include defibrillator pocket infections, extrusion of the implant, and allergic contact dermatitis. Most cases of allergic contact dermatitis are due to the metal and plastic components of the defibrillator, most frequently titanium, epoxy resins, and polyurethane components.[3]

In 1981 Gensch and Schmitt[4] described the first case of RTE. Since then, 22 other cases have been published of allergic contact dermatitis with this cutaneous pattern, characterized by poorly delimited erythematous plaques over the defibrillator implant site[5] and by a histological finding of telangiectases in the superficial dermis.[2,5] In this disorder, patch testing fails to identify any relevant allergen.[6-8] Several possible pathogenic mechanisms have been suggested, including mechanical obstruction of venous flow, formation of electromagnetic fields, and autonomic deregulation.[8-10]

Of particular interest in our case was the location of the plaque, which was not over the implant site, as is most common. To date, only in isolated cases has the lesion appeared near the implant site.[5,8] Moreover, in our patient the lesion disappeared spontaneously a few months after its appearance. While to the naked eye no vesiculation could be observed, histology showed a slight spongiosis, but with other findings consistent with a diagnosis of RTE. The patch tests were positive for beryllium and thiomersal, but according to the technical department of Medtronic, beryllium is not a component of the defibrillator that comes into contact with tissues. We therefore believe that positivity to beryllium was not relevant in the development of our patient's skin condition.

The pathogenesis of RTE remains unknown. Further studies are needed to determine the exact role of various factors in this condition and the possible mechanisms that lead to spontaneous resolution.

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Ocular Syphilis: A Rare Presentation of Secondary Syphilis in an Immunocompetent Patient

Sífilis Ocular: Una Presentación Inusual de Sífilis Secundaria en un Paciente Inmunocompetente

To the Editor:

Syphilis is a sexually transmitted disease that can affect a number of organs, including the eye. First described by Ygersheimer in 1918,[1] ocular syphilis is an unusual manifestation of syphilis, which the Spanish medical community needs to be aware of due to the growing number of cases of syphilis in Spain in recent years. This increase in the incidence of syphilis could lead to a rise in the number of cases with atypical presentation or with neurological complications observed in routine practice, as in the case we describe below.[2]

The patient was a 34-year-old white man with no past history of interest who consulted for a 3-month history of blurred vision and loss of visual acuity in both eyes. The symptoms appeared after he returned to Spain from Brazil, where he had lived for a year for work-related reasons. A preliminary eye examination confirmed reduced visual acuity and vitreitis in both eyes. Treatment was initiated with 60 mg/d oral prednisone. A further examination of the fundus 2 weeks later revealed a yellowish placoid lesion in the superior temporal arcade of the left eye (Figure

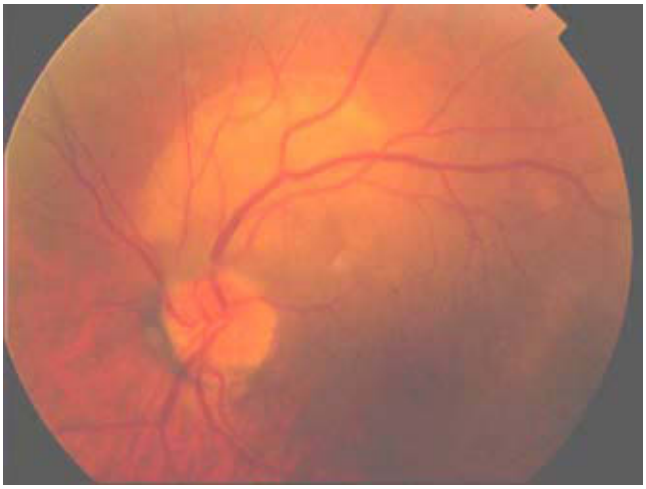

Figure 1 Acute syphilitic posterior placoid chorioretinitis.

