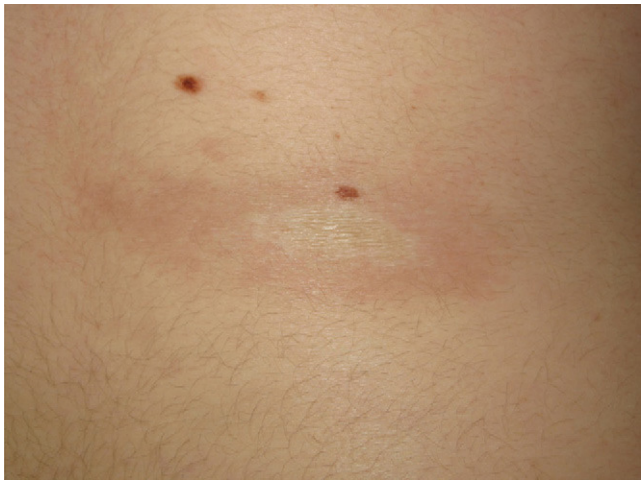

Figure 2 Morphea-like plaque, detailed view.

autoimmune manifestations such as vitiligo and morphea after treatment with interferon are considered likely to achieve a good response, defined as increased disease-free survival.[7,8]

In the last 5 years, we have treated 408 patients with melanoma at Hospital Carlos Haya in Malaga, Spain. Of the 35 who received interferon, 2 developed vitiligo. The first involved a 40-year-old patient who died as a consequence of melanoma 3 years after diagnosis; the second was a 60-year-old patient who remains disease-free 6 years after treatment with interferon. Therefore, our experience does not enable us to corroborate or refute the favorable outcome of patients with vitiligo accompanied by melanoma treated with interferon.

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Lupus Vulgaris Caused by *Mycobacterium bovis*☆

Lupus vulgar causado por *Mycobacterium bovis*

To the Editor:

Tuberculosis is a global pandemic. According to the World Health Organization, a third of the world's population is infected with tuberculosis bacilli. Cutaneous tuberculosis accounts for approximately 1.5% of all extrapulmonary tuberculosis cases,[1] with lupus vulgaris being the most common subtype in industrialized countries.[2] *Mycobacterium tuberculosis* is the main causative agent of lupus vulgaris. However, the disease can also be caused by *Mycobacterium bovis* or bacillus Calmette-Guérin (BCG),[3] an attenuated strain of *M bovis* used in vaccines. We present a case of lupus vulgaris caused by *M bovis*, currently a very rare entity.

The patient was a 78-year-old man, originally from a town in the province of Burgos (Spain), with no relevant personal history. He presented with a skin lesion on the left axilla that had begun to itch 4 years earlier but had first appeared during childhood. Physical examination revealed a brownish erythematous plaque measuring 9.5 × 6 cm on the posterior aspect of the left axilla, with an atrophic center, raised borders, and telangiectasias (Fig. 1). Classic apple-jelly nodules were observed on diascopy. Physical examination revealed a palpable lymph node in the ipsilateral axilla but was otherwise normal. Skin biopsy revealed a dermal infiltrate composed of confluent nonnecrotizing epithelioid granulomas with lymphocytic coronas and multinucleated giant cells (Fig. 2A and B).

Ziehl-Neelsen staining was positive for acid-fast bacilli and skin culture was positive for *M bovis*. Axillary ultrasound revealed an inflammatory lymph node that resolved with treatment. A chest radiograph was normal and a Mantoux test was positive (1 cm). The patient was diagnosed

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Table 1 Cases of Lupus Vulgaris Caused by *Mycobacterium bovis* Reported in the Past 20 Years.

Reference	Sex (Age, y)	Environment	Clinical Course	History of Tuberculosis
Meyer et al.[3]	Female (69)	Bavaria, southern Germany	10 years	Pulmonary tuberculosis during childhood
Tar et al.[7]	Female (33)	Rural area (in contact with livestock)	20 years	None
Flohr et al.[9]	Male (67)	City of Nottingham	Since childhood	None
Twomey et al.[10]	Female (25)	Veterinary practice	9 weeks	None

with tuberculous lymphadenopathy and lupus vulgaris due to *M bovis*. He was prescribed a 2-month regimen of tuberculostatic triple-drug therapy with isoniazid, rifampicin, and pyrazinamide, followed by 4 months of therapy with isoniazid and rifampicin. At the end of treatment, an asymptomatic, cicatricial, anetodermic lesion remained.

