MULTIPLE BASAL CELL CARCINOMAS APPEARING ON ACTINICALLY AGGRESSED SKIN

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Summary

Basal cell carcinoma (BCC) is the most frequent malignant tumour of the skin. It affects almost exclusively Caucasians and has a high incidence in areas with sunny climate. The most common risk factors are exposure to UV radiation and the presence of skin phototype II.

We present the case of a patient with multiple basal cell carcinomas located on the face.

***Keywords**: basal cell carcinoma, ultraviolet radiation, photoaging.*

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Introduction

Basal cell carcinoma (basal cell epithelium, basal cell carcinoma, trichoblastic carcinoma) is a malignant skin tumour originating in the basal cells of the epidermis or appendages.

BCC is considered the most frequent malignant tumour of the skin. It is frequently located on photoexposed areas, the cephalic extremity being the most affected, and has an increased incidence in the elderly.

The clinical aspect includes a variety of appearances and sizes, with specific elements.

BCC develops as a tumour formation with a globular appearance, peripherally bordered by erythematous nodular formations, translucent, with "pearl border" and telangiectasias on the surface. It evolves slowly and asymptomatically.

BCC can exhibit different clinical forms: nodular (it is translucent, sometimes with telangiectasias on the surface, firm to the touch, while the colour varies from pink to pigmented; in its evolution, it can ulcerate centrally, bleeding slightly to minor traumas), ulcerated/ulcus rodens (with the appearance of a nodule with pearly edges that ulcerates rapidly and expands in depth), terebrant (rapidly evolving, mutilating, with deep tissue damage), superficial/pagetoid (appearance of erythematous-scaly plaque, with atrophic center and slightly raised edges, consisting of small pearls, frequently pigmented), morpheaform/sclerodermiform (sclerotic plaque, depressed, imprecisely delimited, adherent to deep tissues, no tendency to ulceration), pigmentary (it is a form rich in black-brown pigment due to the melanocytes contained).

The clinical diagnosis of BCC can be supported by investigations such as dermatoscopy, confocal dermatoscopy, skin ultrasound, optical coherence tomography, but the definite diagnosis is histopathological.

We present the case of a patient diagnosed with multiple facial BCC.

Clinical case

A 71-year-old female patient from a rural area, a farmer by profession and a sheep breeder, with phototype II, comes for a medical consultation regarding multiple round-oval, exophytic formations, located on the face, with

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onset about 2 years earlier, without subjective signs.

There were no similar skin conditions in the family history, and the personal and physiological history was insignificant. The usual clinical and biological examinations were within normal limits.

The patient confirms excessive, repeated exposure to the sun, as well as a history of sunburn.

The dermatological examination shows a cutaneous phototype II, actinically aggressed facial skin, dry, thin, wrinkled, with numerous actinic keratoses and multiple facial tumour formations. On the left forehead, at the level of the nasal pyramid and in the right nasogenian area she has round-oval, nodular, pink, translucent formations, covered by thin epidermis, with telangiectasias on the surface, with approximate sizes of 1.5-3 cm. She has a pigmented formation between the eyebrows, made up of pearly nodules, with depressed centre, with the size of about 0.5 cm.

The presumptive diagnosis was multiple nodular BCC, the clinical and dermatoscopic appearance advocating for this diagnosis.

Electrocautery of tumour formations was practiced for therapeutic purposes with the collection of material for histopathological examination, to establish a definite diagnosis.

Histopathological examination revealed thin epidermis, appearance of solid, ulcerated BCC, with large areas of adenoid-cystic differentiation, with invasion in the deep dermis and desmoplastic stroma.

The definitive diagnosis was multiple nodular BCC.

Discussions

Basal cell carcinoma is the most common form of skin cancer, accounting for about 80% of non-melanotic malignancies.

Keratinocyte tumours are a major public health problem, despite their relatively low mortality rate; the risk for developing skin cancer in the US is now 1: 5 and much higher in subtropical Australia.

These tumours account for about 90% or more of malignant tumours of the skin, and of these, about 70% are basal cell carcinomas, whose incidence is estimated at 150/100,000 inhabitants per year in Europe and at higher values in the US (300/100,000) and Australia (2,000/100,000). Its incidence has doubled in the last 15 years.

Risk factors for the development of BCC include exposure to ultraviolet radiation, other skin conditions and immunosuppression. It usually occurs on sun-exposed areas of the skin, most often on the head and neck (80%), followed by the trunk (15%) and extremities.

