incidence in areas exposed to sunlight in patients with a history of basal or squamous cell carcinoma; b) other carcinogens—frequent occurrence in areas of irradiation, erythema ab igne, or following chronic exposure to arsenic; c) immunosuppression—from treatment in a liver or heart transplant setting or rheumatic diseases; and in patients with hematologic neoplasias or infection with the human immunodeficiency virus (HIV); d) cases described in patients with congenital ectodermal dysplasia or Cowden disease; and e) oncogenic viruses—although the role of Epstein-Barr virus has not been proven.

In conclusion, we present a case of MCC located at a site of vaccination. As we have encountered no similar cases in the literature to date—even though the target population for anti-influenza vaccination overlaps extensively with those at greater risk of developing MCC (individuals aged 65 years or older and immunodepressed patients)—we believe this is a case of simple coincidence. However, the close temporal relationship could indicate that vaccination causes a local immune alteration through an unknown pathogenic mechanism that would facilitate the development of MCC patients with a predisposition to the disease.

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Conflicts of Interest
The authors declare no conflicts of interest.

References

Eruptive Clear Cell Acanthoma

V. Morillo, P. Manrique, I. Zabalza, and J.L. Artola

To the Editor:
Clear cell acanthoma (CCA) was described by Degos et al.1 in 1962. They suggested that this was a benign epithelial tumor of epidermal origin rather than a reactive hyperplasia of inflammatory origin, although they questioned this affirmation 8 years later. In recent years, several authors have vindicated the inflammatory nature of this lesion, and a number of writers view the condition as a localized form of psoriasis.2,3

Eruptive Clear Cell Acanthoma

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To the Editor:
Clear cell acanthoma (CCA) was described by Degos et al.1 in 1962. They suggested that this was a benign epithelial tumor of epidermal origin rather than a reactive hyperplasia of inflammatory origin, although they questioned this affirmation 8 years later. In recent years, several authors have vindicated the inflammatory nature of this lesion, and a number of writers view the condition as a localized form of psoriasis.2,3
CCA is generally solitary and multiple forms are uncommon. However, there is one isolated description in the medical literature of an eruptive form of the disease with more than 400 lesions.

Our patient was a 32-year-old woman with no relevant history, except for the presence of a crusty plaque on the occipital zone of the scalp during adolescence that disappeared spontaneously several years ago. The family history included a father and brother with psoriasis. The patient consulted for the progressive appearance of multiple erythematous papules on her legs and buttocks over the last 20 years (Figures 1 and 2). These were of an elastic consistency, with some slightly scaly lesions. The patient mentioned occasional bleeding when wearing tight trousers. She reported the appearance of similar papules on the trunk and arms over the course of the last year.

A diagnosis of lichen myxedematosus was proposed and biopsies taken of 3 papules, all with similar histopathological findings. Each biopsy revealed a psoriasiform hyperplasia with large cells and clear cytoplasm (Figure 3) that proved intensely periodic-acid-Schiff positive. The boundary with the adjacent healthy epidermis was very well defined in all 3 lesions.

CCA is generally solitary and is most often located on the legs, although the case described by Degos et al. was on the abdomen. The first case of multiple CCA was described in 1964 and some 30 articles have been published since then. Most of the cases described were CCA patients with between 2 and 20 lesions on the legs. The lesions sometimes appeared in association with ichthyosis, varicose veins, psoriasis, or xerotic skin, although whether this is coincidental has neither been confirmed or disproved.

There is also one very interesting case of multiple CCA in a mother and her 2 children, but, as far as we are aware, this is the only familial example described.

These 30 cases include isolated incidences of patients with more than 100 lesions distributed across the trunk and limbs. However, we have only found one case similar to ours in the medical literature in which a woman had more than 400 lesions that appeared progressively.

The etiology of CCA is far from established, and even Degos recognized that the neoplastic nature of the lesions was still not fully confirmed 8 years after his initial description of the condition. Since then, many authors have suggested that CCA shows a psoriasiform reaction pattern, generally on the basis of the following 3 criteria:

1. Many of the CCA described are located within other inflammatory or reactive lesions, either stasis dermatitis, pilonidal cysts or psoriasis plaques.
2. CCA and psoriasis produce very similar histopathological findings and dermatoscopic patterns.
3. A recent study proved that the results of immunohistochemical analysis were similar to those for the normal epidermis and for psoriasis.

Figure 1. Eruptive clear cell acanthoma on the legs.

Figure 2. Multiple clear cell acanthomas on the thighs.

Figure 3. Acanthotic epidermis with clear and enlarged keratinocytes. (hematoxylin-eosin, ×20).
In view of the above it is possible to posit that our patient was simply experiencing an exceptional manifestation of familial psoriasis. The patient reported that private phototherapy sessions several years ago had led to apparent improvements in the lesion, although they had later reappeared. We initially administered broad-band UVB phototherapy with no response. We then proposed PUVA treatment, but the patient has so far refused any further treatment.

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Conflicts of Interest
The authors declare no conflicts of interest.

Allergic Contact Dermatitis to Hydrocortisone as a Complication of Tattoo Care
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To the Editor:
Many complications have been described in the context of tattooing, including various types of infection, the appearance of tumors, granulomatous reactions, and contact allergy.1 Allergic contact dermatitis (ACD) has been described in these cases in relation to some of the pigments used, especially the red color. However, the person with the tattoo may also use many products to care for the tattoo—corticosteroids, antibiotics, healing or antiseptic ointments—that may provoke ACD. We describe 2 cases of ACD from hydrocortisone use in patients who applied an ointment containing hydrocortisone recommended by the tattooist for the localized care of their tattoos.

The first patient was a 21-year-old man who attended the emergency department with severe eczema on the left leg. The lesions were located around a permanent black tattoo created 10 days previously. The patient stated the tattoo was to be completed in 2 sessions and that the upper part was incomplete (Figure 1A). He applied Terra Cortril ointment (hydrocortisone and oxytetracycline in petroleum jelly as excipient; Farmasierra Laboratorios) as recommended by the tattooist following the tattoo session, and the skin lesions appeared a week later. The emergency department prescribed Diprogenta cream (betamethasone and gentamicin; Shering Plough) and Dexa Tavegil (dexamethasone and clemastine; Novartis Consumer Health) and the lesions healed. Despite the recommendations, 2 months later the patient decided to complete the tattoo and apply Terra Cortril ointment, causing the skin lesions to return 2 days later (Figure 1B). Treatment with Diprogenta once more produced a good clinical response. Patch testing for contact dermatitis was performed with the Spanish Group for Research Into Dermatitis and Skin Allergies (GEIDAC) standard battery—showing a

References