Acta Dermatovenerol APA

Acta Dermatovenerologica
Alpina, Pannonica et Adriatica

2023;32:127-128
doi: 10.15570/actaapa.2023.24

Colocalizing paraneoplastic alopecia areata and nodular basal cell carcinoma

Martina Lambertini[1,2] ✉, Costantino Ricci[3,4], Bor Hrvatin Stancic[5,6], Martina Mussi[1,2], Barbara Corti[1,7], Cosimo Misciali[1,2], Bianca Maria Piraccini[1,2], Emi Dika[1,2]

[1]IRCCS Azienda Ospedaliero-Universitaria di Bologna, Bologna, Italy. [2]Dermatology Unit, Department of Medical and Surgical Sciences (DIMEC), Alma Mater Studiorum University of Bologna, Bologna, Italy. [3]Pathology Unit, Maggiore Hospital-AUSL Bologna, Bologna, Italy. [4]Department of Medical and Surgical Sciences (DIMEC), University of Bologna, Bologna, Italy. [5]Dermatovenerology Department, Ljubljana University Medical Center, Ljubljana, Slovenia. [6]Faculty of Medicine, University of Ljubljana, Ljubljana, Slovenia. [7]Pathology Unit, IRCCS Azienda Ospedaliero-Universitaria Policlinico di Sant'Orsola, University of Bologna, Bologna, Italy.

Keywords: basal cell carcinoma, alopecia, alopecia areata, scalp

Received: 27 March 2023 | **Returned for modification:** 8 August 2023 | **Accepted:** 17 August 2023

To the Editor,

Basal cell carcinoma (BCC) is the most common type of skin cancer, and its incidence continues to rise. Nodular BCC is the most common subtype and accounts for 57.6% to 78.7% of all BCCs (1); moreover, it is also the most common variant in the scalp region (2). BCCs in this region have been known to mimic alopecia areata patches (3). In addition, scalp BCCs can be potentially aggressive, which may be due to delayed diagnosis in concurrence with a rich vascular supply. These BCCs have a higher risk of recurrence and are more difficult to treat (4). Alopecia of the scalp, on the other hand, is a relatively common cause of patchy hair loss with a wide variety of associated conditions, one of which can be a secondary manifestation of neoplasm, either malignant or benign, and either local or metastatic (5).

A 33-year-old male with no history of previous skin tumors presented at our outpatient clinic due to a 12-month history of a progressing nodular lesion on the vertex of the scalp and a surrounding alopecic patch that developed 6 months after the onset of the nodule. Physical examination revealed a nodular erythematous shiny lesion surrounded by a patch of alopecia (Fig. 1a). No other patches of alopecia were detected. Dermoscopy showed polymorphous vessels, mostly arborizing and of various calibers, on an erythematous background and scarce granules of pigment at the periphery of the lesion. Diffuse yellow dots were noted in the alopecic area (Fig. 1b and 1c). The clinical and dermoscopic features of the nodular lesion and of the alopecic patch were highly suggestive of a nodular BCC surrounded by alopecia areata (AA). Therefore the nodular lesion was totally excised and a 5 mm punch biopsy was performed on the surrounding patch of alopecia, which proved the suspected diagnosis. The AA fully resolved without targeted treatment 3 months after complete excision of the BCC.

To the best of our knowledge, this is the third report of paraneoplastic AA colocalizing with a primary BCC (6, 7). In an analogue case reported by Choi et al., they believe that this occurrence may be related to an immune reaction primarily to the BCC, and that, due to similarity of cytokeratin components between BCC cells and the outer root sheath cells, this caused another immune reaction against the hair follicle. Cogan et al. describe a case of

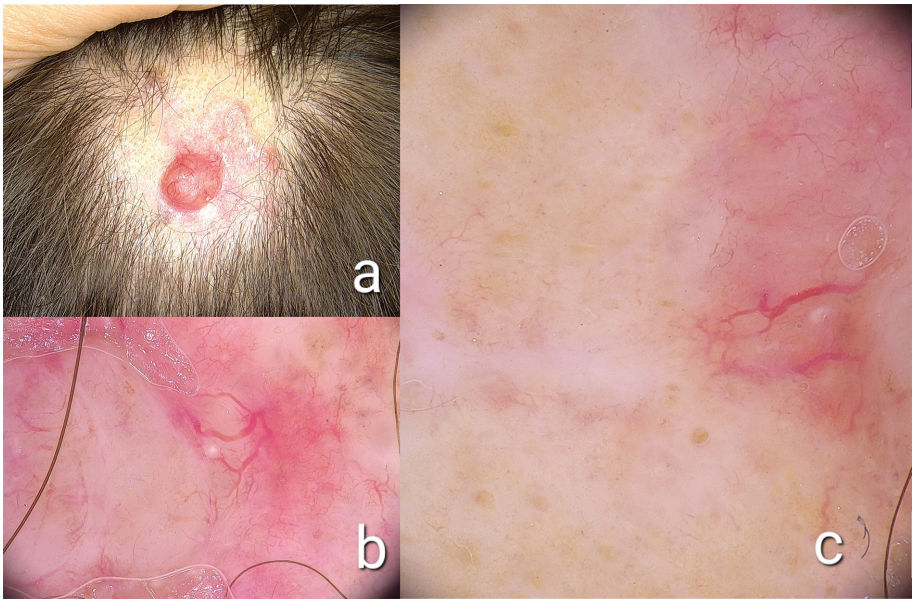

Figure 1 | a) Clinical presentation of the lesion showing an erythematous shiny nodule of the vertex with a surrounding alopecic patch; b) dermoscopic presentation of the nodule revealing arborizing vessels of various calibers on an erythematous background and scarce granules of pigment at the periphery; c) dermoscopic images of the alopecic patch showing diffuse yellow dots.

✉ **Corresponding author: mlambertini@hotmail.it**

paraneoplastic AA associated with a primary low-grade cutaneous carcinoma with squamous and trichoblastic features; the authors hypothesize that the inflammatory response to the neoplasm affected the immune privilege of the adjacent hair follicles, allowing the CD8+ T lymphocytes to attack the hair follicles, causing AA (8). Paraneoplastic AA has been linked to both distant cutaneous carcinomas and non-cutaneous malignancies, including gastric cancer, thymoma, metastatic malignant melanoma, breast cancer, and Hodgkin lymphoma (9–12). Perinevoid alopecia is considered a rare variant of AA adjacent to a pigmented melanocytic nevus (13), a benign lesion.

Paraneoplastic AA must be distinguished from alopecia neoplastica (AN), a rare form of scarring alopecia, in which neoplastic cells directly damage and destroy hair follicles. AN can be further subdivided into primary AN (caused by neoplasms originating from the scalp skin) and secondary AN (caused by cutaneous metastasis).

Furthermore, the occurrence of paraneoplastic AA in conjunction with a coexisting BCC or primary skin tumor remains infrequent. Nevertheless, we emphasize the critical significance of a comprehensive dermatoscopic evaluation. Identification and thorough excision of the primary tumor holds the potential to expedite complete remission of the disease.

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