The accumulated degenerative effects of solar radiation set the ground for the proliferative process, explaining at the same time the frequency of carcinomas on the exposed skin.

Ultraviolet (UV) radiation has a short wavelength (100-400 nm) and depending on the UV wavelength, it classifies as: UVC 100-280 (290) nm, UVB 280-315 (320) nm and UVA 315-400 nm.

UVs with wavelengths below 300 nm are absorbed at the level of epidermis, and those with wavelengths above 300 nm are absorbed by collagen, haemoglobin in the blood, bilirubin in the tissue and fat beta-carotene in the dermis.

After repeated and prolonged exposure to UV, photoaging of the skin may occur as a result of the deterioration of the DNA cleavage-repair function.

Photocarginogenesis occurs due to the accumulation of the degenerative effects caused by repeated exposure, especially to UVB, and less to UVA.

Caucasians are more frequently exposed than Blacks, people with cutaneous phototype II being more likely to develop basal cell carcinomas.

The profession has a predisposing role. People excessively exposed to solar radiation due to their occupation (farmers, fishermen, sailors, etc.) develop more easily basal cell carcinomas, often multiple.

BCCs have an increased incidence in the elderly. They have a chronic evolution, increase slowly in size, patients usually coming to the doctor after years from the onset. Genetic studies justify this slow evolution, by DNA replication with prolongation of the synthesis phase. Clinically, the tumour doubles in size in a few years, since active proliferative tumour cells are in a small percentage.

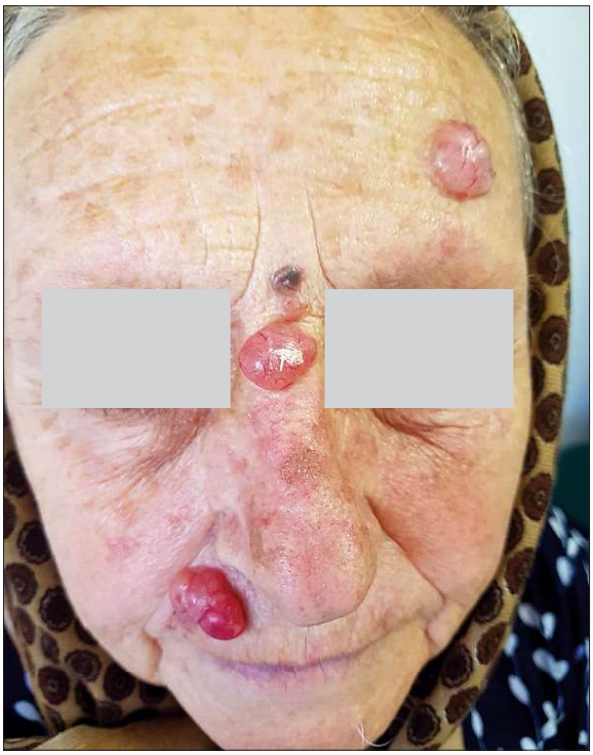

Figure 1. Facial nodular BCC, clinical appearance

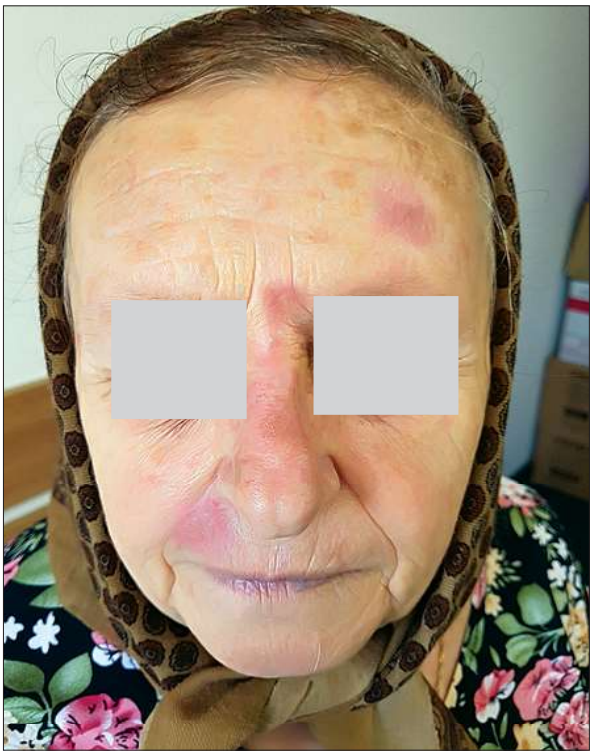

Figure 2. Image after treatment.

