Skin of sailor: cutis rhomboidalis nuchae, actinic keratosis, squamous cell carcinoma and basal cell carcinoma. Case report

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ABSTRACT

Chronic exposure to environmental ultraviolet radiation (UVR) plays a key role both in photocarcinogenesis and induction of accelerated skin ageing. Sailors commonly experience a significant number of cutaneous problems, related to the exposure to environmental factors due to their working conditions. Among these factors, sun exposure is able to determine both acute and chronic skin damage, mostly linked to the effects of the ultraviolet (UV) radiation on epidermal and dermal structures.

We report a case of sailor with cutis rhomboidalis nuchae, solar elastosis, actinic keratosis (AK), numerous squamous cell carcinoma (SCC) and basal cell carcinoma (BCC) of the head and trunk.

KEY WORDS: ultraviolet, sailor, actinic keratosis, squamous cell carcinoma, basal cell carcinoma

INTRODUCTION

Ultraviolet (UV) radiation spectrum is the major component of solar radiation, with multitude of effects on the skin [1].

The most important biologically active functional components of UV radiation spectrum are UV-A (~ 320–400 nm) and UV-B (~ 290–320 nm) components. UV-B is responsible for more severe damage to skin, with acute erythematogenic effect and long term carcinogenic potential, inducing photoaging and mutagenic damage to nucleic acids. UV-A, less absorbed by biological targets in the skin, penetrates deeper than UV-B and is less erythematogenic. It promotes reactive oxygen species (ROS) accumulation and induces direct cell damage, carcinogenesis and contributes to photoaging and many photodermatoses [2].

Over the past few decades the incidence of skin cancer has been rising at an alarming rate. The increase is most likely a result of several factors: depletion of the protective ozone shield due to climate change and people's careless behavior in the sun. Extended sun exposure increase the probability of skin cancer as well as other chronic damage to the skin by sun [1, 2].

We report a case of sailor with cutis rhomboidalis nuchae, solar elastosis, actinic keratosis, numerous squamous cell carcinoma and basal cell carcinoma of the head and trunk.

CASE REPORT

A 67-years-old man, sailor came for dermatology office with numerous lesions in the skin of the head and trunk.

A patient for about 40 years sailed by ship, trader, boat, canal boat and fishing boat.

Physical examination revealed the solar elastosis with multiple skin lesions after damage of sun. Numerous lesions on the forehead and face type actinic keratosis (Figure 1). Besides: rhomboidalis nuchae cutis (Figure 2), numerous freckles, lentigines (Figure 1–3). Within four years he had surgically cut: BCC on the forehead, two SCC on the trunk (area the shoulder joint on the right side) (Figure 3) and four atypical naevi.

Discussion

Sailor commonly experience a significant number of cutaneous problems, related to the exposure to environmental factors due to their working conditions. Among these factors, sun exposure is able to determine both acute and chronic skin damage, mostly linked to the effects of the ultraviolet (UV) radiation on epidermal and dermal structures. In particular, UV-A appears to play a major role in the deterioration of der-

FIGURE 1.
Numerous lesions of the type actinic keratosis on the forehead and head. The scars after BCC on the right temporal area.

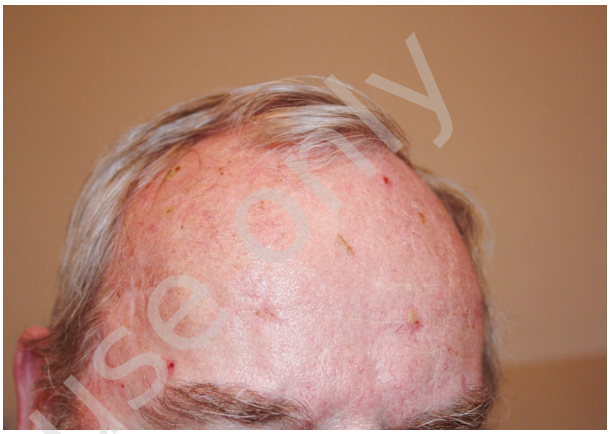


FIGURE 2.
Characteristic thickened, leathery skin of neck with accentuated markings and furrows of cutis rhomboidalis nuchae.

