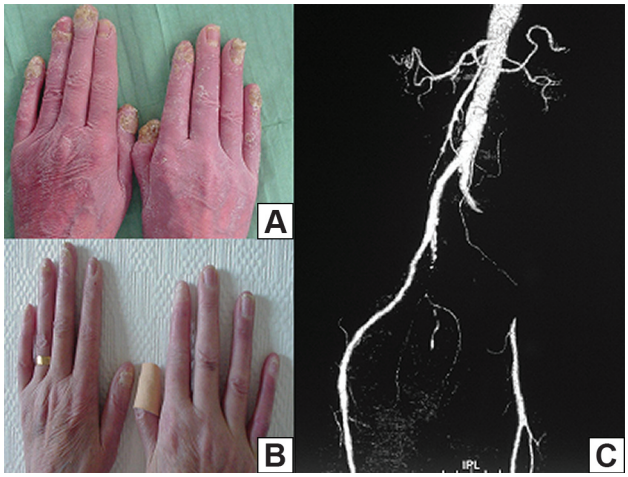

Figure1. Comparative pictures of nail lesions before the first **(A)**, and after the third **(B)** injection of Infliximab. **C)** Angioscanner showing the left primitive iliac arterial thrombosis, as the left external iliac, left common femoral, right primitive iliac, and right deep iliac thrombosis.

anti-phospholipid antibodies during the treatment can be useful. In the case of positivity, the benefit of continuing this therapy and the instauration of an appropriate prevention of arterial thrombosis should be discussed. ■

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BCG vaccine-induced lupus vulgaris

Bacille Calmette-Guérin (BCG) is an attenuated form of *Mycobacterium bovis* with which vaccination is used in many developing and developed countries on a national scale to offer protection against tuberculosis (TB) infection. It is included in the Compulsory Immunization Program (CIP) in Egypt. In 2005, Bellet and Prose classified skin complications of BCG immunization into complications in immunocompetent and immunodeficient hosts. The cutaneous complications in immunocompetent hosts were further subdivided into frequent complications (erythema, soreness, abscess, ulcer, keloid, and blister formation), and rare complications (cutaneous granulomas, BCG granuloma in Kawasaki disease, fixed drug eruption, BCG-induced lupus vulgaris (LV), and BCG infection following mesotherapy) [1]. Thus, BCG-induced LV is rare; its risk ranges from 5/1,000,000 and 1/100,000-175,000. About 60 cases have been reported in the literature [2]. It has been reported after single BCG vaccination, however, multiple BCG vaccinations seem to markedly increase the risk [3]. We report a patient with an LV-like reaction following BCG vaccine.

An 18-year-old girl presented with a painless, slowly progressively expanding, reddish, discoid, soft plaque on the left deltoid region of 10 years duration. The patient's past medical history revealed BCG vaccination on her left deltoid region 8 years prior to the onset of the lesion (in early infancy as is customary in Egypt), with no history of revaccination later. Physical examination revealed an 8 × 6 cm, indurated, reddish, scaly plaque with areas of scarring *(figure 1A)*. Diascopy of the lesion revealed yellow-brown apple-jelly nodules. There was no tenderness, anaesthesia or regional lymphadenopathy.

Histopathological examination revealed a dense dermal inflammatory infiltrate of well-developed epithelioid granulomas, with some Langhans giant cells, surrounded by a mantle of mononuclear cells. Organisms were not identified in tissue by the Ziehl-Neelsen stain. Cultures from biopsied tissue using Lowenstein-Jensen medium were negative at 8 weeks. A Mantoux test (performed with 10 TU) was strongly positive at 48 h, with an area of redness and induration measuring 15 mm in diameter, vesicle formation and subsequent ulceration (*i.e.* necrotizing reaction indicating TB infection). Chest X-ray and routine laboratory investigations including complete blood count and erythrocyte sedimentation rate were normal.

In view of the previous BCG vaccination in the same region of skin lesion and its clincohistopathological features, the diagnosis of post-BCG vaccine LV was made.

Our patient received triple therapy with rifampicin 600 mg, isoniazid 300 mg, and pyrazinamide 2,000 mg daily for 2 months, followed by rifampicin and isoniazid for 6 months, with good response, leaving a thin atrophic scar *(figure 1B)*.

Previous studies reported that the time interval between vaccination and the onset of BCG-induced LV ranges from a few months to 4 years, with an average of 1 year [2, 4]. In our case, time interval was 8 years. This is, to our knowledge, the first report of such a long interval.

The factors that may be responsible for the development of post-BCG LV include inherent susceptibility, the virulence of BCG organism, the amount of the inoculum and

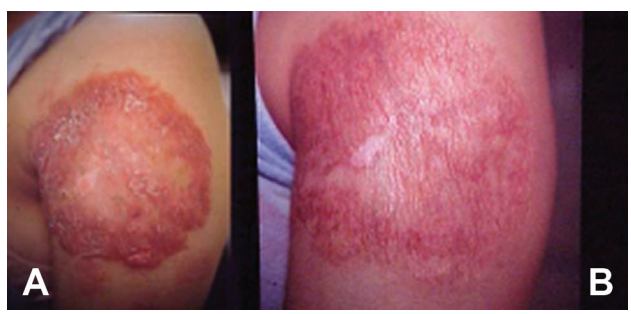

Figure 1. Vaccination site. **A)** At presentation. **B)** After 8 months of therapy.

