iron-deficiency anemia has also been described.[7,8] However, all of these presumed associations were based on individual cases and not on systematic study, and taking into account that a large study conducted in South Africa found pigmented fungiform papillae in 6% of males and 8% of women,[2] it is probable that they were merely coincidental. In a more recent study, 30% of black women and 25% of black men had pigmented fungiform papillae.[4]

From a clinical point of view, pigmented fungiform papillae usually develop in the second or third decade of life,[4] though they may begin in childhood. The condition has been observed in black and Japanese individuals,[8] and in Australian aborigines[6] and Indians.[6] Its incidence in those races is unknown but is considered substantially lower than in the black race.[4,5,7,8]

The pathogenesis of pigmented fungiform papillae is unknown. Based on the presence of pigmented fungiform papillae in a mother and daughter, Werchniack et al[9] suggested autosomal dominant inheritance; however, this had not been previously described or corroborated in other articles. The reason for the abnormalities being limited to the fungiform papillae also remains unknown. The histological features of pigmented fungiform papillae include numerous melanophages in the lamina propria of the papillae with no inflammatory infiltrate.[4,9] The pigment located within the melanophages stains positive for melanin with Fontana-Masson and negative for iron with Prussian blue.[9] The acquired nature of the lesions and the presence of melanophages suggests a transient period of inflammation, but the lack of inflammatory infiltrates is a histological marker of the condition.[9]

The differential diagnosis should include other causes of pigmentation of the oral mucosa such as hemochromatosis, pernicious anemia, amalgam tattoo, or Addison disease. However, a clear diagnosis can be reached in all those disorders on the basis either of the distribution and clinical characteristics of the pigmentation or the accompanying manifestations.

No effective treatment of pigmented fungiform papillae has been described,[9] although in 1 case associated with iron-deficiency anemia a moderate reduction in pigmentation was reported after treatment of the anemia.[7]

We describe the first case of pigmented fungiform papillae in an indigenous South American woman and we believe that this condition may be observed in all intensely pigmented races. Given increasing migration into Europe, more cases will be seen; it is important to recognize pigmented fungiform papillae of the tongue to avoid incorrect diagnoses and avoid unnecessary additional tests.[4,10]

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Delayed Foreign Body Reaction to Steel Wire Suture Resembling Basal Cell Carcinoma☆

Reacción retardada a cuerpo extraño por alambre de acero inoxidable simulando un carcinoma basocelular

To the Editor:

A foreign body is any live or inanimate material introduced in the human body, and the body responds by using its mechanisms of defense. Although a broad definition would also include microorganisms that elicit an immune response, foreign bodies are usually considered to be inorganic compounds or high-molecular-weight organic materials that resist destruction by inflammatory cells.[1] These substances can enter iatrogenically during surgical procedures, as is the case with foreign body reactions to suture material.[2]

We describe an 87-year-old man with a history of prostate cancer, atrial fibrillation, hypertension, and chronic bronchitis who had undergone surgery 30 years earlier for a malignant neoplastic process classified by the hospital at the time as nasal natural killer lymphoma; no further information was available. In March 2010 the patient consulted for an excrescent mass from 5 months previously that was present on the nasal bridge, on

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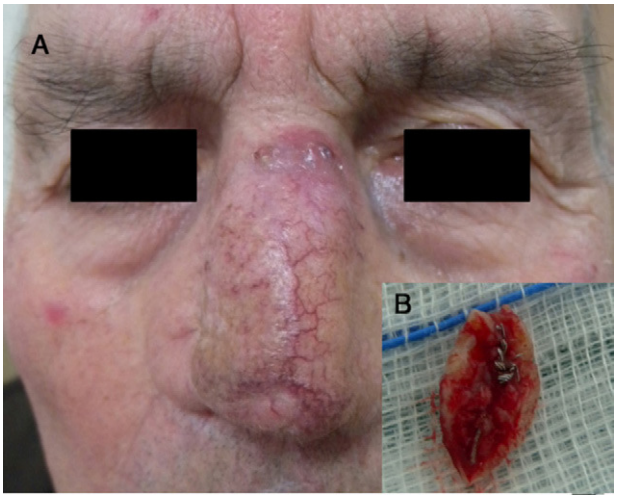

Figure 1 A, pearly papule of 1.5 cm in diameter over the previous scar. B, surgical specimen containing the steel suture wire.

top of an old scar. The examination revealed an erythematous, pearly plaque with a maximum diameter of 1.5 cm adherent to the deep layer (Fig. 1), and dermoscopy showed spider telangiectasia and grayish-blue areas.

