

epithelioid cells in areas of degenerated collagen, are an uncommon feature that defines what has been called the sarcoid variant of necrobiosis lipoidica.<sup>9,10</sup>

## Conflicts of Interest

The authors declare that they have no conflicts of interest.

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## Can Psoriasis Produce Irreversible Alopecia?<sup>☆</sup>

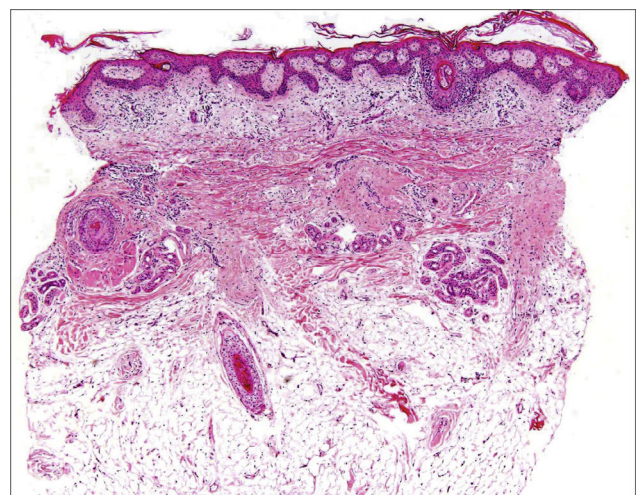


### ¿La psoriasis produce alopecia irreversible?

Dear Editor:

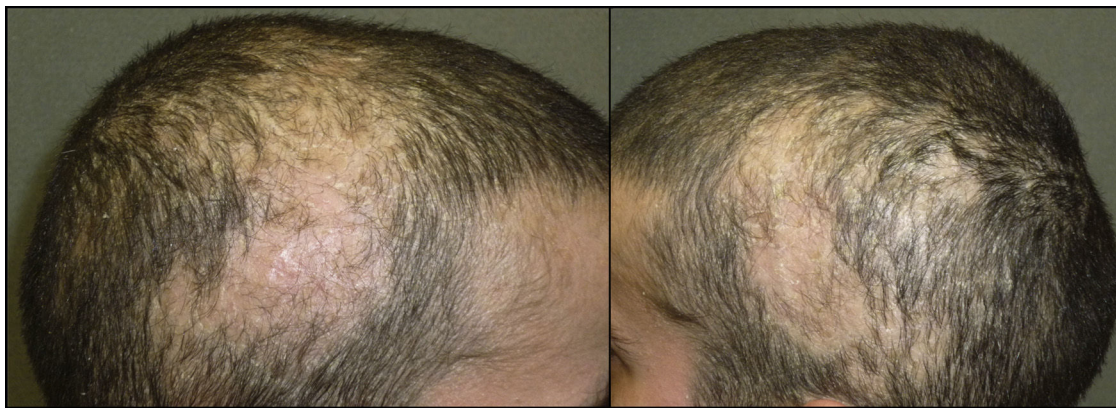
Can psoriasis cause irreversible alopecia? This was the question posed by one of our patients, a 21-year-old woman with no medical history of interest who had plaque psoriasis affecting the scalp, the upper part of the chest, and the midface area. Her condition, which had first developed more than 10 years earlier, was initially well controlled with topical drugs, but deteriorated rapidly when she was 18 years of age, causing significant psychosocial stress. Methotrexate, the first systemic treatment prescribed, was withdrawn due to poor tolerance. After that a very good response was achieved with ciclosporin, but her condition relapsed immediately when an attempt was made to reduce the dose. Ciclosporin was followed by etanercept, with minimal improvement; ustekinumab, which required frequent cycles of oral corticosteroids to control flares; and infliximab, to which she had a good response until the eighth cycle,

when her condition became progressively more severe. The scalp was the site with the most severe and extensive lesions, and hair density was significantly reduced on the psoriatic plaques. When infliximab was discontinued, the patient was admitted to hospital to facilitate more intensive topical care and to start treatment with prednisone and methotrexate 12.5 mg/wk, a regimen that achieved sig-



**Figure 1** Vertical section. Note the marked reduction in hair follicle density and how follicles have been replaced by fibrous tracts.