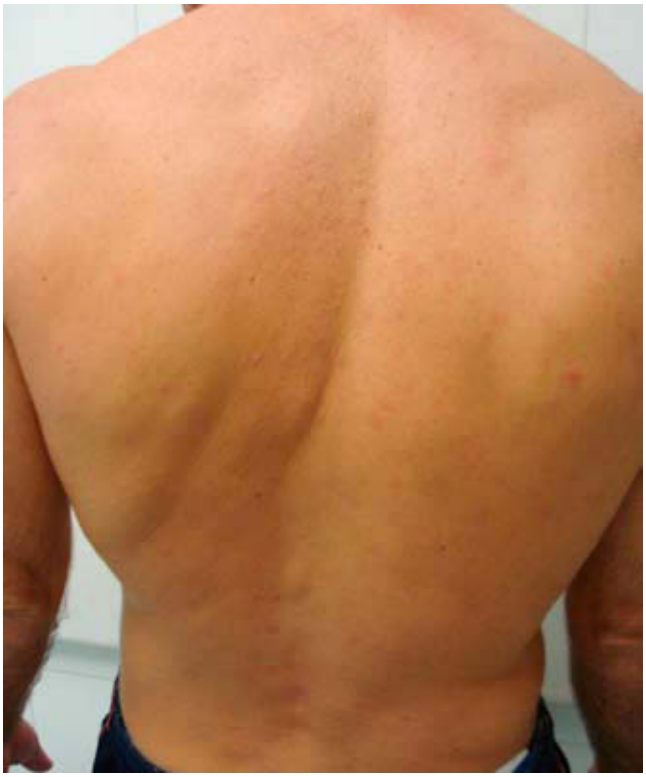

Figure 2 Pink maculopapular rash on the trunk.

1). The patient also reported a rash that he attributed to the corticosteroid treatment. A clinical suspicion of toxic dermatosis led us to consult the dermatology department. Physical examination revealed a pink, nonpruritic, maculopapular rash, mainly affecting the trunk (Figure 2). There was no evidence of either palmoplantar or mucosal involvement or of any other systemic symptoms. The patient did not recall ever having lesions in the genital area. Serology tests showed the nontreponemal rapid plasma reagin (RPR) to be positive at a titer of 1/64. *Treponema pallidum*-specific immunoglobulin (Ig) G and treponemal (hemagglutination) tests were also positive. Neurological examination was normal, and there were no significant findings for other tests (serologies for hepatitis B and C viruses, human immunodeficiency virus (HIV), toxoplasma, and cytomegalovirus, Mantoux, and chest x-ray). The patient was diagnosed with ocular syphilis. He refused both admission to hospital and a lumbar puncture, and so was prescribed 2.4 million units of intramuscular procaine penicillin once a day and 500 mg of probenecid orally 4 times a day for 14 days. He responded favorably and the ocular and skin alterations resolved completely. A month after concluding treatment, the patient's RPR serology titer—which continues being monitored—was 1/16.

Ophthalmologic manifestations of syphilis are very variable. They typically appear during secondary syphilis and can affect any segment of the eyeball.[2] Although scleritis and uveitis are the most frequent forms of presentation, keratitis and conjunctivitis may also be observed.[3] Ocular disorders are more common in patients with syphilis and HIV, and the higher risk of bilateral involvement and of extension to the posterior pole means that these patients ultimately run a serious risk of vision loss.[4-6] The acute syphilitic posterior placoid chorioretinitis presented by our patient is a recently described ocular manifestation that is very rare in HIV-negative patients.[7,8]

A diagnosis of ocular syphilis is usually established on the basis of positive treponemal or specific serologies and compatible signs and symptoms. However, nontreponemal tests (such as the RPR or the Venereal Disease Research Laboratory tests) are not sufficiently sensitive in later syphilis stages when ocular manifestations are common.[4] Therapeutic management should be the same as for neurosyphilis, that is, a study of the cerebrospinal fluid obtained via lumbar puncture, and treatment with intravenous penicillin G or intramuscular procaine penicillin plus oral probenecid for 2 weeks. Oral steroids administered at low doses may prevent a Jarisch-Herxheimer reaction, which could have serious consequences in a patient receiving treatment for ocular syphilis—triggering, for example, rapid vision loss. In such patients, the inflammatory lesions in the eye intensify, both in the anterior pole and in the vitreous, retina, and choroid.

