

Tuberous Parietal Lesion Secondary to Sirsasana, the Yoga Headstand Posture[☆]



Lesión tuberosa parietal secundaria a *Sirsasana*, una postura de yoga invertida

Sirsasana is one of the most common inversion postures in yoga and is proposed to increase blood flow to the brain, improving memory and other intellectual functions.^{1,2} When practicing this posture the body weight rests on the central-parietal region of the cranium. Beginners should maintain this posture for 1 min, subsequently increasing to 5 min. The posture should be performed under the supervision of an instructor to avoid injury.²

We describe a reactive skin injury caused by long-term practice of *Sirsasana*.

The patient was a 62 year-old man with no relevant past medical history other than the practice of an inverted yoga posture for 30 minutes several times a day since the age of 15. He presented with a persistent, asymptomatic, raised lesion in the interparietal region that had appeared more than 20 years previously. The size of the lesion had increased during the first few years and then subsequently stabilized. Occasional ulceration and infection of the lesion resolved spontaneously or after antibiotic treatment for approximately 10 days. Physical examination revealed a hard, oval-shaped, tuberous lesion in the interparietal region of about 10 cm in anteroposterior length and 6 cm in width with a centrally eroded surface (Fig. 1, A and B). Blood tests revealed no significant abnormalities. Radiograph of the skull showed an increase in soft tissue in the parietal region and associated periosteal reaction. Based on these findings, a contrast-enhanced computed tomography

scan of the brain was performed, revealing an extracranial soft-tissue mass in the upper frontal convexity along the midline, with discrete underlying periosteal reaction and no clear involvement of the outer table of the diploe, consistent with a reactive process. Skin biopsy showed marked orthokeratotic hyperkeratosis and mild epidermal acanthosis. The dermis showed focal fibrosis with proliferation of small vessels, dense perivascular lymphocytic infiltrates, and isolated siderophages (Fig. 2, A and B). Magnetic resonance imaging of the brain revealed thickening of extracranial soft tissue at the level of the coronal and sagittal sutures and the external table. The latter showed hypointensity in all sequences indicating sclerotic bone reaction. These findings were consistent with fibrotic changes affecting the extracranial soft tissues and sclerotic bone reaction in the underlying cortical bone (Fig. 3).

The patient continues to practice yoga at the same frequency and intensity, despite being warned of the probable link between that activity and the lesion.

The lesion remains stationary after 24 months of follow-up.

In the diagnosis of frequently ulcerous, tuberous lesions of the cranium, the first step is to rule out soft-tissue tumor. The majority of soft-tissue tumors present clinically as deep, slow growing masses, and the differential diagnosis is established based on histopathology.³ Imaging tests allow for better delineation of the lesion and help to determine its relationship with adjacent structures, and thus should be performed before conducting histological studies.⁴ Once a tumoral origin is ruled out, various reactive lesions should be considered, particularly nodular fasciitis⁵ and cranial fasciitis.⁶ Both are benign fibroblastic proliferations of unknown etiology, sometimes associated with previous trauma.⁷ These lesions present clinically as firm, well-defined masses that initially grow rapidly and then

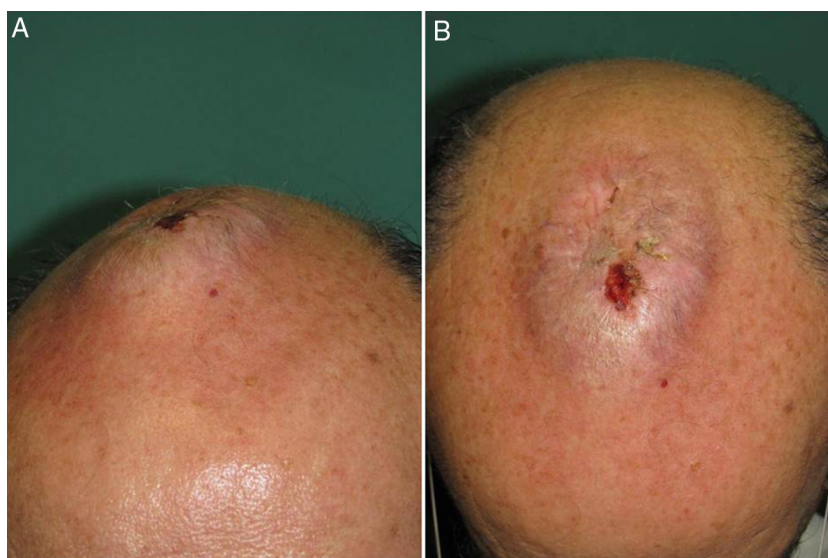


Figure 1 A, Tuberous lesion in the interparietal region. B, Oval-shaped tuberous lesion 10 cm in anteroposterior length and 6 cm wide with central surface erosion and crusting.