Because basal cell carcinoma was suspected, the lesion was biopsied to rule out recurrence of the lymphoproliferative process, at which time a stainless steel suture wire was found (Fig. 1).

On further questioning, the patient reported that a bone autograft from the hip had been used in one of the previous nasal reconstruction operations. The x-ray showed other suture wires used to fix an abnormal bone structure that corresponded to the hip graft used for nasal reconstruction (Fig. 2).

Histology revealed the presence of a diffuse inflammatory infiltrate in the dermis, composed predominantly of plasma cells, lymphocytes, and occasional multinucleated giant cells (Fig. 3). No epidermal involvement or

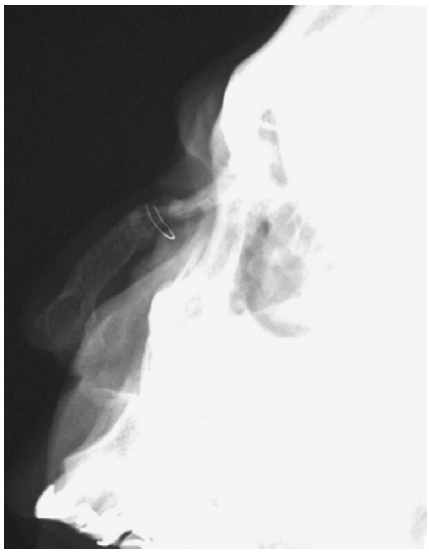

Figure 2 Autograft radiography. Radiopaque suture wire and hip graft used to replace the normal nasal anatomy.

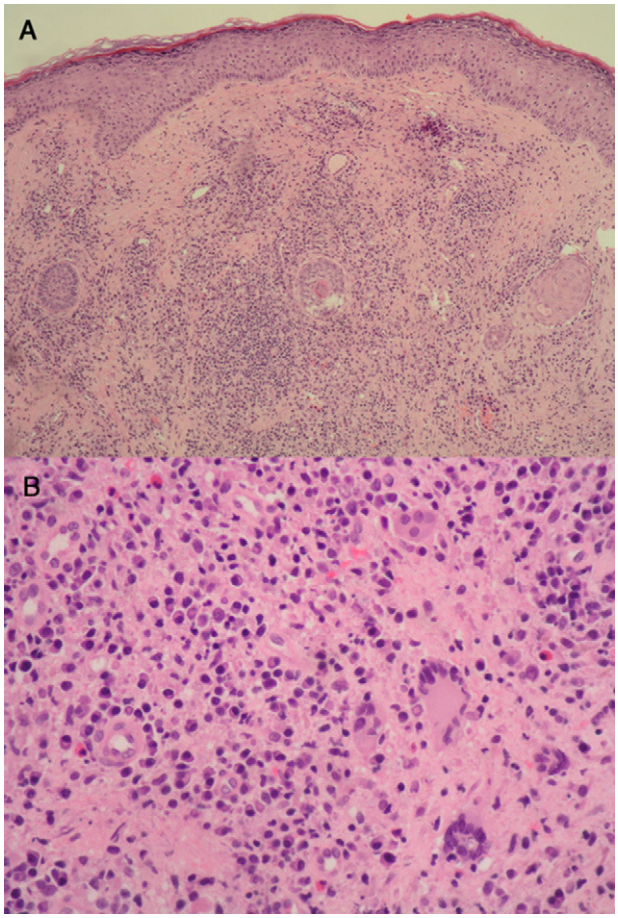

Figure 3 A, diffuse inflammatory infiltrate in the dermis. Epidermis with no significant histologic abnormalities (hematoxylin-eosin, original magnification, x25). B, lymphoplasmacytic inflammatory infiltrate containing isolated multinucleated giant cells (hematoxylin-eosin, original magnification, x200).

well-formed granulomas were observed. Lymphoid cells showed no atypia, and immunohistochemistry ruled out any recurrence of a lymphoid neoplasm. Removal of the wire and direct suture during the biopsy resolved the symptoms.

