

Shiitake dermatitis was first described in a series of 23 cases published in 1977 by Nakamura,¹ who went on to publish a longer series in 1992.² To date, approximately 100 cases have been reported worldwide, mostly in China and Japan, only a few in Europe,⁸ and none in Spain.

Shiitake dermatitis appears in certain people following consumption of undercooked or raw shiitake mushrooms; this suggests that these individuals must be susceptible or hypersensitive to a thermolabile substance present in the mushroom.

As biopsy results usually indicate nonspecific spongiotic dermatitis,² shiitake dermatitis is diagnosed on the basis of the appropriate clinical signs and symptoms in a patient who has eaten undercooked or raw shiitake mushrooms.^{2-4,8}

It has a clinical picture similar to that of the flagellate dermatitis induced by drugs such as bleomycin,⁴ although the mechanism of action does not appear to be the same.⁹ While lentinan, the polysaccharide found in shiitake mushrooms, is thought to be responsible for the lesions of flagellate dermatitis^{2-4,8} due to the production of IL-1, the exact pathogenesis is poorly understood.

The negative patch test results cast doubt on the hypersensitivity hypothesis. However, the results of scratch and prick-to-prick test vary, and in some cases, like that of our patient, these tests have been positive.¹⁰ We therefore posit that the symptoms described were the result of a systemic allergic reaction to the shiitake mushroom.

This is the first case of shiitake dermatitis to be reported in Spain, and it yielded a positive scratch test result. We believe that this case supports the hypothesis that this dermatitis is caused by a delayed hypersensitivity reaction and that cutaneous hypersensitivity tests could therefore be useful tools for diagnosing the condition and determining its exact pathogenesis, which remains unclear.

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Comment on: Nonsexually Transmitted Acute Ulcer of the Vulva Associated With Influenza A Virus Infection[☆]

Úlcera vulvar aguda de transmisión no sexual asociada a infección por virus influenza A

To the Editor:

We read with great interest the letter published by Esteve-Martínez et al¹ on nonsexually transmitted acute ulcer of the vulva, or *ulcus vulvae acutum*, associated with influenza A virus infection, and would like to congratulate the authors on their concise and thorough review of this condition.

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We agree with their conclusion that it is important to be familiar with this type of vulvar ulcer and its association with influenza A virus infection,^{2,3} and would like to draw readers' attention to fact that this ulcer can also occur in elderly patients, regardless of whether or not they report having had sexual relationships. One interesting case we saw recently was that of a 20-year-old woman with necrotizing genital ulcers that had required debridement in the gynecology department at our hospital.

The patient, with no relevant past history, reported that she had experienced fever and generalized joint pain 11 days before being seen in the dermatology department. She had been prescribed treatment with antipyretic medication and a single dose of oral levofloxacin by her primary care physician. Four hours after ingestion, the patient noted vulvar discomfort and an inflamed area. In the next 24 hours, she also developed pharyngeal discomfort and a worsening general state of health, leading her to visit the emergency department, where a polymerase chain reaction (PCR) assay confirmed influenza A subtype H1N1 infection. The

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

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