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**Figure 2** Bilateral parietal and temporal alopecic plaques.

nificant improvement and good tolerance. Three months after the hospitalization, inflammation and scaling on the scalp were minimal, but the patient had large alopecic plaques on the parietal and temporal scalp, cicatricial in appearance. Histologic study of tissue from the edge of one of these plaques revealed psoriasiform epidermal changes, reduced hair follicle density, patchy peri-infundibular lymphocytic inflammatory infiltrate, and fiber tracts as well as absent and atrophic sebaceous glands (Fig. 1). After excluding other possible causes of alopecia (including bacterial and fungal superinfection), a diagnosis of psoriatic scarring alopecia was made. Two years later, the bald plaques persist, although partial repopulation has occurred (Fig. 2). The patient has continued to experience psoriasis outbreaks, which some months ago required treatment with a combination of adalimumab and methotrexate, a regimen that achieved partial control of the lesions.

Scarring alopecia associated with scalp psoriasis was first described by Shuster in 1972,<sup>1</sup> but has not yet been fully accepted; major doubts require clarification. In recent editions of dermatology books, psoriatic alopecia is described as a form of nonscarring alopecia; however, some 20 cases of scarring alopecia related to psoriasis have been reported in the international literature (Table 1). The article by Runne and Kroneisen-Wiersma<sup>2</sup> is the one that comes closest to the initial descriptive study by Shuster, encompassing the various clinical forms of alopecia in psoriasis, including a cicatricial type. Psoriatic scarring alopecia is usually characterized by a circumscribed pattern of hair loss and histology showing psoriasiform changes with perifollicular, or more specifically peri-infundibular, lymphocytic infiltrate and fibrous tracts. Atrophy of the sebaceous glands was initially described as characteristic of psoriatic plaques, with or without alopecia, but other authors have subsequently related this finding more specifically with psoriatic scarring alopecia.<sup>3</sup> Also of note is the patchy follicular involvement, which would explain the subsequent hair repopulation, even when fibrosis is detected in the biopsy sample.

A more recent development is psoriatic alopecia induced by tumor necrosis factor (TNF) inhibitors. The prevalence of psoriasis as a paradoxical reaction to anti-TNF is estimated to be between 1.5% and 5%. It is considered to be particularly associated with infliximab and to frequently affect flexural

areas and the scalp. Associated alopecia is exceptional, and scarring alopecia has been reported in only 1 case.<sup>10</sup>

The etiology and pathogenesis of psoriatic scarring alopecia is not clear and several questions arise. If psoriasis causes inflammation and abnormal keratinization of the epidermis, why does it not have the same effect on follicular epithelium and alter the maturation of hair follicles with dystrophic bulbs, fine hairs and predominance of hairs in the telogen phase. If inflammation is severe, it is not surprising that inflammatory cells should invade the follicular epithelium, leading eventually to its destruction. On the other hand, if neutrophils are one of the cells primarily involved in the psoriatic process, why is folliculotropic lymphocytosis observed? Neither can we ignore the possible role of external factors, such as scratching, superinfection and even the weight exerted by the thick scales on the scalp, which may impede the normal growth and maturation of the follicle.

In conclusion, psoriatic scarring alopecia is a rare and controversial condition which some authors consider to be a separate entity and others see as a secondary alteration. How then should we answer our patient's question? Does she have irreversible scarring alopecia? We do not know, and more clinical studies with histologic correlation and longer-term follow-up are needed. For the moment, for us and our patient, her condition is a "persistent" alopecia.

### Conflicts of Interest

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**Table 1** Characteristics of Reported Cases of Scarring Alopecia Associated With Psoriasis.

