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The Evolution Of Biochemical Indices After Basal Cell Epithelioma Removal - Case Report.

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ABSTRACT

Abstract. The paper proposes new exposure data on etiopathogenesis basal cell epithelioma and present a clinical case investigated dermatoscopic, biochemically, treated surgically and guided to avoid relapses. The case presented is part of typical cases of pigmented basal cell carcinoma. Biochemical and haematological investigations performed one day before the excisional intervention (results 1) and 30 days (results 2) after the intervention: It is recommended to monitor biochemical investigations in which alterations were found, and ways for raising the immunological status.

Keywords: Cell proliferation, UV rays, pigmented basal cell epithelioma, surgical excision

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Introduction

Basal cell carcinoma is the most common cancer in the white population, accounting for about 75% of all skin cancers [1] skin cancer is uncommon in people of color when compared to Caucasians. When it does occur, it is often associated with increased morbidity and mortality. Differences in survival rates may be attributed to skin cancers being diagnosed at a more advanced stage, and socioeconomic factors such as lack of adequate insurance coverage and lack of transportation can function as barriers to timely diagnosis and early treatment. In addition to advanced stage at presentation, malignant skin lesions in skin of color often present in an atypical fashion. Because skin cancer prevention and screening practices historically have been lower among Hispanics, Blacks, and Asians, and given the changing demographics in the United States, interventions that are tailored to each of these groups will be needed. Public educational campaigns should be expanded to educate people of all skin types with emphasis on skin cancers occurring in areas not exposed to the sun. Mortality rate is low, but can evolve, causing extensive tissue distruction [2]. Maybe, but very seldom metastasis (< 0.1% of cases). The risk of developing a basal cell carcinoma is commonly associated with exposure to UV rays,

particularly intense exposure to the sun at young ages [3].

The transition from the G1 phase of the cell cycle is regulated by the interaction of several groups of proteins, D-cyclin (prad-1, bcl-1) being one of them. It has been demonstrated that UVA determines D1- cyclin overexpression, with consequences for cell cycle progression and cell proliferation. Cellular proliferation accelerated tumor formation determines and contributes to instability and genomic mutation, which can support tumor progression [4] which constitutes approximately 95% of the UV irradiation in natural sunlight, represents a major environmental challenge to the skin and is clearly associated with human skin cancer. Here, we show that a low, nonlethal dose of UVA induces dose-dependent cell cycle progression in human HaCaT keratinocytes. We found that UVA induced cyclin D1 accumulation, whereas siRNA knockdown of cyclin D1 blocked the UVA-induced cell cycle progression, indicating that this process is mediated by cyclin D1. UVA irradiation also induced AKT activation; when cells were incubated with phosphatidylinositol-3-OH kinase/AKT inhibitor or infected with dominant-negative AKT, cyclin D1 up-regulation, cell cycle progression, and proliferation were inhibited, suggesting that AKT activation is required for UVA-induced cell cycle progression. In contrast, extracellular signal-regulated kinase (ERK. Are as yet undiscovered intimate mechanisms through which UVA rays hasten the progression of the cell cycle and induce tumour formation and the role of D1- cyclin in these mechanisms. Surely the time that will be understood by transmission by which UVA signals D1- cyclin, chemoprevention will be target on critical areas [4]which constitutes approximately 95% of the UV irradiation in natural sunlight, represents a major environmental challenge to the skin and is clearly associated with human skin cancer. Here, we show that a low, nonlethal dose of UVA induces dose-dependent cell cycle progression in human HaCaT keratinocytes. We found that UVA induced cyclin D1 accumulation, whereas siRNA knockdown of cyclin D1 blocked the UVA-induced cell cycle progression, indicating that this process is mediated by cyclin D1. UVA irradiation also induced AKT activation; when cells were incubated with phosphatidylinositol-3-OH kinase/AKT inhibitor or infected with dominant-negative AKT, cyclin D1 up-regulation, cell cycle progression, and proliferation were inhibited, suggesting that AKT activation is required for UVA-induced cell cycle progression. In contrast, extracellular signal-regulated kinase (ERK.