Lupus vulgaris is a chronic, progressive form of cutaneous tuberculosis. It is usually caused by *M tuberculosis*, but cases caused by *M bovis* and BCG have also been reported. It generally affects previously sensitized patients, who have a delayed, strongly positive hypersensitivity reaction to the tuberculin skin test. The skin lesions are caused by hematogenous or lymphatic dissemination from, or contiguity with, a primary focus in another organ.[3] Occasionally, lupus vulgaris develops after vaccination with BCG or direct inoculation.[4,5] An important feature of the disease is that it can lead to the formation of scars, which may cause contractures and deformities. In long-standing cases, lupus vulgaris may progress to squamous cell carcinoma.[1]

The clinical differential diagnosis for lupus vulgaris includes sarcoidosis, lymphocytoma, discoid lupus erythematosus, tertiary syphilis, deep fungal infection, and lupoid leishmaniasis. The histologic differential diagnosis includes sarcoidosis, tuberculoid leprosy, deep fungal infection, and foreign body reaction.[6]

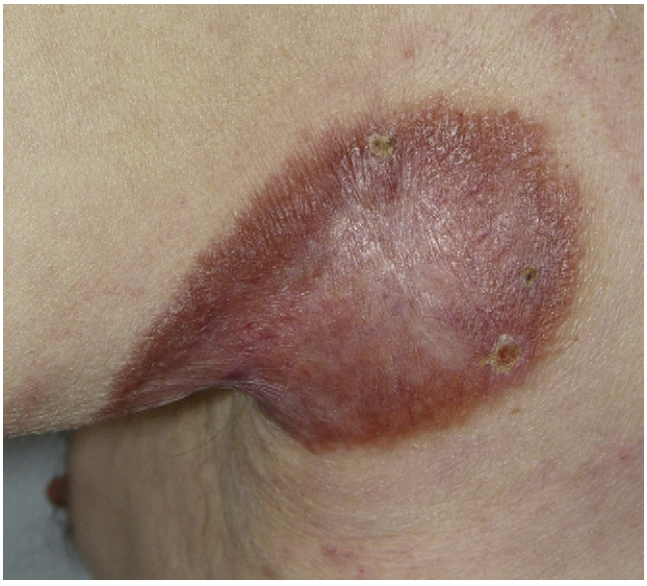

Figure 1 Brownish erythematous plaque located on the posterior aspect of the left axilla, with an atrophic center, raised borders, and telangiectasias.

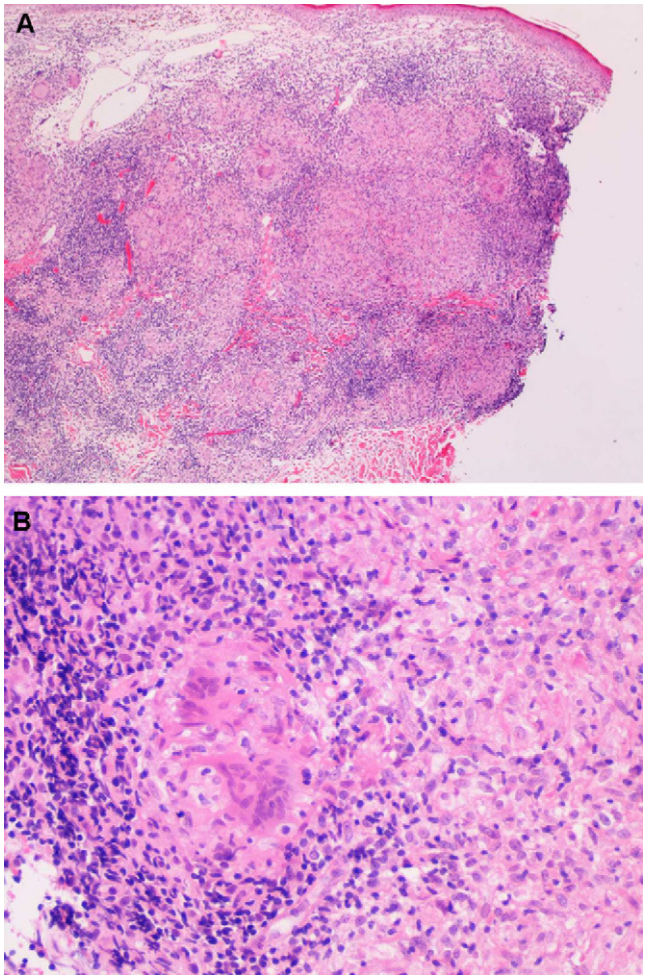

Figure 2 A, Low-power view of the biopsy specimen, showing a dense granulomatous infiltrate in the middle dermis that becomes less severe in the deep dermis (hematoxylin-eosin, original magnification ×40). B, Detail of a noncaseating granuloma with lymphocytic corona and multinucleated giant cells (hematoxylin-eosin, original magnification ×200).

