

QUIZ SECTION

Alopecic Lesions on the Scalp in a Young Woman: A Quiz

Elena del Alcazar Viladomiu, Arantxa López Pestaña, Sara Ibarbia Oruezabal, Maria Paula Gutiérrez Tamara and Anna Tuneu Valls

Servicio Dermatología, Hospital Donostia, Paseo Dr. Begiristain 107-116, Edificio Amara 2ª planta, ES-20014 Donostia-San Sebastián, (Gipuzkoa), Spain. E-mail: elenadelalcazarviladomiu@gmail.com

A 21-year-old woman with an unremarkable medical history was referred to our department with a 2-month history of alopecic lesions on her scalp. Physical examination revealed 2 skin-coloured, dome-shaped cystic tumours with a central keratin plug, associated with alopecia on the temporal and occipital scalp regions, respectively. A melanocytic naevus was seen near one of the patches (Fig. 1a, b). Dermoscopy

revealed numerous broken hair shafts and fine vellus. She did not report any itching or pain associated with the lesions and had no systemic symptoms. Bacterial culture of a slightly yellow fluid was negative and a biopsy was performed (Fig. 1c, d).

What is your diagnosis? *See next page for answer.*

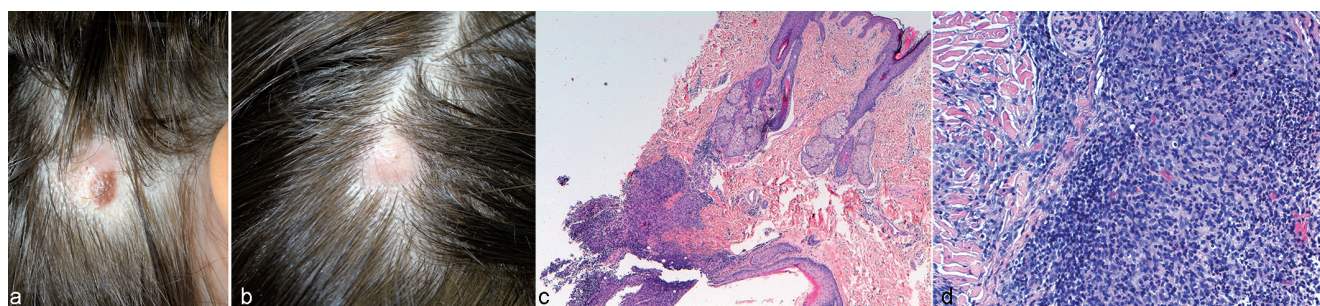


Fig. 1. (a) Alopecic lesion on the temporal scalp region with a raised central tumour and melanocytic naevus on top. (b) The second lesion on the occipital scalp region. Note a central keratin plug in both tumours. (c) Histopathology: a dilated hair follicle with a cystic cavity in the deep dermis, surrounded by a non-necrotizing granulomatous inflammatory reaction (haematoxylin and eosin (H&E), original magnification $\times 40$). (d) Deep dermal infiltrate, comprised mostly of numerous lymphocytes and histiocytes (H&E, original magnification $\times 200$).

ANSWERS TO QUIZ

Alopecic Lesions on the Scalp in a Young Woman: A Comment

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Diagnosis: Pseudocysts of the scalp or Alopecic and aseptic nodules of the scalp

“Pseudocysts of the scalp” were first described in the Japanese literature in 1992 by Iwata et al. (1). In 2009, Abdennader & Reygagne termed this entity “alopecic and aseptic nodules of the scalp” (AANS), a term that better characterizes the disease, because the nodules are always alopecic, the material from the puncture is sterile, and pseudocysts are not always present. While some authors consider “AANS” to be the most appropriate term (2), others favour “pseudocysts of the scalp” (3). We prefer the term “AANS” because it defines the disease more accurately.

The clinical picture of AANS is very characteristic, with the presence of one or a few dome-shaped nodules associated with alopecia limited to the lesional area and normal surrounding scalp (1, 2, 4). The nodules may be asymptomatic or cause mild pain, and the loss of hair occurs early on with the formation of the nodule. The AANS is occasionally associated with blood or yellowish discharge (5). The nodules are located mainly on the vertex, although they can appear on any area of the scalp (2, 6). AANS predominantly affects males and young adults between 16 and 48 years of age (6).

Histopathologically, these nodules show mixed inflammatory infiltrates with lymphocytes, histiocytes and giant cells. In most cases there are granulomas in the deep dermis or an ill-defined cyst wall (4). The material from the puncture is always sterile. However, the exudate may differ according to the predominant cellular type of the infiltrate and whether a vessel is eroded (4). Periodic acid–Schiff (PAS), Grocott–Gomori’s methenamine silver and Ziehl–Neelsen stains are always negative.

The aetiology of AANS is unknown (4, 5). A follicular occlusion may occur, followed by dilatation of the hair follicle. This dilatation may produce a deep folliculitis or a granulomatous reaction with an inflammatory infiltrate composed of lymphocytes, histiocytes and giant cells, with necrosis and erosion of vessels, causing the discharge and the pseudocyst. On the other hand, non-scarring alopecia probably occurs because the granulomatous infiltrate is located around the lower part of the hair follicle, beneath the bulge where the stem cells are located (2, 4). However, an immune process similar to that found in alopecia areata can-

not be excluded (2, 4). Some authors think that AANS could be part of the spectrum of follicular occlusion diseases, such as dissecting cellulitis of the scalp (DCS), acne conglobata or hidradenitis suppurativa (2, 4).

The main differential diagnosis is DCS. This entity is characterized by multiple, painful nodules, which are often interconnected, forming fistulas, and the alopecia is potentially cicatricial. Other differential diagnoses are an inflamed trichilemmal cyst, alopecia areata, bacterial or dermatophytic folliculitis and metastasis (4).

Signs of trichoscopy, such as broken hair shafts, black and yellow dots and fine vellus, are similar to those seen in alopecia areata and the early stages of DCS (7).

Different treatments, such as doxycycline, intralesional corticosteroids, repetitive punctures and surgical excision, have been reported (1–6). Our patient was treated with doxycycline 100 mg/day for 2 months with disappearance of her cutaneous lesions. The anti-inflammatory effect of doxycycline probably explains its efficacy in AANS.

In conclusion, AANS, as recently described in the literature, is probably an underdiagnosed entity. It is important to consider the possibility of AANS in order to avoid erroneous diagnosis and unnecessary treatments.

The authors declare no conflicts of interest.

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