Another molecules that mediates apoptosis is transmembrane protein Fas [5]. Fas ligand (FasL), a protein that belongs to the “tumor necrosis factor” (TNF induces apoptosis by binding to the Fas receptor (Fas). Fas/FasL R interactions plays an essential role in regulating the immune system and cancer progression. It is essential for the development of skin cancer disorders this interaction. In normal skin, Fas is expressed in cytoplasmic domain of membrane cells in the basal layer [6]. After exposure to UV rays, Fas expression is described throughout the epidermis and subexpressed appears again after prolonged exposure to UV rays. This is why basal cell carcinoma lesions stained negative for Fas [6]. However, the basal cell carcinoma expressed diffuse FasL, and it may be evidence of slippage under supervision by inducing apoptosis in immune level of peripheral lymphocytes [3].

A significant number of basal cell carcinomas appears on non-photoexposed areas, suggesting that there are other risk factors responsible for the occurrence of injuries aside from UV rays [7]. These include immunosuppression given by immunosuppressive medication (long-term corticotherapy), or to patients with AIDS and low number of CD4. It has been demonstrated that exposure to arsenic, a toxic metal that is widely spread in the environment may contribute to carcinogenesis [8].

Even though the most exposed areas are those in the head region, yet the carcinomas also appear on other parts of the human body [9]. Everyone is more or less exposed, but with a higher risk are farmers, workers in the refinery, those who drank contaminated water or living near industrial areas [10]. It has been described a cocarcinogenic skin mechanism involving the inorganic arsenic and exposure to UV rays. It has been demonstrated that this combination increases the malignant phenotype, by increasing the secretion of MMP-9, invasiveness, D1-cyclin expression and loss of p16 expression [11]both human skin carcinogens,

may act together as skin co-carcinogens. We find human skin keratinocytes (HaCaT cells. Other factors involved in the susceptibility of developing a basal cell carcinoma, are proteins that mediate the detoxification process, including UV radiation individual answers, by protecting vis oxidative stress, as well as the family of enzymes glutathione S-transferase (GST) [12]. They are involved in cellular defense mechanism in relation to harmful chemical products, produced either endogenous or with an exogenous source and which they eliminate [13]. UV radiation produces reactive oxygen species that peroxidase lipids and DNA damage through hiperoxid training, especially at the level of the skin.

Several polymorphisms have been described of the GST (e.g. GSTT1, GSTM1, GSTM3) associated with altered ability to Detox, influencing the risk of occurrence of basal cell carcinoma by altering the defense against oxidative stress induced by UV and thus promoting increased sensitivity to UV rays [14]. The gene CYP2D6, which codifies cytochrome P450, is involved in the detoxification of photosensitising agents. By O-demethylation it metabolizes and eliminates approximately about 25% of medicinal substances used.

CYP2D6, polymorphism correlates with an increase in the number of basal cell carcinoma by increasing photosensitivity that this Statute can determine and support [15]in the total BCC case group and subgroups, how many genes influence BCC numbers and their relative importance. In this study, we assessed the influence of two further candidates, glutathione S-transferase GSTP1 and cyclin D1 (CCND1 .

Material and method

In the experiment, we selected at random from the lot of patients studied, a patient diagnosed with basal cell carcinoma, whom I selected clinical and paraclinical findings in order to be etiopathogenic analysis.

Case study

Patient D.N., aged 68 years, an agricultural engineer with cutaneous phototype II, are presented in December 2016, from a medical check-up, for a tumor located at the level of the average 1/3 cheek as, supramandibular.

