Basal Cell Carcinoma, chrysalides-like structures, oncogenes, and sebaceous nevus: some considerations

Carcinoma basocelular, estruturas crisálides, oncogenes e nevo sebáceo: algumas considerações

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ABSTRACT

Dermoscopy is increasingly used to diagnose non-melanocytic skin tumors. Basal cell carcinoma presents some highly suggestive structures for its diagnosis. The presence of chrysalides-like structures is also frequently found in other benign and malignant skin tumors, and their significance in basal cell carcinoma has yet to be determined. HPV is a virus with oncogenic potential and its involvement in the pathogenesis of basal cell carcinomas is also not yet clearly understood.

Keywords: carcinoma, basal cell; dermoscopy; oncogenes

RESUMO

A dermatoscopia é ferramenta diagnóstica cada vez mais utilizada no diagnóstico dos tumores cutâneos não melanocíticos. O carcinoma basocelular apresenta algumas estruturas bastante sugestivas de seu diagnóstico. O achado de estruturas crisálides é também frequente em outros tumores benignos e malignos da pele. Seu significado no carcinoma basocelular ainda está por ser determinado. O HPV é vírus de potencial oncogênico. Sua participação na fisiopatogenia do carcinoma basocelular ainda não foi claramente entendida.

Palavras-chave: *carcinoma basocelular; dermoscopia; oncogenes*

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Basal cell carcinoma (BCC) is the most common malign neoplasia. Although it is non-aggressive in most cases, some patients suffer from mutilations on the nose, ears, lips, and face in general. In extreme cases, the enucleation of the eye may be necessary. Dermoscopy is a tool for early detection of these tumors. The most frequent dermoscopic findings in BCCs are globules, dots/, white or gray-brownish amorphous areas, and ulcerations. The most common vascular patterns are arboriform vessels, thin telangiectasias, and diffuse erythema.[1]

Sebaceous nevus (SN) is a congenital hamartoma that most often affects the scalp or face, and clinically manifests through yellowish plaques with verrucous surface. Some secondary neoplasias can arise on SN.[2] While human papillomavirus (HPV) involvement in the physiopathogeny of squamous carci-

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noma is clearly defined, little is known about its role in BCC.[3] This paper presents two cases that illustrate and discuss such considerations.

CASE 1

A 56-year-old white male patient presented with a papular lesion of approximately 1 cm (at its widest) located in the right scapular region, reporting pain and slow growth. A pearly papule with erythema and pigmentation areas was observed clinically. Polarized dermatoscopy evidenced the findings depicted in Figure 1. A punch biopsy was carried out, with histopathology as shown in Figure 2.

CASE 2

A 20-year-old white male patient presented congenital lesion on his scalp, with recent growth and bleeding. An ulcerated yellowish-erythematous plaque of 2 cm in diameter, with a lesion of vegetative and papillomatous appearance, was observed in the clinical examination. Dermatoscopy showed the findings seen in Figures 3 and 4.

Histopathology revealed epidermal hyperkeratosis, focal parakeratosis, acanthosis, and irregular papillomatosis. In the dermis – represented in higher proportions basically free of hair follicles, with some follicular sprouts and a few sebaceous glands – a dilated adnexal glandular structure with focal apocrine differentiation (hidrocystoma like) was observed. Basaloid keratinocyte hyperplasia, the clusters of which presented peripheral nuclear palisade and stromal retraction projecting into the underlying dermis, was observed in other epithelial segments (Figure 5).

DISCUSSION

The description of chrysalide structures was enabled by the advent of polarized light dermoscopy. These structures have been very useful in the diagnosis of melanomas, since they are correlated to the regression of dermal invasion. Chrysalides have an orthogonal orientation in melanomas, while the formation of rosettes is more frequently observed in actinic lesions. In BCC, those structures are disorganized and form lines or bright white areas, which were observed in the examination of the first patient.[4]

While the meaning of chrysalide structures in BCC is not precisely known, they have been found in up to 50% of cases and seem to constitute an additional dermatoscopic element of those lesions. Some authors associate the presence of chrysalides with the tumor's thickness, whereas others prefer to correlate them with the presence of stromal fibroplasia – especially in morpheaform and infiltrative subtypes.[4] It is possible that chrysalides have a role in the classic pearly shininess that can be seen with the naked eye.

