

# Hard Nodular Plaque on the Scalp

Ryan A. Gall, MD; Alyson J. Brinker, MD; John D. Peters, MD



A 35-year-old man presented to the dermatology clinic with a slow-growing plaque on the scalp of 10 years' duration. The lesion was mildly pruritic and was never associated with any pain or discharge. He denied antecedent trauma or infection. A hard, erythematous, nodular, alopecic plaque with punctate hyperkeratosis on the left posterior temporal and parietal scalp was noted on physical examination. The lesion was slightly tender to palpation.

## WHAT'S YOUR DIAGNOSIS?

- a. acne keloidalis nuchae
- b. dissecting cellulitis of the scalp
- c. folliculitis decalvans
- d. kerion
- e. platelike osteoma cutis

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Dr. Gall is from the National Capital Consortium, Bethesda, Maryland. Drs. Brinker and Peters are from the Dermatology Department, Naval Medical Center San Diego, California.

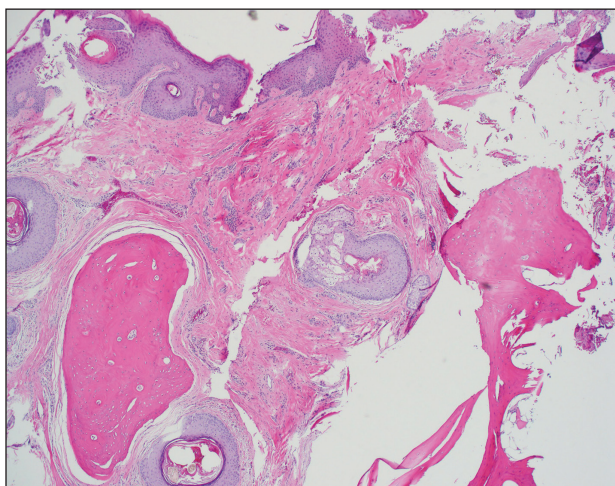
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Correspondence: Ryan A. Gall, MD, National Capital Consortium, 4301 Jones Bridge Rd, Bethesda, MD 20814 (ryan.gall.md@gmail.com).  
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## THE DIAGNOSIS: Platelike Osteoma Cutis

**H**istopathologic examination revealed extensive cutaneous ossification in the dermis and subcutis with dermal fibrosis and minimal surrounding inflammation (Figure 1). There was no evidence of infection or neoplasm. Further evaluation did not demonstrate any additional physical dysmorphia, and there were no imbalances of calcium-phosphate metabolism or abnormalities in parathyroid hormone or thyroid hormone function. A diagnosis of platelike osteoma cutis (PLOC) was favored. Computed tomography of the head showed material at the posterior skull of similar density to the adjacent calvarial skull and centered within the dermis, consistent with osteoma cutis (Figure 2).



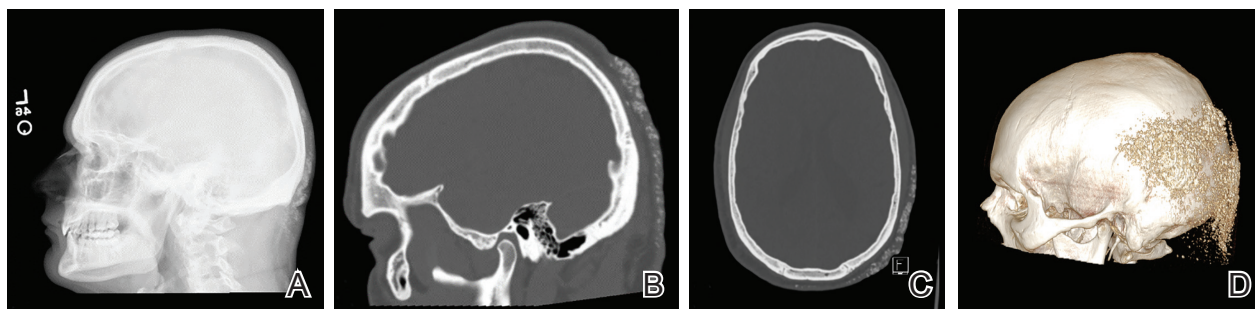
**FIGURE 1.** Platelike osteoma cutis. Biopsy showed extensive cutaneous ossification in the dermis and subcutis with dermal fibrosis and minimal surrounding inflammation (H&E, original magnification  $\times 40$ ).