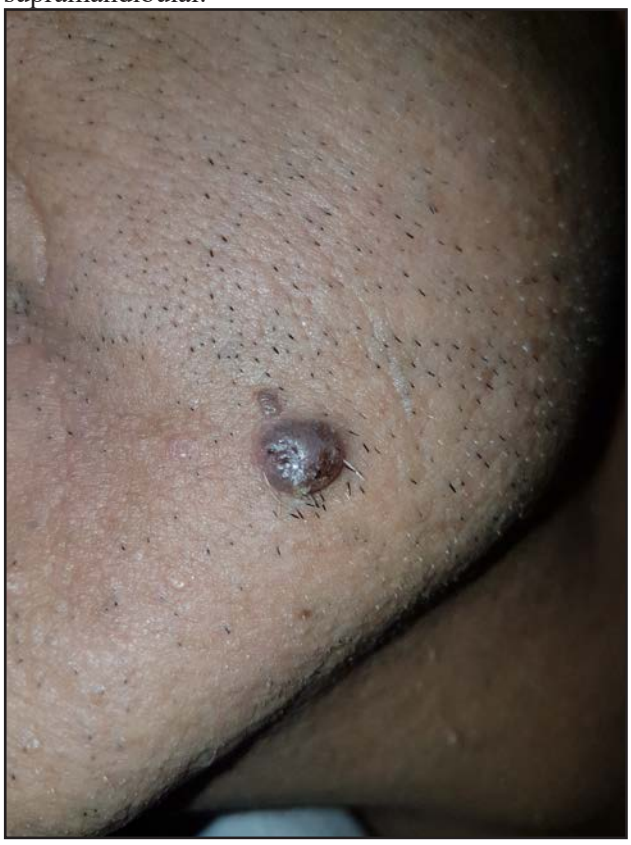

Figure 1. Supramandibular tumor formation

It is found that the formation had the dimensions of 1/0, 8 cm, with an elevation of 7 mm. Colouring the tumor was dark brown, being pierced by two small ulcers (Figure 1).

Medical history: the patient states that skin lesions appeared in full health, About 3 years ago, but increased in volume and ulcerated in the last 2 months.

Enquiries: The dermatoscopic analysis of the lesion was performed and after excision, it was sent to the laboratory of electron microscopy, for preparation

of bioptical parts and carrying out of specific shots. Were collected biological samples for hematological and biochemical investigations.

Results

On the dermatoscopic examination, there are red - pink areas and fine capillaries. In terms of pigmentation, these signs are evident, notably bluish-gray globules of various sizes (Figure 2).

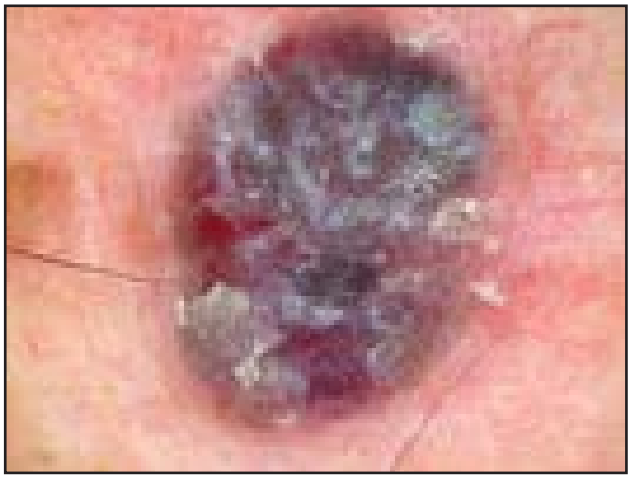

Figure 2. Pigmented basal cell epithelioma

Biochemical and hematological investigations performed the day before the excision intervention (1 results) and 30 days (2 results), after intervention:

Table I. Biochemical and hematological investigations performed before the excision, and after intervention

