

patient was admitted and treated with oseltamivir. During her stay in hospital, several deep, bilateral genital ulcers with necrotic-looking borders were noticed. Several swab specimens were obtained for standard culture, *Neisseria gonorrhoeae* culture, and PCR testing for *Chlamydia trachomatis*, herpes simplex virus types 1 and 2, and samples were taken for serology. Empirical treatment was initiated with ceftriaxone (single dose), oral acyclovir, and amoxicillin-clavulanic acid. The ulcers, however, continued to deteriorate and it was decided to debride the area. In view of the worsening condition of the patient, and the negative results for the viral cultures, the PCR tests, and serology for syphilis and human immunodeficiency virus (HIV), her physicians decided to request dermatologic evaluation.

At the time of this evaluation, there was marked vulvar edema and the posterior region of the right labia minora had been resected, leaving an ulcerated area with a clean base. On the left side of the vulva, there was a 1.5-cm exudative ulcer with a fibrinous base. The lesions were painful and accompanied by bilateral enlarged inguinal lymph nodes of less than 1 cm. Topical treatment with absorbent dressings and fusidic acid was prescribed. The ulcers improved and began to heal within a few days; 3 weeks later, they had healed completely. Serology was negative for HIV I and II, syphilis, and cytomegalovirus. Epstein-Barr virus serology was negative for immunoglobulin (Ig) M antibodies and positive for IgG antibodies. No antinuclear antibodies were detected.

The differential diagnosis we considered included other causes of acute genital ulcer such as sexually transmitted diseases, lesions caused by trauma, complex aphthosis, and ulcerative lesions associated with autoimmune diseases or inflammatory bowel disease.⁴⁻⁶ In our case, the final diagnosis was *ulcus vulvae acutum* in association with H1N1 influenza A virus infection, although we cannot rule out the possibility that the drugs taken by the patient during her illness contributed to the aggressive course of the disease and the necrotic appearance of the lesions. There have been several reports of acute genital ulcers associated with influenza

virus infection in the last 3 years, but it is not known whether this association is due to new mutations of the virus or to the concomitant ingestion of drugs. Prospective studies will help to determine whether the prevalence and course of *ulcus vulvae acutum* in patients with influenza differ depending on whether they receive oseltamivir or purely symptomatic treatment. Familiarity with this disease in emergency and dermatology departments will improve the management of these patients and prevent unnecessary interventions.

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Ustekinumab for the Treatment of Palmar-Plantar Pustulosis[☆]

Tratamiento de la pustulosis palmo-plantar con ustekinumab

To the Editor:

Palmar-plantar pustulosis is a chronic disorder characterized by sterile pustules and scaly erythematous lesions on the palms and soles. Its etiology is unknown and its relationship to psoriasis remains controversial; some authors consider it to be a variant of psoriasis, whereas others con-

sider it to be a distinct condition.¹ It is more common in women, with a peak incidence between 30 and 40 years of age. Its close association with smoking is well known, and this has recently led some authors to suggest that it could be an autoimmune disease induced by tobacco.² It is also associated with thyroid disease, skeletal lesions, diabetes, and celiac disease. This disorder is difficult to manage, not only because of its lack of response to different treatments, but also because it has a strong impact on patients' quality of life. There is no specific treatment. Therapeutic options include topical treatment with steroids and retinoids, systemic treatment with cyclosporin, retinoids, methotrexate, and colchicine, and oral and topical phototherapy,³ but the response is usually poor and combination treatment is frequently needed. There is scientific evidence of the efficacy and safety of biological agents in patients with psoriasis vulgaris.⁴ Furthermore, these drugs have recently achieved good responses in patients with other forms of psoriasis, such

[☆] Please cite this article as: de Unamuno-Bustos B, et al. Tratamiento de la pustulosis palmo-plantar con ustekinumab. *Actas Dermosifiliogr*. 2011;102:833-834.



Figure 1 Plantar lesions before starting treatment with ustekinumab.

as palmar-plantar psoriasis. López-Esteban⁵ and Kasche⁶ described 2 patients with palmar-plantar pustular psoriasis resistant to combination treatment which improved after treatment with etanercept. There have also been reports of isolated cases of palmar-plantar psoriasis with good results after treatment with ustekinumab.⁷ Ustekinumab is a human monoclonal antibody that binds with high specificity and affinity to the p40 subunit of interleukin (IL) 12 and IL-23, and blocks the differentiation of helper T (T_H) cells into T_H1 and T_H17 cells, decreasing the release of IL-17. It is indicated for the treatment of moderate or severe psoriasis in adults with no response, contraindications, or intolerance to other systemic therapies. References to the use of ustekinumab for the treatment of palmar-plantar pustulosis are rare, although Gerdes et al. described its use in 4 patients with palmar-plantar pustulosis with a satisfactory response in 2 of them.⁸ It has been shown that there is an increase in some inflammatory mediators such as IL-17F and IL-8, which induce neutrophil activation and accumulation, thus promoting the formation of pustular lesions.^{9,10} It therefore appears reasonable to assume that ustekinumab could be a good therapeutic option since it reduces the levels of both mediators.