Definitive diagnosis is established by culture, although a positive result is obtained in less than 10% of cases. Ziehl-Neelsen staining of biopsy specimens can also yield false negative results due to the paucibacillary nature of lupus vulgaris. In such cases, mycobacterial DNA can be identified by means of polymerase chain reaction.[2,6]

Lupus vulgaris caused by *M bovis* is currently very rare. Though clinically indistinguishable from lupus vulgaris caused by *M tuberculosis*,[7] lupus vulgaris due to *M bovis*

usually appears after contact with infected animals or ingestion of contaminated milk; dissemination from a pulmonary focus is less common. *Mycobacterium bovis* is a member of the *M tuberculosis* complex, which includes the species *M tuberculosis*, *M bovis*, *Mycobacterium africanum*, *Mycobacterium microti*, and *Mycobacterium canetti*,[8] the causative agents of tuberculosis in humans and animals.

Our review of the literature identified 4 cases of lupus vulgaris caused by *M bovis* in the past 20 years (Table 1). In 2005, Meyer et al.[3] described the case of a 69 year-old woman who had contracted pulmonary tuberculosis as a child. In 2009, Tar et al.[7] and Flohr et al.[9] presented cases of patients suspected of having been infected with *M bovis* through contact with cattle or by ingesting unpasteurized milk. In 2010, Twomey et al.[10] described a case of occupational infection with *M bovis* in a veterinarian.

As a result of efforts to eradicate bovine tuberculosis and due to the prevalence of milk pasteurization,[9] lupus vulgaris caused by *M bovis* is now very rare. Nevertheless, it should be considered, primarily in patients who live in rural areas or work in high-risk occupations such as livestock rearing or veterinary medicine.[10]

In conclusion, we present a case of lupus vulgaris caused by *M bovis*, now regarded as a very rare pathogen. This case involved a considerable delay in diagnosis, as the lesion had been present since childhood.

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Systemic Follicular Lymphoma With Cutaneous Manifestations and Exclusively Cutaneous Recurrence☆

Linfoma folicular sistémico con afectación cutánea y recidiva únicamente cutánea

To the Editor:

Follicular lymphoma (FL) constitutes approximately 30% of all non-Hodgkin lymphomas in Western countries. Clinical presentation is typically in the form of lymphadenopathy, hepatomegaly, splenomegaly and bone marrow infiltration.[1,2] Extranodal involvement is less frequent than in large B-cell lymphoma[2] and does not appear to affect prognosis; this is not the case with B-cell lymphoma.

We report the case of a patient diagnosed with FL who developed skin nodules in the course of her disease. The lymphoma recurred after treatment, but it was exclusively limited to the skin, an unusual observation in this disease.

The patient was a 54-year-old woman who was referred to the hematology department in August 2004 for thrombocytopenia detected during a routine blood analysis (platelet count 70×10^9/L). Monoclonal B-cell lymphocytosis in the peripheral blood and bone marrow, enlarged subcentimeter abdominal lymph nodes and splenomegaly were observed during the study. The diagnosis was chronic B-cell lymphoproliferative syndrome and the patient was offered splenectomy, which she refused at that time. At follow-up in January 2007, enlarged mediastinal and retroperitoneal lymph nodes ($\leq$5 cm in diameter) were observed, prompting the performance of diagnostic and therapeutic splenectomy. Histologic study of the spleen showed a proliferation of small lymphoid cells, with a micronodular growth pattern, predominantly in the germinal centers of the white pulp, with infiltration of the red pulp. The cells had a $CD20^+$, $Bcl2^+$, $Bcl6^+$, IgD^- $CD23^-$, cyclin $D1^-$ and $p53^-$ phenotype and a low proliferative index, findings which were consistent with the diagnosis of FL.

In March 2007, the patient was referred to the dermatology department due to the gradual appearance of

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