Osteoma cutis describes the formation of bone within the skin. It occurs when hydroxyapatite crystals in a proteinaceous matrix are deposited within the skin, ultimately leading to the formation of bone ultrastructure. Ossification of the skin most often occurs secondary to trauma, inflammation, or neoplasm; however, it rarely may be a primary event.<sup>1,2</sup>

Platelike osteoma cutis is a rare form of primary cutaneous ossification in which bone forms within the skin in a platelike manner. It most frequently affects the scalp but also has been observed on the trunk and extremities.<sup>1</sup> A driving metabolic or endocrine abnormality typically is not identified.<sup>2</sup>

Platelike osteoma cutis can occur as an isolated finding or as a feature of Albright hereditary osteodystrophy (AHO) or progressive osseous heteroplasia (POH). In addition to cutaneous ossification, AHO involves short stature, endocrinopathy, obesity, shortened fourth and fifth metacarpals, and mental retardation. Progressive osseous heteroplasia is characterized by progressive ossification of the skin and deeper tissues such as muscle and fascia, leading to severe movement restriction; it is believed to be a localized nonprogressive variant of POH.<sup>3,4</sup> Mutations in the guanine nucleotide binding protein, alpha stimulating activity polypeptide 1 gene, *GNAS1*, a key regulatory gene involved in AHO and POH, have been found in several cases of PLOC.<sup>3</sup> Our patient lacked any dysmorphic features or laboratory abnormalities suggestive of AHO or POH. Moreover, testing of the tissue and blood for the *GNAS1* mutation was negative. Treatment of PLOC often is difficult. Our patient underwent a trial of ablative fractional laser resurfacing, which failed to lead to perceivable improvement.

The differential diagnoses include a kerion, dissecting cellulitis of the scalp, folliculitis decalvans, and acne



**FIGURE 2.** A, Lateral radiograph of the skull demonstrated amorphous density within the superficial tissues of the posterior scalp. B and C, Sagittal and axial computed tomography images showed this material to be of similar density to the adjacent calvarial skull and centered within the dermis. D, A 3-dimensional reconstruction showed the platelike nature of this cutaneous ossification. Radiographic images courtesy of Derek Grady, MD (San Diego, California).

keloidalis nuchae. A kerion is a manifestation of tinea capitis characterized by an inflammatory plaque, often with pain or tenderness. Kerions most frequently occur in children aged 5 to 10 years.<sup>5</sup> Failure to treat a kerion may result in scarring alopecia. Treatment consists of oral antifungals.

Dissecting cellulitis of the scalp is thought to occur secondary to follicular occlusion. It is characterized by boggy suppurative nodules primarily on the posterior and vertex scalp. Patchy hair loss is present and typically progresses to cicatricial alopecia. Histology characteristically shows areas of dense, predominantly neutrophilic, perifollicular dermal infiltrates.<sup>6</sup>

Folliculitis decalvans is a primary neutrophilic cicatricial alopecia that primarily occurs in adults. Patients with folliculitis decalvans tend to have multiple pustules on the periphery of confluent areas of scarring alopecia. It is theorized that an immune response to staphylococcal superantigens contributes to this disease process.<sup>7</sup>

The clinical findings of acne keloidalis nuchae include inflammatory pustules and papules with keloidlike plaques on the posterior neck and scalp. It occurs predominantly in teenaged and adult males of African

ancestry.<sup>8</sup> Treatment is aimed at reducing inflammation and preventing exacerbating factors. Severe disease courses may lead to scarring alopecia.

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