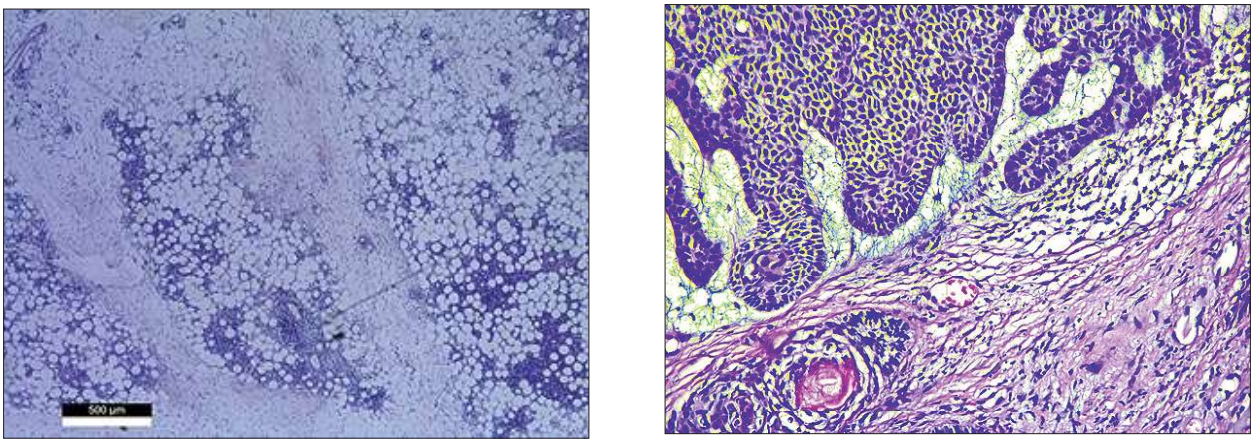

Figure 3. Solid BCC histopathological images. Collection for histological examinations.

The pathogenesis of BCC involves the release of the hedgehog signalling pathway (Shh), which leads to the activation of transcription factors associated with glioma (Gli), due to the inactivation of mutations in the hedgehog antagonist and receptor, PCTH1. Mutations in the p53 gene are also frequently present.

BCC originates in pluripotent cells in the basal layer of the epidermis that can differentiate into hair follicles, sebaceous glands or sweat

glands. There is considerable histopathological variation among BCCs, due to their origin, which allowed the description of several subtypes. The most common histopathological subtypes are nodular and superficial. In addition, there are some rare variants, which include basal squamous cell carcinoma, morpheaform carcinoma, keratosis, granular cell carcinoma, adamantoid carcinoma, and clear cell carcinoma.

The clinical appearance is variable, but classically the lesions are described in the form of pearly, shiny nodules with a demarcation limit that can be represented by crust, ulceration, or bleeding.

The definite diagnosis is histopathological, BCC has quite typical cytological and architectural features, but in certain situations raises problems of differential diagnosis with squamous cell carcinoma, trichoepithelioma (Brooke's multiple benign cystic epithelioma), desmoplastic trichoepithelioma, trichoblastoma, actinic keratosis, sebaceous gland tumours, sebaceoma, sebaceous carcinoma.

Establishing correlations between histopathological appearance, tumour location, clinical data on patients and their subsequent evolution will lead to the identification of new elements with a prognostic role for BCC.

In the case presented by us, a patient with actinically aggressed facial skin, with multiple actinic keratoses, developed multiple BCCs that appeared in the same period and had an accelerated growth, in about 1 year and 6 months reaching 2.5-3 cm. Our patient developed two types of basal cell carcinomas of the face: nodular and pigmentary.

Conclusions

It is well known that actinic skin aggression is responsible for the appearance of BCC. Multiple, concomitant, or successive carcinomas may occur in individuals with numerous actinic keratoses.

The peculiarity of the case consists in the appearance of multiple basal cell carcinomas that presented two different clinical aspects and developed in a relatively short period of time, being known that this type of tumour evolves slowly.

The patient will be followed at an interval of 6 months and then annually for at least 5 years, for the early detection and treatment of recurrences and/or other BCCs.

The presentation of this case brings into discussion the need for epidemiological screening programs, education, and prevention campaigns for the population, to reduce the number of patients who may develop BCC.

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Conflict of interest
NONE DECLARED

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