Stainless steel is sufficiently strong, flexible, ductile, and biocompatible to be used in most maxillofacial implants.[3] The material is also inexpensive and easy to handle and, therefore, has been frequently used in needles, wires, and plates for reconstructive surgery of the facial region. In implants, it has now been replaced with other materials, such as titanium.[4]

Foreign body reaction to stainless steel implants is rare. Deterioration of the wire used in sternotomy reconstruction has been reported, however.[5] In these cases, the reaction appeared in the months after surgery and the patients consulted for atypical chest pain.

The appearance of a reaction 30 years after the wire was implanted is also rare, and cannot be explained by the patient's history, as there were no injuries or recent procedures in the area. Moreover, the patient did not wear glasses or use an oxygen mask. In 2006 Surov et al[6] described a similar case of delayed reaction in an 84-year-old man with a World War II grenade injury 60 years previously who developed a mass in the

area caused by a foreign body reaction to steel fragments from the weapon.

Our letter describes a new case of foreign body reaction to stainless steel wire 3 decades after implantation. In this patient the lesion resembled basal cell carcinoma. This case report is unusual in that the condition presented 30 years after implantation with no previous triggering injury. Foreign body reaction should be included in the differential diagnosis of any skin process that develops over a surgical scar, even if the process takes place many years after the operation.

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Embolia Cutis Medicamentosa (Nicolau Syndrome) After Glatiramer Acetate Injection☆

Embolia cutis medicamentosa (síndrome de Nicolau) tras inyección de acetato de glatirámero

To the Editor:

Injection-site reactions are some of the most common complications in subcutaneously administered treatments.

We report the case of a 31-year-old woman with no known drug allergies, a history of asthma and hidradenitis suppurativa, neither of which was being treated, and relapsing-remitting multiple sclerosis. For four-and-a-half years, the patient had been receiving treatment with daily subcutaneous injections of 20 mg of glatiramer acetate (GA, Copolymer-1, Copaxone, Sanofi-Aventis, Barcelona, Spain).

She visited the emergency department due to acute pain in the left buttock after an injection of glatiramer acetate; the patient had not experienced the pain with previous injections. There was a whitish plaque at the injection site that became erythematous and necrotic over the following 5 days.

Physical examination (Fig. 1) showed a reddish-gray, livedoid plaque on the left buttock, measuring approximately 3 cm, with geographic borders, a necrotic center, and a more intensely erythematous-violaceous border. On the caudal part of the lesion there was a deep, round, adherent scab measuring approximately 8 mm in diameter.

Questioning of the patient revealed that she complied with the injection protocol: the same injection site was not used in less than a week, the drug was left at room temperature 20 minutes before use, and the needle was placed in the correct position. Furthermore, the patient had continued to inject the treatment in the thighs and abdomen in the following days and no lesions had appeared at those sites. She reported a similar event in the same buttock a year earlier that had resolved without treatment and left an area of residual hypopigmentation.

A biopsy was performed of the peripheral area of the skin lesion on the buttock and showed a partially necrotic epidermis with coagulative necrosis of the dermal collagen, fat necrosis, and some fibrin clots in the small blood vessels (Fig. 2). Analyses, including a complete blood count, biochemistry with liver and kidney function tests, immunoglobulins, complement, antibodies to extractable nuclear antigens, antinuclear and anticardiolipin antibodies, and coagulation studies showed no relevant abnormalities.

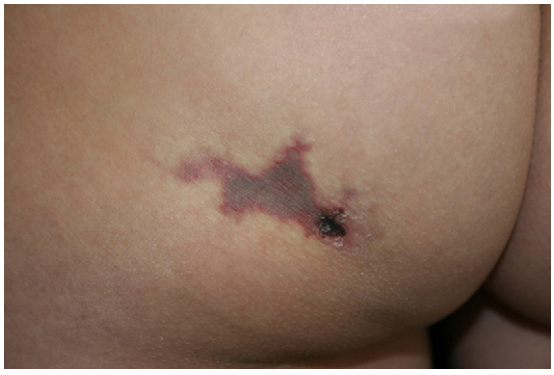

Figure 1 Violaceous livedoid plaque, with erythematous borders and a central scab located on the left buttock.

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