Name analysis	Results (1)	Results (2)	Reference range
HDL Cholesterol	↑ 67 mg/dL	58 mg/dL	> = 40-60 mg/dL
LDL Cholesterol	147 mg/dL ↑	126 mg/dL	< 129 mg/dL
Total cholesterol	217 mg/dL ↑	198 mg/dL	< 200 mg/dL
Triglycerides	94 mg/dL	89 mg/dL	< 150 mg/dL
Total lipids	672 mg/dL	588 mg/dL	400-700 mg/dL
Alaninaminotransferaza (ALAT)	↑ 47 U/L	38 U/L	< 41 U/L
Aspartataminotransferaza (AST)	31 U/L	30 U/L	< 40 U/L
Serum creatinine	0.92 mg/dL	0, 88mg/dL	< 1.2 mg/dL
Albumin	46.8% ↓	62%	52-68%
Alpha 1 globulin	2.5%	2.8%	2-5%.
Alpha 2 globulin	15.8% ↑	12.6%	6.6-13.5%
Beta globulins	13.6%	12.8%	8.5-14.5%
Gamma globulins	21.3% ↑	20.2%	11-21%
Albumin/immunoglobulins	0.88 ↓	1.28	1.2-2.23
Total protein	7.3 g/dL	7.8 g/dL	6.6-8.7 g/dL
Serum glucose	↑ 113 mg/dL	95 mg/dL	60-99 mg/dL
Serum urea	30 mg/dL	33 mg/dL	< 49 mg/dL
VSH	18 mm/h	16 mm/h	< 20 mm/h

LDL - Low density lipoproteins
HDL - High density lipoproteins
VSH – Erythrocyte sedimentation rate

Discussion

The case presented is part of typical cases of pigmented basal cell carcinoma, uncomplicated, appeared at an elderly person with a fototip (light complexion, with ultraviolet radiation action vulnerability) and with a history of prolonged exposure to sunlight (professional). Surgical excision treatment has radically resolved tumor progression. To avoid the appearance of other skin tumors, it is recommended that the patient avoid exposure to the sun, possibly using antisolar lotions with high photoprotection (50%).

From the analysis of the laboratory investigations (Table I) shows that the patient presents alterations in cholesterol metabolism (increased total cholesterol and LDL), changes in liver functions (ALAT increase), changes in serum protein became (an increase in alpha 2 globulin and albumin and the decline of the ratio gammaglobulins/ immunoglobulins). At the same time, it is observed and an increase of blood glucose, which confirms the existence of dismetabolic changes.

The same analysis conducted at 30 days after from the excision intervention, shows an improvement in the results of the investigation, all located in physiological parameters. This finding was made in circumstances which did not intervene in the said interval, changes in diet, or initiation of a medication. It is possible that the main question to be posed by metabolic regulating action of ACTH. This hormone controls the development and hormonal secretion of the adrenal cortex. By stimulating the fasciculated area of the adrenal cortex, activates the synthesis and secretion of glucocorticoids (cortisol and corticosterone). It is known that the stimulatory effects of ACTH on the adrenal secretion are mediated by cyclic AMP [16].

Secondary properties of ACTH is due to metabolic effects of glucocorticoid hormones, triggering glucid, protein and lipid metabolism. Regulating the secretion of ACTH is achieved about neuro-humorous, with the indirect participation of the hypothalamus. The main factor regulating humoral is the blood concentration of glucocorticoid hormones, notably cortisol. Under their influence, the secretion of ACTH falls through a negative feedback mechanism, namely the increase in the case of decrease in glucocorticoids [17].

However, a hepatic, hypolipidemic, hypoglucid diet, rich in vitamins and natural minerals is recommended for the patient, monthly monitoring of changes in biochemical investigations.

Conclusions

Surgical excision of skin tumor is a radical method for optimum treatment, radical, but requiring prior dermatoscopic investigations, then histopathological investigations, concomitant with the monitoring of standard biological parameters. Maintaining optimal immune status by playing confidence in the success of the medical-surgical intervention or medical conditions associated with compliance with the hours of rest, a balanced diet and avoiding exposure to solar radiation, may contribute to the prophylaxis and successful treatment of cutaneous basal cell epitheliomas, as well as improving the patient's biological status.

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