For patients allergic to penicillin, prior desensitization is recommended as the treatment of choice.[4,9]

The dermatologist potentially plays an important role in diagnosing ocular syphilis, which should be suspected in the presence of a skin rash in patients with painful red eye, vision loss, and headache. It is important to be aware of this clinical picture in order to associate the cutaneous and ocular manifestations as part of a single systemic disease, given that early treatment often leads to full clinical recovery and prevents irreversible loss of vision.[4,5]

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Erlotinib-induced Acneiform Rash Not Affecting Previously Irradiated Skin

Erupción Acneiforme Onducida por Erlotinib que Respeta el Área de Piel Previamente Irradiada

To the Editor:

Erlotinib is a tyrosine kinase inhibitor that acts by blocking the activity of the epidermal growth factor receptor (EGFR). This receptor is frequently overexpressed and mutated in many solid tumors, and is also abundant in the basal cells of the epidermis and in follicular keratinocytes, where it contributes to the differentiation and development of the hair follicle.[1] Currently, erlotinib is indicated for the treatment of non-small cell lung cancer and pancreatic cancer, and is being investigated as a treatment option in cancers of the head and neck, ovary and kidney. Adverse skin reactions are the most frequent adverse effect of erlotinib. Among these reactions, acneiform rash is a dose-dependent response that has been observed in most of the patients treated with erlotinib.[2] The pathogenesis of acneiform rash is unknown, although it may be related to follicular hyperkeratosis, plugging and obstruction of the follicular ostium, and an altered hair growth cycle, accompanied by an intense inflammatory response.[1] We describe the case of a patient with laryngeal cancer receiving treatment with erlotinib, who developed an acneiform rash that spared previously irradiated skin.

The patient was a 46-year-old man with moderately differentiated squamous cell carcinoma of the glottic larynx, with supraglottic extension (pT3N1M0), who underwent total laryngectomy and cervical lymphadenectomy. Three months after surgery he received radiation therapy to the resection bed and cervical chains at a dose of 50 Gy over 6 weeks. Two months after the final radiation therapy session the patient began treatment with erlotinib (150 mg/d orally). Ten days after beginning drug therapy, numerous papules and confluent pustules appeared that were distributed on the face, trunk, and arms. Surprisingly, the rash spared 2 rectangular areas located on the anterior and posterior area of the neck and upper trunk, which had been included within the fields of radiation therapy (Figure 1). Biopsy of the skin affected by the rash demonstrated acute superficial folliculitis (Figure 2), whereas biopsy of the irradiated area only demonstrated some discrete perivascular infiltrates formed predominantly of lymphocytes. The patient was treated with oral doxycycline (100 mg/d). After 2 months, the oncologists decided to suspend the treatment with erlotinib due to inefficacy. The skin lesions gradually improved and had completely disappeared by 3 months.

In the case described, the type of lesions and the time of onset were similar to those found in most patients who suffer this adverse skin reaction caused by treatment with erlotinib.[1] The main interest of the case presented is that the lesions spared previously irradiated skin. To date, very few cases have been reported of EGFR-inhibitor-associated acneiform reactions that have spared irradiated skin,[3-8] and erlotinib was involved only in 3 of those cases.[6-8]

The pathogenesis of this event is unknown. One theory suggests that radiation therapy causes atrophy of the sebaceous glands, which would explain the lack of lesions in the irradiated area.[5] Our case does not support this hypothesis, because pilosebaceous units were observed in the irradiated area.

The effects of radiation therapy vary according to the time since the treatment. During the first 3 weeks, radiation therapy leads to increases in basal layer proliferation and in the mitotic index. Several weeks after irradiation there is a local reduction in the drug effect, due to either a progressive loss of endothelial cells and the drug not reaching the irradiated area, or to a modification of epidermal sensitivity to EGFR inhibitors.[3] There are reports of cases in which following the concomitant administration of an EGFR inhibitor during the course of radiation therapy