Radiation-induced alopecia after fluoroscopically guided interventional procedures is a form of radiodermatitis.[1,2] As occurred in our patient, exposure to a relatively low dose of radiation (3–7 Gy) can lead to transient epilation caused by damage to the actively dividing matrix cells of the anagen hair follicles.[1,3] Anagen effluvium develops within several days or weeks, and is usually followed by transient miniaturization of anagen hairs and the emergence of broken hairs as a result of the loss of follicle stem cells.[1] Furthermore, the premature catagen entry of follicles in late anagen can also trigger observable telogen shedding 2.5–4 months after exposure.[4] As occurred in our case, hair regrowth generally occurs 2–4 months after low radiation dose exposure and therefore no treatment is currently indicated. Nevertheless, permanent alopecia can be expected after radiation doses above 7 Gy.[2]

Temporary alopecia following therapeutic embolization of aneurysms, arteriovenous malformations, and tumors has been previously reported.[4–10] The complexity of posterior circulation aneurysm coiling procedures, such as those performed in our patient, has been linked to longer fluoroscopy time, increasing the risk of radiation-induced skin damage.[8] Cumulative radiation dose, intervals between sessions, total irradiated area, and variations in the incidence angle are important determinants of injury severity.[1] In our patient, the location and geometric configuration of the bald patch are consistent with prolonged radiation exposure in the same area with limited variation of the direction of application during the fluoroscopically guided interventions.

Our report emphasizes the need for awareness of the risk of alopecia originating from fluoroscopically guided techniques. While rare and probably underdiagnosed, this adverse effect may increase in incidence due to the growing use of minimally invasive endovascular procedures for the diagnosis and treatment of neurovascular disorders.

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Pediatric generalized morphea that developed at a BCG vaccination site

Morfea generalizada pediátrica de aparición en el sitio de la inyección de la vacuna BCG

To the Editor:

An 8-year-old girl was referred to our department with a linear sclerotic lesion on the upper left arm that had appeared several months earlier. The lesion had developed on the upper lateral aspect of the left arm, at the site of a Bacille Calmette-Guerin (BCG) vaccine, given more than 7 years earlier. It had gradually spread in a linear pattern and new sclerotic plaques appeared in increasing numbers over the subsequent months. Physical examination revealed a linear, slightly sclerotic plaque on the forehead (scleroderma en coup de sabre), linear pigmented, shiny plaques on the upper left arm, and sclerotic plaques on the right abdomen, left axilla, and back (Fig. 1A–C). The girl also complained of muscle weakness in the upper left arm and occasional headaches. A biopsy specimen taken from the BCG vaccination site revealed thickened collagen bundles throughout the dermis (Fig. 2). Patchy lymphocytic infiltrates and fibrosis of septal connective tissues were seen in the subcutaneous tissue. Laboratory tests revealed positive antinuclear

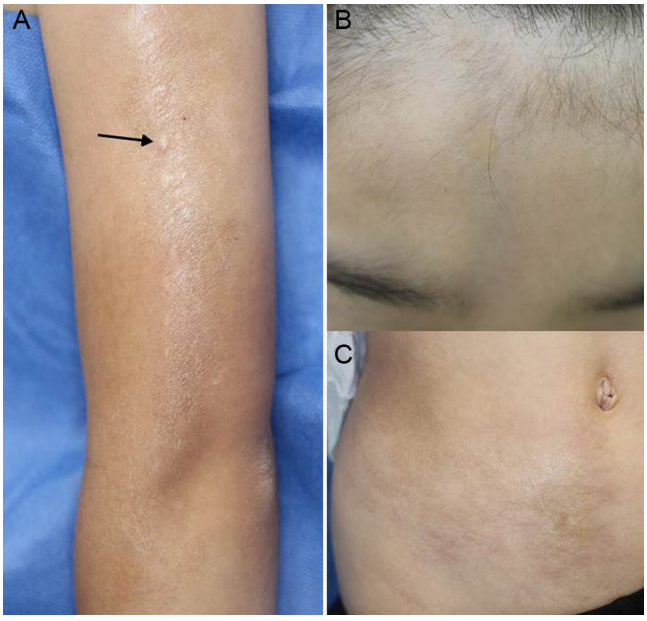

Figure 1 (A) Clinical appearance of the linear sclerotic plaque on the patient's arm. The Bacille Calmette-Guerin vaccination scar is shown by the arrow. (B) Linear sclerotic plaque on the forehead. (C) Morphea plaque on the right abdomen.