Year	Authors	Cases	Psoriasiform	Fibrosis	Histology	Sebaceous Gland Atrophy	Cultures Bacteria and fungi	Follow-Up	Repopulation
1972	Shuster <sup>1</sup>	6	Yes	Yes	Yes	No	Negative	10 y	1 patient
1983	Fernandes Rodrigues et al. <sup>4</sup>	1	Yes	Yes	Lymphohistiocytic	No	Negative	—	—
1990	Wright and Messenger <sup>5</sup>	3	Yes	Yes, patchy	Peri-infundibular lymphocytic	Yes	Negative	3 mo	—
1992	Van de Kerkhof and Chang <sup>6</sup>	1	Yes	Yes	Lymphohistiocytic	No	Negative	—	—
1992	Runne and Kroneisen- Wiersma <sup>2</sup>	5	Yes	Yes, patchy	Peri-infundibular lymphocytic	Yes	Negative	—	—
1995	Kretzschmar et al. <sup>7</sup>	1	Yes	Yes	Lymphohistiocytic	No	Negative	—	—
1999	Bardazzi et al. <sup>3</sup>	4	Yes	Yes	Peri-infundibular lymphocytic	Yes	Negative	—	—
2000	Schön et al. <sup>8</sup>	1	Yes	Yes, patchy	Lymphocytic	No	Negative	—	—
2013	Almeida et al. <sup>9</sup>	1	Yes	Yes	Lymphocytic	Yes	Negative	3 wks	—
Our case		1	Yes	Yes, patchy	Lymphocytic peri-infundibular	Yes	Negative	2 y	Partial



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## Michelin Baby Associated With Facial Abnormalities<sup>☆</sup>



### Bebé Michelin asociado a anomalías faciales

To the Editor:

The term Michelin tire baby (MTB) was first used in 1969 by Ross<sup>1</sup> to refer to the case of a baby with multiple, widespread skin folds associated with hemihypertrophy and a broad bridge of the nose. We report the case of a girl with MTB and mild associated dysmorphic alterations.

A 3-month-old girl of Romanian origin, born at 34 weeks, with no history of consanguinity, was seen for excessive skin folds. She had no personal or family history of interest. Physical examination revealed abundant circumferential folds of skin of soft but solid consistency around the limbs and neck (Fig. 1). In addition, we observed microcephaly with frontotemporal flattening and hypotelorism, small, low-set ears, and teletelia. Skin biopsy showed an excessive accumulation of adipose tissue in the superficial dermis (Fig. 2). Cardiology study and transfontanellar ultrasound were normal. Ophthalmologic examination revealed convergent strabismus, microphthalmos, and blepharophimosis. The karyotype was normal, 46 XX.

A proposal has been made to replace the original term Michelin tire baby by the term circumferential skin folds syndrome or phenotype when the condition is associated with extracutaneous alterations.<sup>2</sup> These associated manifestations, detected in up to 74% of MTB patients, include facial abnormalities, neurologic disturbances (mental retardation, delayed psychomotor development, hypotonia, convulsions), ocular and cardiac alterations, genital hypoplasia or malformation, scoliosis, and pectus excavatum (Table 1).<sup>2–5</sup> Our patient presented teletelia, not previously reported in association with MTB. To date, no consistent pattern of inheritance has been identified.<sup>6</sup>



Figure 1 Abundant circumferential skin folds of soft but solid consistency around the limbs and neck.

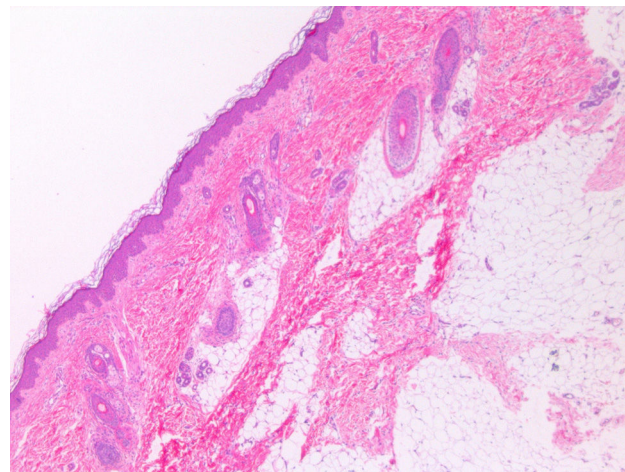


Figure 2 Skin biopsy showed an excessive accumulation of adipose tissue in the superficial dermis.

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