

about our published article: 'Botulinum toxin type A for the treatment of inverse psoriasis'.

We have done botulinum toxin injections with needles of 5 mm in length with a grade of 30° on the cutaneous surface; this made possible a depth changeability of 1–2 mm that we thought insignificant for the local and systemic diffusion. There is no evidence in literature that the intradermic administration is more related to a clinical manifestation of botulinum-like syndrome than the intramuscular administration.

The botulinum toxin therapy is a symptomatic therapy, and in the particular case of the psoriatic patients, the hypothesized modalities of the action are the blockage of the proallogenic neuromediators release and the blockage of the cholinergic glandular activity, as described in the literature. The histological changes after the treatment were not to be foreseen because there weren't any documented histological changes after the treatment in the patients affected with hyperhidrosis.

The erythema area was defined with photographic controls and the subjective symptomatology (pain and itch) was evaluated on a visual analogue scale. We didn't consider the Psoriasis Area Severity Index because of the limited extension of the lesions, and the score would have been extremely low to be significant for the evaluation of the seriousness of the inverse psoriasis.

The patients treated with botulinum toxin for the district hyperhidrosis in extended areas, with the same modalities of the dilution and the dosage, never had a symptomatology similar to that of neuropathies with a damage of the little fibers. We underline that the botulinum toxin action is transitory, and it doesn't damage the fibers but it reduces the neuromediators release.

About the potential toxicity of the botulinum toxin, we did not find in the data of the literature, and of our experience in the observation of more than 4000 treatments of palmo-plantar and axillary hyperhidrosis during the last 10 years, any severe adverse effect, cardiovascular, respiratory and neuromuscular alteration and any allergic reaction.

It has to be underlined that the patients enlisted in our study were resistant to the traditional therapies and with an important local symptomatology. In these patients, the hypothesis of the application of the botulinum toxin therapy is still experimental; therefore, many studies are necessary to verify this and to compare the results of the traditional therapies.

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Autologous serum skin test: is the paradigm falling?

Editor

We have read the discussion between Asero *et al.*¹ and Guttman-Yassky *et al.*² with great interest. In the light of our clinical experience and our data,³ we support the view put forward by Guttman-Yassky *et al.* Autologous serum skin test (ASST) needs a more critical interpretation! We even suggest that the significance of ASST reactivity should be re-evaluated in chronic idiopathic urticaria (CIU). First of all, we have to emphasize that ASST shows large variations, with rates of positivity ranging from 0 to 52.5% among healthy controls as seen in the table below.

Reference	CIU	Controls
Mari ⁴	58%	45%
Tedeschi <i>et al.</i> ⁵	–	0% (3 cases)
Sabroe <i>et al.</i> ⁶	44.51%	2.5%
Asero <i>et al.</i> ⁷	–	0% (20 cases)
Sabroe <i>et al.</i> ⁸	34.61%	2.56%
Guttman-Yassky <i>et al.</i> ⁹	53.1%	40.5%
Taskapan <i>et al.</i> ³	52.5%	55.55%

Our study, which was published in 2008, revealed that 42 of 80 patients with CIU (52.5%) had ASST reactivity. However, 25 of 45 healthy controls (55.5%) also had a positive ASST. The highest rate of ASST positivity was found in healthy women; almost 70% of healthy women showed ASST positivity. The fact that the rate of positivity was highest in the young (female) population suggests the role of immune status and female sexuality (e.g. hormonal factors, menstrual cycle) in ASST reactivity.³

As a dermatologist and allergist performing ASST for almost 10 years, I do not agree with Dr R. Asero *et al.* Even if ASST is abandoned in the light of recent data, this does not mean taking the risk of going some 20 years back in our approach to CIU; in contrast, it encourages further research.

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Improvement of psoriasis after antibiotic therapy with cefuroxime axetil

Editor

We read with interest the article of Ali and Whitehead¹ about the possible relationship between eradication of *Helicobacter pylori* and improvement of skin psoriasis. We have the feeling that authors should also stress the possible effect of antibiotic therapy on psoriasis, regardless of the effect on *H. pylori*. We would like to share our experience with antibiotic therapy with cefuroxime axetil in patients with psoriasis.

Cefuroxime axetil, a prodrug of the cephalosporin cefuroxime, has broad spectrum antibacterial activity. Cefuroxime axetil has also been suggested in therapy of localized scleroderma (morphea) and subacute cutaneous lupus erythematosus due to its immunosuppressive and anti-inflammatory activity.²

The aim of the study was to evaluate the efficacy of long-term cefuroxime axetil administration in psoriasis patients.

The study included 15 patients (6 women and 9 men) with

moderate to severe, active plaque psoriasis. Mean age of patients was 49 years (range 23–68). All patients had more than 10% body surface area (BSA) involvement and psoriasis area severity index (PASI) of more than 12. Mean duration of the disease was 12.8 years (range 0.5–23 years) and mean duration of the recent flare was 2.6 months (range 0.5–5.0 months). Four of the patients fulfilled additional classification criteria for psoriatic arthritis (CASPAR) criteria for psoriatic arthritis. Eight patients developed a flare while being on topical therapy only: five patients were on methotrexate 15 mg/week and two were on ciclosporin A at a dose of 3.5 mg/kg. In each case, previous treatment remained unchanged for not shorter than 8 months. None of the patients had symptoms of an active infection; however, in nine cases the flare was triggered by a preceding infection of the upper respiratory tract. Patients had no concomitant diseases (there was especially no indication of *H. pylori* infection). In every patient cefuroxime axetil was added according to the following scheme: 250 mg twice daily for 3 weeks, followed by 125 mg once daily for 11 weeks. Baseline monitoring included clinical assessment, registration of concomitant non-antipsoriatic drugs, determination of PASI, blood pressure, and laboratory tests, including full blood count, serum biochemistry and urinalysis. These parameters were evaluated after 4, 8 and 12 weeks of therapy. Patients were observed for possible adverse effects.

The overall response rate for achieving a reduction of $\geq 75\%$ from baseline PASI (PASI 75) was 26% after 4 weeks, 29% after 8 weeks and 21% after 12 weeks. In all patients only recently developed skin lesions healed. Older plaques, especially on the knees and elbows, remained unchanged. No significant improvement in scalp psoriasis or nails was observed. Three patients with psoriatic arthritis indicated partial improvement in arthralgia. Off-treatment response was not analysed, as patients were, during the 12-week time period, investigated for possible application of biological treatment or other systemic drugs. In most cases such therapy was applied after discontinuation of cefuroxime axetil. No severe adverse effects were noted. Two female patients complained of transient symptoms corresponding to candida vaginitis. These results show that patients on cefuroxime axetil show significant improvement of their skin disease after 4 and 8 weeks of therapy. The duration of improvement was not stable and in some patients slight deterioration was seen after 12 weeks of therapy. However, these results indicate that antibiotic therapy itself may cause improvement of psoriatic skin lesions, regardless of concomitant *Helicobacter* infection or eradication.

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