sustained remission was achieved with etanercept, a TNF antagonist.

References

1. Guhl G, Díaz-Ley B, Fernández-Herrera J. Uso de fármacos biológicos en dermatosis fuera de la indicación aprobada. Segunda parte: etanercept, efalizumab, alefacept, rituximab, daclizumab, basiliximab, omalizumab y cetuximab. *Actas Dermo-sifiliogr.* 2008;99:5-33.
2. Campione E, Mazzotta AM, Bianchi L, Chimenti S. Severe acne successfully treated with etanercept. *Acta Derm Venereol.* 2006;86:256-7.
3. Shirakawa M, Uramoto K, Harada FA. Treatment of acne conglobata with infliximab. *J Am Acad Dermatol.* 2006;55:344-6.
4. Iqbal M, Kolodney MS. Acne fulminans with synovitis-acne-pustulosis-hyperostosis-osteitis (SAPHO) syndrome treated with infliximab. *J Am Acad Dermatol.* 2005;52(5 Suppl 1):S118-20.
5. Gupta AK, Skinner AR. A review of the use of infliximab to manage cutaneous dermatoses. *J Cutan Med Surg.* 2004;8:77-89.
6. Lebowitz B, Sapadin AN. Infliximab for the treatment of hidradenitis suppurativa. *J Am Acad Dermatol.* 2003;49:S275-6.
7. Mekkes JR, Bos JD. Long-term efficacy of a single course of infliximab in hidradenitis suppurativa. *Br J Dermatol.* 2008;158:370-4.

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Annular Lesions Induced by a Chlorine Tablet

Lesiones anulares por una pastilla de cloro

To the Editor:

Chlorine is widely used in the chemical industry, as a disinfectant in swimming pools, and even as an antiseptic in Dakin solution. People can be exposed to chlorine in road traffic accidents,¹ in the workplace, by mixing cleaning products, or through swimming pool accidents. Liquid chlorine (sodium hypochlorite) and chlorine powder, tablets, or granules (symclosene) are used in swimming pools. Sodium hypochlorite is highly corrosive.¹

Trichloroisocyanuric acid (symclosene) is a stabilized chlorine disinfectant and a mild irritant to dry skin, but in the presence of water it decomposes into isocyanuric acid and hypochlorous acid, the latter of which is corrosive.²

There are no reported cases of chemical burns from symclosene tablets in the literature. We describe a chemical burn secondary to contact with a symclosene tablet.

The patient was a 43-year-old man whose 9-year-old daughter, without him noticing, placed a chlorine tablet inside his swimming trunks during a play session in a swimming pool. The tablet remained lodged between the patient's skin and the cloth of his swim trunks. Some 15 minutes later, after leaving the pool, the patient noted a sensation of burning on the left buttock and discovered the chlorine tablet.

On the left buttock there were 2 well-circumscribed circular lesions of 6 cm in diameter, characterized by elevated erythematous areas with an uneven surface (Figures 1 and 2); the lesions were pruritic and tender. The lesions were bathed in abundant clean water for 10 minutes. The pruritus and pain continued for 48 hours and the lesions healed spontaneously within 7 days with the patient washing daily with soap and water.