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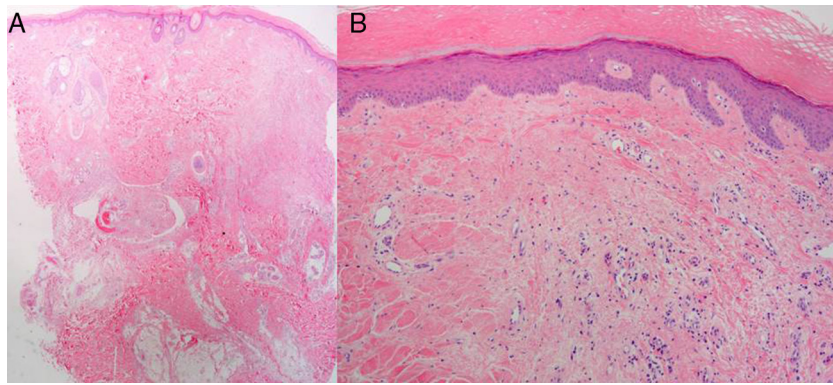


Figure 2 A, Biopsy of the parietal region of the scalp (hematoxylin-eosin, original magnification $\times 2$; panoramic image) B, Hematoxylin-eosin, original magnification $\times 4$. Histopathological findings: marked orthokeratotic hyperkeratosis and mild acanthosis; focal dermal fibrosis with proliferation of small vessels and perivascular lymphocytic infiltrates.

stabilize,⁷ as seen in our patient. Both forms of fasciitis share similar histological features, with loose, disorganized bundles formed by the proliferation of large spindle cells, myofibroblastic differentiation, no pleomorphism, and abundant non-atypical mitoses.^{5,7} In our case the biopsy ruled out fasciitis, leading to a diagnosis of reactive lesion secondary to long-term practice of Sirsasana. We believe that the development of this lesion was mainly due to the dedication of our patient to his exercises, which considerably exceeded the recommended daily duration.

Although yoga exercises are usually safe and promote health, some risks are associated with certain poses, such as inverted postures.¹ From the dermatological point of view we have not found an association between skin lesions and the practice of yoga. However, some problems have been described in connection with the practice of Sirsasana. For example, intraocular pressure can be increased in healthy individuals, an effect that is reversed after cessation of the inverted posture (this increase may be more pronounced in people with glaucoma or optic neuropathy secondary

to glaucoma,⁸ and may be associated with the progression of glaucoma⁹). Moreover, the central retinal vein can become occluded due to vascular thrombosis caused by an intermittent increase in conjunctival venous pressure and a decrease in venous drainage.² Finally, cervical compressive myelopathy and cervical listhesis can be caused by the biomechanical alterations induced by the inverted posture.¹⁰

Given the steady increase in the number of people practicing yoga daily, we believe that the dermatologist should be aware of the possible complications associated with this practice and should be alert to associated skin problems that may occur.

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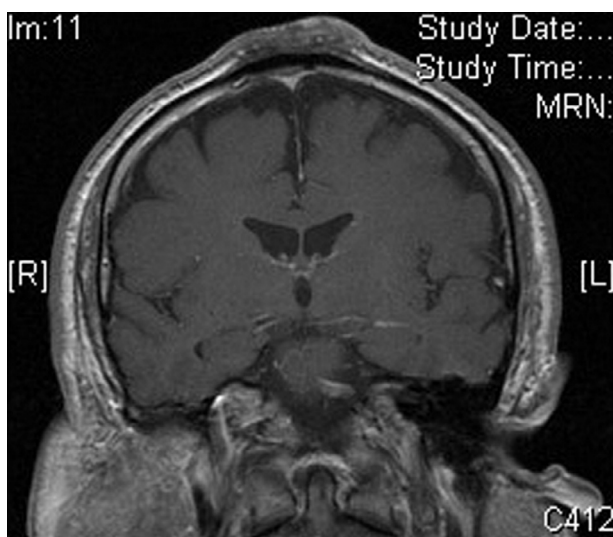


Figure 3 Magnetic resonance imaging scan. Note the thickening of extracranial soft tissues (skin, subcutaneous tissue, and galea) and the external table, which shows hypointensity.