We present the case of a 64-year-old woman with a 14-year history of palmar-plantar pustulosis. On examination she had scaly erythematous pustular lesions on both the palms and soles (Fig. 1) that made walking very difficult and caused significant pain. For 5 years the patient had been



Figure 2 Clearance of the lesions after treatment with ustekinumab.

treated with different drugs such as ciclosporin, acitretin, or methotrexate, sometimes in combination with topical treatments such as corticosteroids and keratolytics, without obtaining complete remission of the lesions. Psoralen-UV-A sessions were also conducted, including sessions in which this therapeutic option was combined with other treatments such as acitretin and ciclosporin; these led to improvement in the palmar lesions but not in the plantar lesions. In 2007, efalizumab therapy was started; the patient remained clear of lesions for 2 years until treatment was discontinued after the drug was withdrawn from the market. Within 2 months there was a recurrence of the lesions on the palms and soles, and treatment was therefore started with etanercept at a dose of 50 mg/wk. Given the lack of response to etanercept after 5 months of treatment, it was substituted by adalimumab, which was continued for 6 months without achieving satisfactory results. In 2010, treatment was begun with ustekinumab at a dose of 45 mg every 12 weeks. After 2 doses, there was obvious improvement in the lesions with almost complete clearance, leaving only residual lesions (Fig. 2). At the time of the last follow-up, 8 months after beginning ustekinumab therapy, the patient remained free of lesions. Whenever this indication was not included in the summary of product characteristics of the drugs employed, we applied for compassionate use and obtained informed consent from the patient.

Thus, in our case, ustekinumab proved to be an effective therapeutic option that led to a significant improvement

in the lesions and increased the patient's quality of life. Although our data are insufficient to assess its long-term effect, we believe that ustekinumab therapy could be of value in cases of palmar-plantar pustulosis resistant to other treatments. Further research into the mechanism by which the drug achieves the observed improvement is warranted, as well as studies with more patients to either confirm or reject the usefulness of ustekinumab in this problematic disease.

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Multiple Warts Appearing Exclusively on Psoriasis Plaques[☆]

Verrugas múltiples localizadas exclusivamente sobre las placas de psoriasis

To the Editor:

Common warts (verruca vulgaris) are found very frequently in children and adults (prevalence, 10%) and can sometimes prove difficult to treat, particularly when multiple or recalcitrant.

We report the case of a patient who developed multiple warts on psoriasis plaques.

The patient was a 58-year-old man with a history of hypertension and an immunoglobulin A monoclonal gammopathy for which he was not receiving any treatment at the time. He had a history of plaque psoriasis with joint involvement since the age of 15. Treatment with topical corticosteroids and oral methotrexate had achieved only slight improvement in this condition. He attended our clinic because of an outbreak of small erythematous-desquamative lesions distributed symmetrically across his

arms and trunk on which multiple spiny whitish papules had developed. The appearance of the papules was consistent with a diagnosis of viral warts (Fig. 1). The patient had never previously had warts. The results of the additional tests performed were normal, and serology for human immunodeficiency virus and hepatitis B and C viruses was negative. Skin biopsy revealed an acanthotic epidermis with papillomatosis, hyperkeratosis with focal parakeratosis, and no clear signs of viral cytopathic effect. A chronic inflammatory infiltrate was found in the underlying dermis. Genotyping for the human papillomavirus (HPV) performed using hybridization techniques (Clinical Arrays, Genomica) was positive for genotypes 6 and 31. The histologic diagnosis was verruca vulgaris on plaque psoriasis. Methotrexate was withdrawn and the patient started treatment with acitretin 35 mg/d. Virtually all the warts disappeared within 1 month, but the psoriasis plaques persisted. These were then treated with narrowband UV-B phototherapy with excellent results (Fig. 2).

On reviewing the literature we found 1 report of a case of psoriasis on viral warts in a patient treated with interferon- α .¹ It has been suggested that the HPV may contain proteins that stimulate keratinocyte proliferation. These proteins, and others included in the viral capsid, can be recognized by preactivated CD4+ lymphocytes and lead to the generation of specific antibodies (antigenic activation). The autoimmune reaction triggered in this way could lead to complement activation, neutrophil chemotaxis, and formation of the Munro microabscesses so characteristic

[☆] Please cite this article as: Carnero L, et al. Verrugas múltiples localizadas exclusivamente sobre las placas de psoriasis. *Actas Dermosifiliogr.* 2011;102:835-836.