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the inoculation technique [2]. Determining a definitive diagnosis of LV is a difficult task as no mycobacteria can usually be isolated. However, the detection of mycobacterial DNA by polymerase chain reaction (PCR) is conclusive evidence of their presence but does not prove their pathogenecity [5, 6].

In conclusion, with the increasing incidence of TB and multi-drug resistance of the organism, it is likely that BCG vaccination will be promoted. However, the use of a less virulent vaccine and proper vaccination techniques to minimize BCG complications is advised. The proper selection of those receiving the vaccines should be considered. In case of revaccination, prior tuberculin tests, as well as screening for active TB, are indicated, and vaccination should be restricted to those who are tuberculin-negative and lack active TB focus. ■

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Thrombotic thrombocytopenic purpura associated with systemic lupus erythematosus

Our patient, with mild SLE with apparent symptoms limited to the skin, developed fulminant TTP.

A 55-year-old man was seen with erythemas on his dorsal hands, feet and ears in March 1996. Over the preceding 10 years, he occasionally suffered from arthralgia, photosensitive dermatitis, and Raynaud's phenomena, and for over 5 years, from erythemas on his limbs. These were responsive to topical steroids. Painful erosions occurred in January 1996. He was subsequently admitted to our hospital. There was no relevant medical, family or drug history. Physical examination revealed tender erythematous plaques with scale and crusts on his fingers, the plantar surface of feet and his ears *(figure 1A)*. Laboratory studies showed the following elevated abnormal values: IgG 2,460 mg/dL, antinuclear antibody titer 320 × (Homo), anti-dsDNA antibody 90.2 IU/mL, anti-ssDNA antibody 149.4 IU/mL, anti-SSA antibody 55 IU/mL, anti-SSB antibody 100 IU/mL. The following were within normal limits: hemogram, hepatic and renal function, anti-phospholipid antibody, C3, C4, CH50, urinalysis and chest x-ray film. A biopsy obtained from an erythematous lesion on the finger showed hyperkeratosis, hypergranulosis and liquefaction in the epidermis, and perivascular lymphocyte infiltrate in the dermis *(figure 1B)*. We diagnosed chilblain lupus associated with systemic lupus erythematosus. He fulfilled 5 items from the 1982 American College of Rheumatology criteria for SLE. His disease activity was relatively low (SLEDAI index: 4). He was treated with oral tocophenol nicotinate and a topical steroid, with a good response for the skin symptoms.

Follow up for the next 5 years was unremarkable. On April 13, 2001, he presented no physical symptoms except brown discoid pigmentation on his fingers (SLEDAI index: 2). On April 16, he suffered from a headache, appetite loss and a sore throat. He visited the emergency room of our hospital with jaundice and petechiae on his limbs. His blood tests revealed the following abnormal values: RBC 210×10^4/uL, Hb 6.9 g/dL, WBC 8900/uL, plt 3×10^3/uL, AST 125 IU/L, ALT 42 IU/L, LDH 3035 IU/L, CK287 IU/L, T Bil 6.6 mg/dL, CRP 1.9 mg/dL, BUN 56.7 mg/dL, and Creatinin 1.9 mg/dL, and a peripheral blood smear revealed schistocytes. Direct and indirect Coomb's tests were negative. Bone marrow examination showed hypercellular marrow (NCC 25×10^4/uL). Neither the haptoglobin level nor ADAMTS-13 activity could be examined. He was admitted to the intensive care unit. Pulse therapy with intraveneous mPSL 1g/day for 3 days was initiated and plasmapheresis, under the diagnosis of TTP. He developed focal seizures with deterioration in consciousness. Despite the intensive treatments, he died of multiple organ failure 3 days later. An autopsy revealed

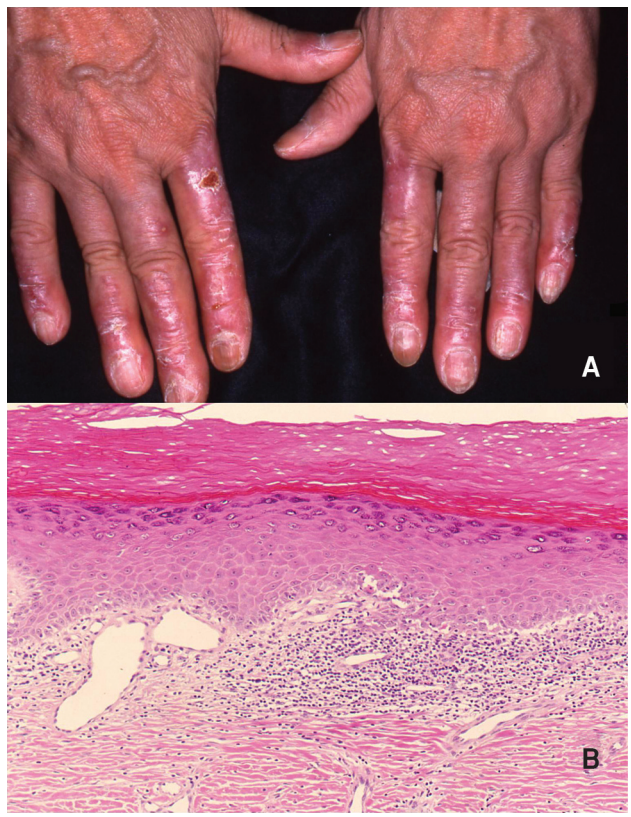

Figure 1. A) Tender erythematous plaques on fingers. **B)** Hyperkeratosis, hypergranulosis and liquefaction in epidermis, and perivascular lymphocyte infiltrate in dermis. (haematoxylin and eosin; original magnification 100 ×).

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