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An Amelanotic Dysplastic Melanocytic Nevus Induced by Vemurafenib[☆]



Nevus melanocítico atípico amelanótico inducido por vemurafenib

Vemurafenib is a B-raf kinase inhibitor developed to treat metastatic melanoma in patients with the *BRAF* V600E mutation, which is found in 40% to 60% of patients with advanced melanoma. It acts by inhibiting the BRAF/MEK/ERK mitogen-activated protein kinase (MAPK) pathway at the BRAF/MEK step. Recent studies have reported that vemurafenib is associated with a 63% reduction in risk of death.^{1,2} Adverse effects include joint pain, photosensitivity, alopecia, fatigue, hyperkeratosis, xerosis, nonspecific rash, keratoacanthoma, and squamous cell carcinoma.³⁻⁵ We report the case of a patient with stage IV melanoma who developed a clinically amelanotic dysplastic melanocytic nevus 2 months after starting treatment with vemurafenib.

The patient was a 42-year old white man who had been diagnosed with a stage IV melanoma on the right arm 6 years earlier. Sentinel node biopsy results were positive and the patient underwent right axillary lymph node dissection followed by interferon therapy. Follow-up screening 6 years after surgery led to the detection of 2 pulmonary nodules and metastatic lesions in the C5 and T11 vertebrae, both iliac wings, and the sacrum. After confirmation of the presence of the *BRAF* V600E mutation, treatment was started with 960 mg of vemurafenib every 12 hours. Two months into treatment, we examined a 10 × 8-mm² papular lesion on the left thigh that had been absent at the preceding visit, 1 month earlier, and had grown to this size gradually. The lesion was dome-shaped, skin-colored, and asymptomatic (Fig. 1). With a suspected diagnosis of hypertrophic actinic keratosis, complete excisional biopsy of the lesion was performed. Histologic findings included a proliferation of melanocytes in the epidermis and papillary dermis, with mild architectural disorder (Fig. 2). Melanocytes were arranged in nests in the epidermis and papillary dermis, and nuclear pleomorphism was mild. Immunohistochemical staining with Melan-A and HMB-45 showed a proliferation of melanocytes in the basal layer of the epidermis and scattered single melanocytes in the upper layers (Fig. 3). All these findings were consistent with a dysplastic nevus.

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In cells with wild-type *BRAF*, vemurafenib increases phosphorylation of ERK (extracellular signal-regulated kinase) and thus paradoxically induces hyperactivation of the MAPK signaling pathway, promoting cell proliferation and survival. Hyperactivation is responsible for adverse effects such as keratoacanthoma, squamous cell carcinoma, and actinic keratosis. This paradoxical activation effect can also trigger progression of benign melanocytic lesions to melanoma.⁶ Atypical melanocytic proliferation and the development of new melanomas have been described in patients undergoing treatment with vemurafenib, and vemurafenib-induced changes in preexisting nevi have been observed by dermoscopy.⁷



Figure 1 Papular skin-colored lesion on the left thigh.

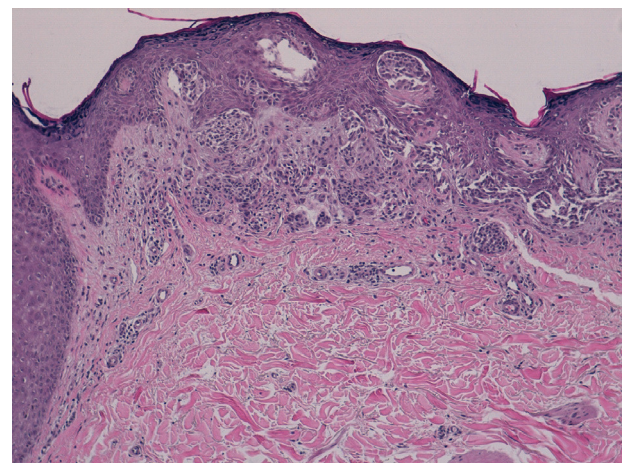


Figure 2 Proliferation of melanocytes in the epidermis and papillary dermis, with mild architectural disorder (hematoxylin-eosin, original magnification ×40).