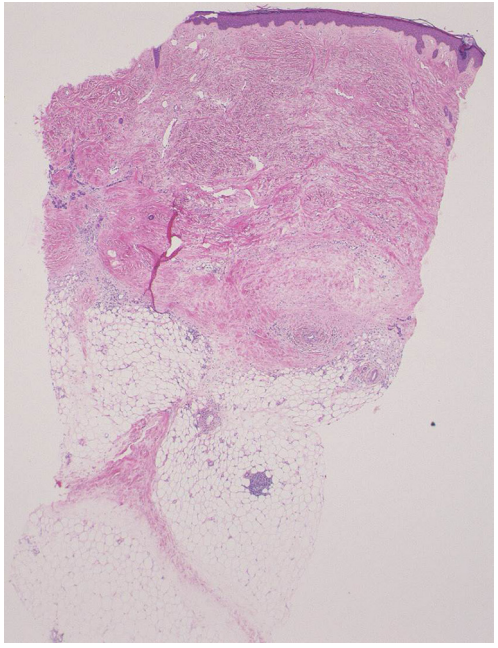

Figure 2 Histological features included thickened collagen bundles in the dermis, fibrosis of septal tissues, and patchy lymphocytic infiltrates in the subcutaneous tissues.

antibodies (1:320, speckled and nucleolar) and values within the normal range for rheumatoid factor and anti-DNA, anti-centromere, and anti-U1RNP antibodies. Liver and kidney function, serum complement levels, creatine phosphokinase, aldolase, and myoglobin were also all within normal ranges. Three-dimensional computed tomography of the scalp did not reveal any abnormal findings. The patient was initially treated with oral prednisolone (15 mg/d), followed by the addition of methotrexate (6 mg/wk), which led to satisfactory results.

Morphea is sometimes triggered by local stimuli, such as minor trauma, irradiation, vaccination, implantation of silicon prostheses, needle biopsy, laparoscopy, and drug injections; it can also arise at the site of surgical and herpes zoster scars.[1–7] The Koebner phenomenon is seen in various disorders, including morphea. The pathogenesis of this phenomenon has not yet been fully elucidated. Upon epidermal injury, several proinflammatory cytokines, such as interleukin 1, tumor necrosis factor α, and granulocyte macrophage-colony stimulating factor (GM-CSF), are released, possibly inducing further inflammation. Ueki[8] proposed a 2-step theory to explain the pathophysiology of the Koebner phenomenon. The first step, referred to as a nonspecific inflammatory step, would involve multiple environmentally induced factors such as cytokines, stress proteins, adhesion molecules, and autoantigens translocated from intracellular areas, while the second, disease-specific, step would involve disease-specific reactions mediated by T cells, B cells, autoantibodies, and immune complex deposition under the restriction of susceptible backgrounds.

Dermatological complications of BCG vaccination generally include induration and severe ulceration, but cases of granuloma annulare, abscesses, papular tuberculids, lupus vulgaris, and benign and malignant tumors have also been reported. Keloid formation at the BCG vaccination site is well known, but few cases of morphea have been reported to date.[9,10] In one of the cases, the lesions appeared on the shoulder a year after BCG vaccination, which was proposed as a possible trigger for the skin changes.[9] Another case was reported in a series of 7 cases of sclerodermatous conditions after bone marrow transplantation, but a detailed description was not provided.[10] Our patient developed linear scleroderma that started at the site of a BCG scar, without prior keloid scarring. Previous trauma may be associated with persistent inflammation and the release of various mediators such as cytokines and growth factors, or neurotransmitters from degenerative peripheral nerves.

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