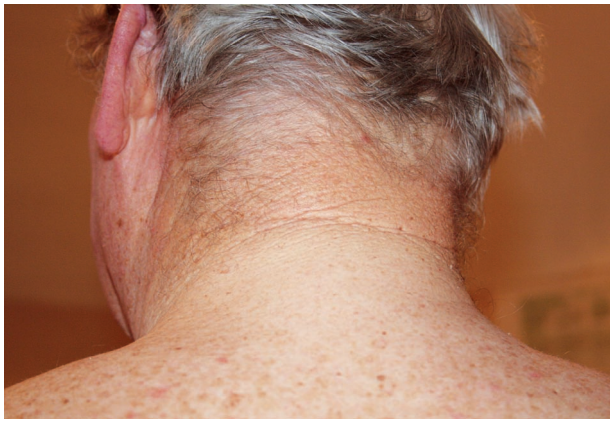
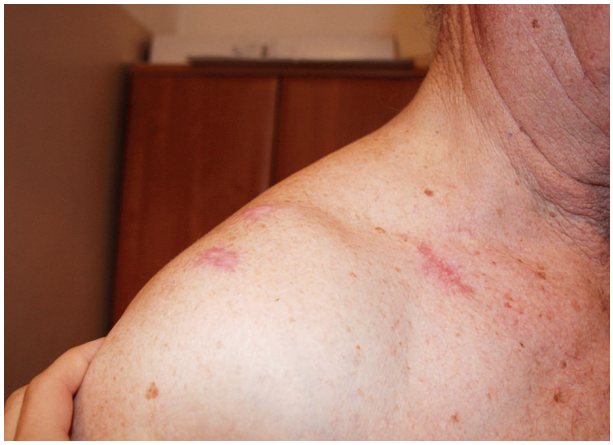


FIGURE 3.
Numerous freckles and lentigines. The scars after two SCC on the trunk (area the shoulder joint on the right side) and one scar after atypical naevus.



mal structure leading to the photoaged appearance of the skin, while UV-B is mainly responsible for skin cancers. Peculiar clinical features of skin damage in sailor include dryness, irregular pigmentation, wrinkling, stellate pseudoscars, elastosis, inelasticity, telangiectasia, comedones and sebaceous hyperplasia. Furthermore, the high incidence of actinic keratosis and non-melanoma skin cancers (BCC, SCC), on sun-exposed areas, confirms the need for occupational health policies focusing on issues such as photoprotection [1–3].

Solar elastosis is a well-recognized manifestation of chronic sun exposure, mainly affecting white patients. Well-established clinical manifestations of SE include Favre–Racouchot syndrome, cutis rhomboidalis nuchae, actinic comedonal plaques, elastomas, elastotic nodules of the ears, elastotic bands, collagenous plaques of the hands and colloid millia [3].

Cutis rhomboidalis nuchae of Jadassohn (CRN) is a manifestation of exuberant dermal actinic elastosis that occurs in the neck. It commonly affects fair-skinned elderly individuals who were chronically exposed to UVR. The exacerbated production of thick and disarrayed elastic fibers is due to a chronic inflammatory process, with mast cell degranulation and activation of fibroblasts that produce collagen-degrading metalloproteinases.

Long-term, chronic sun exposure causes thickening of the most superficial layer of the skin (the epidermis) and abnormalities in the composition of the middle layer of the skin (the dermis) [4].

CRN is usual association, actinic keratosis, basal cell carcinoma, squamous cell carcinoma or keratoacanthoma may appear in sun-damaged skin.

In study of Brazilian authors CRN, elastosis, poikiloderma and solar melanosis were significantly associated with actinic keratoses and basal cell carcinoma in their analysis [4, 5].

In our patient there were other factors of the risk cited by the authors: age, blue eyes, blond hair and time of exposure in the sun (40 years of work at sea).

In the German study (KORA-survey 2000) (a total of 2,823 adults) significant increasing trends with age were found for all UV-light-associated entities (Elastosis, cutis rhomboidalis nuchae, morbus Favre–Racouchot, lentigines solaris, lentigines seniles, actinic keratosis) [5]. Skin cancers are the most common malignancies globally and keratinocytic tumors make up 90% of all skin cancers. Basal cell carcinoma makes up more than 70% of kerotinocytic tumors and is seen 4 to 5 times more frequently than squamous cell carcinoma. These tumors can develop de novo or following the progression of precursor lesions such as actinic keratosis.

The most common factor in the etiology of keratinocytic tumors is exposure to ultraviolet (UV) light. The relationship between UV light and skin cancer development has first been defined in 1875 and has been proven in the 1950s with the demonstration of DNA mutations that developed due to UV exposure [6].

The p53 gene is the most commonly mutated gene in human cancers and a p53 mutation is reported in more than 50% of human cancers. A p53 mutation is frequently reported to develop as a CC-TT base change in keratinocytic skin cancers and the mutation is believed to develop in the early stages of carcinogenesis.

Actinic keratoses (AK) are dysplastic keratinocytic lesions confined to the epidermis, which are caused by ultraviolet radiation, and are one of the most frequent diagnoses among dermatologists worldwide.