The emergence of secondary neoplasias in SN is rare before puberty. Currently, it is known that BCC arises in approximately 0.8% of patients with that type of lesion. In most of the cases cited, it was actually trichoblastoma cases. [2]

In patient 2, yellowish lobular structures were compatible with a diagnosis of SN, while the multiple whitish projections containing blood vessels were very suggestive of a wart virus. In this case, the BCC diagnosis was carried out through histopathology – although in the dermoscopic re-evaluation, the diffuse erythema and thin telangiectasias were compatible with the

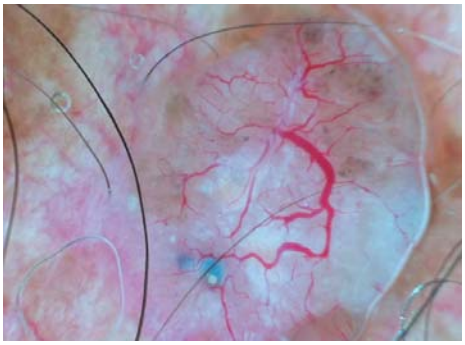

Figure 1: Dermoscopic examination through polarized light showing globular lesion with dots, bluish-gray globules, ovoid nests, exuberant arboriform vessels, and bright white structures – some arranged in the shape of bands, others with round shapes

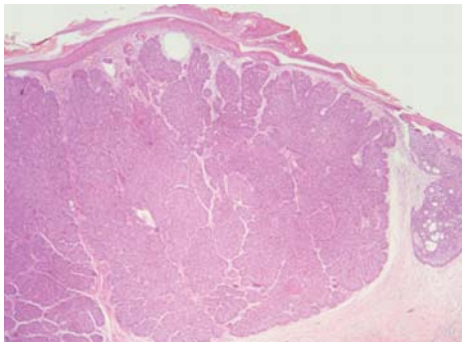

Figure 2: Thinned and rectified epidermis over dermal nodular lesion formed of basaloid cells (obj X4)

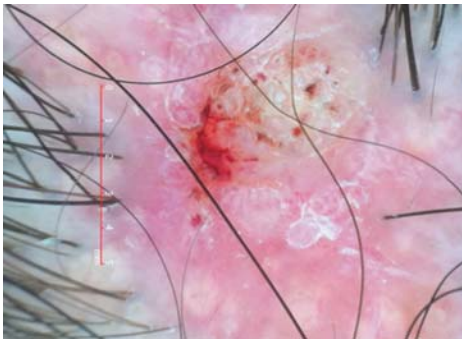

Figure 3: Diffuse erythema with yellowish lobular structures. In the central upper portion, structure has irregular papillomatous with bleeding points in the center. Bleeding areas compatible with ulcers can be observed

Figure 4: Occipital end of the lesion. Diffuse erythema blotting/blurring the yellowish lobular structures, with better visualization of thin telangiectasias (sometimes resembling an arboriform appearance), can be observed

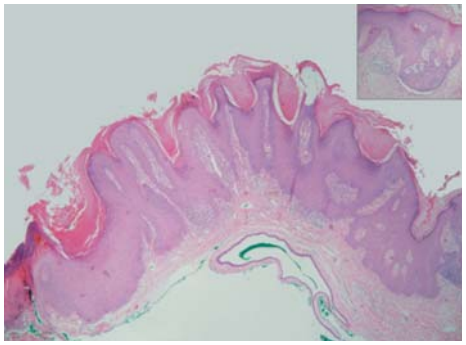

Figure 5: Epidermal hyperkeratosis, papillomatosis, and acanthosis. Note the cystic structure in the high dermis, the superficial cluster of basaloid cells with peripheral nuclear palisade, and stromal retraction pockets (detail) (obj X4)

SN diagnosis. The diagnosis was ultimately interpreted as a combination of SN with superficial BCC.[1,2]

HPV's oncogenicity in BCC has yet to be determined. The virus is known to interact with cellular proteins, altering their function or expression levels.[3] In 2011, Paolini and colleagues demonstrated that certain proteins are overexpressed in BCC, and that a high expression of p16INK4a and pAkt[2] is often associated with the presence of beta HPV species, e.g., HPV15. The activation of the p16INK4a and Akt/P13k route in the presence of those viruses suggests that HPV infection may participate in carcinogenesis in some subtypes of BCC.[1,7] The implications of that fact in the biological behavior of BCCs has yet to be determined. In our patient, finding the wart on a sebaceous nevus with the development of a superficial BCC is aligned with those authors' proposals.[3]

CONCLUSION

Chrysalide structures are frequently found in BCCs. Although their disorganized distribution is an important indication in the diagnosis of those tumors, their clinical significance remains uncertain. 1,2 The oncogenic role of HPV seems to be associated with the overexpression of cellular proteins and can be relevant in certain BCC types.[3] ●

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