Lesions are treated mainly for preventing reasons (malignancy), however AK are also treated for cosmetic and symptomatic purposes. Predisposing risk factors are chronic sun exposure, outdoor occupation, fair skin (Fitzpatrick skin types I and II), light eye color, frequent sunburns and at least one actinic keratosis [7].

The morphology of actinic keratoses can vary widely. The most common presentation is that of a pink scaly patch or plaque on an erythematous base.

Actinic keratoses typically are a few millimeters in size, they can reach confluent patches of a few centimeters in diameter.

Although these lesions can be found anywhere on the body, they are typically located on sun-exposed areas such as the face, neck, and extremities. It is thought the irradiation from the sun produces genetic mutations in keratinocytes as well as loss of tumor suppressor genes such as p53 [5]. These changes coupled with suppression of the cutaneous immune response through inhibiting the ability of Langerhans cells to present antigen to helper TH1 lymphocytes result in the development of actinic keratoses [8].

Physician office visits for the diagnosis of AK and nonmelanoma skin cancer is increasing, such tendency is probably due to the heightened public awareness of the prevalence of precancerous and cancerous skin conditions.

The aim of German study was to assess the prevalence of UV-induced actinic keratosis and further skin lesions [9]. The questionnaire revealed a pre-employment UV radiation exposure in 104 seafarers, sunbed use in 26 subjects and a median work-related UV radiation exposure at sea of 20 years. The diagnosis of actinic keratoses was made in 94 seafarers and the clinical diagnosis of skin cancers in 48 seafarers (28 basal cell

carcinoma, 11 squamous cell carcinoma, 9 malignant melanoma). Actinic keratoses and squamous cell carcinoma were related to the duration of seafaring time in years.

Actinic keratosis is an indicator of cumulative UV exposure and may progress into squamous cell carcinoma (SCC). The p53 gene plays a central role in the development of SCC, and mutations in this gene are found in 90% of SCC and up to 100% of AK cases [10].

SCC is the second most frequent type of skin cancer, and its incidence has increased over the last several years in all regions of the world.

Clinically, induration, pain, large size, marked hyperkeratosis, ulceration, bleeding, rapid growth, and recurrence or persistence may be markers of AK progression into SCC. The risk of SCC metastasizing ranges between 0.5% and 3%.

Röwert-Huber J. recommend an AK classification system that describes these lesions as squamous cell carcinomas (SCCs), using the terminology 'early in situ SCC Type AK I', 'early in situ SCC type AK II' and 'in situ SCC Type AK III', thereby giving clinicians better guidance for diagnosis and specific treatment recommendations [11].

Basal cell carcinoma (BCC) of the skin is now the most common malignancy in the human population.

This neoplasia has a low degree of malignancy and mortality due to the typical slow growth and reduced potential to metastasize, in addition to its early diagnosis, as it is preferentially located in areas exposed to sunlight.

Ultraviolet radiation (UVR) is the main environmental risk factor associated with the genesis of the BCC, as shown by a higher frequency of lesions in sun-exposed areas. In addition to the immunosuppressive action on the skin, UV-B radiation generates mutagenic photoproducts in the DNA, which promotes mutations in genes such as *PTCH* and p53. In turn, UV-A radiation has mainly indirect effects by generating cytotoxic and mutagenic free radicals [12].

The most important constitutional risk factors are: fair skin (difficulty to tan and predisposition to sunburns), light-colored eyes and hair, family history of BCC, and freckles in childhood. Noteworthy behavioral factors are: professional activity unprotected from UVR, rural activities, and sunburns in youth [12, 13].

The tumor may occur at any age, but a higher frequency is noted in males and in older age. Nearly 80–85% of BCCs involve the head and neck regions and 25–30% are localized in the nasal area.

Basal cell carcinoma (BCC) is the most common type of facial skin cancer. It represents alone approximately 65% of all epitheliomas, and the incidence is 4-fold higher than that of squamous cell carcinoma [12].

One of the most negative features of this disease is frequent tumor recurrence. Unfortunately, all of the traditional diagnostic criteria have failed to definitively predict which patients should be considered at high risk of recurrence [13].

Careful monitoring must be undertaken for at least 3 years; however, the most appropriate course is a lifetime of regular follow-up.

CONCLUSION

Chronic exposure to environmental ultraviolet radiation (UVR) plays a key role in both photocarcinogenesis and induction of accelerated skin ageing.

For the current state of skin a patient has 'earned' by a lifetime.

As age progresses, people with light skin and hair, with presence of skin lesions resulting from chronic solar exposure, such as solar elastosis, cutis rhomboidalis nuchae, actinic keratoses and a great number of melanoses, have a greater risk of developing premalignant and malignant cutaneous lesions.

Policies of photoprotection, photoeducation and early diagnosis in professionals exposed to solar radiation must be promoted by medical societies and trade unions as preventive strategies of occupational damage.

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