Commentary

iMedPub Journals
www.imedpub.com

Journal of Neoplasm
ISSN 2576-3903

2022
Vol.7 No.6:16

Transformation of Solar Keratoses into Squamous Cell Carcinoma

Richard David*

Department of Dermatology, University of Maryland School of Medicine, Baltimore, United States

***Corresponding author:** Richard David, Department of Dermatology, University of Maryland School of Medicine, Baltimore, United States, E-mail: David_R@umaryland.edu

Received date: October 17, 2022, Manuscript No. IPJN-22-15233; **Editor assigned date:** October 19, 2022, PreQC No. IPJN-22-15233 (PQ); **Reviewed date:** October 31, 2022, QC No. IPJN-22-15233; **Revised date:** November 10, 2022, Manuscript No. IPJN-22-15233 (R); **Published date:** November 17, 2022, DOI: 10.36648/2576-3903.7.6.16.

Citation: David R (2022) Transformation of Solar Keratoses into Squamous Cell Carcinoma. J Neoplasm Vol.7 No.6: 16.

Description

Melanoma and pigmented actinic keratosis may share clinical features, making diagnosis challenging for dermatologists. Dermoscopy has been used to improve the clinical diagnostic accuracy of pigmented skin lesions, despite the fact that histopathology is typically used to distinguish between these two entities. To discuss the difficulties in their preoperative differential diagnosis, we present the clinical and dermoscopic characteristics of two pigmented actinic keratoses. Keratinocytic tumors typically appear on sun-damaged skin. Although there are insufficient precise epidemiologic data regarding the frequency with which AKs progress into invasive carcinoma, they are biologically regarded as a type of Squamous Cell Carcinoma (SCC) in situ. Patients with multiple AKs are thought to have a 14% cumulative risk of developing SCCs, and AKs may also be associated with other skin cancers like basal cell.

Development of Actinic Keratoses

Actinic keratoses typically present as thick, crusty, or scaly patches that frequently feel dry or rough. They typically measure between 2 and 6 millimeters in diameter, but they can reach several centimeters. Quite, AKs are much of the time felt before they are seen, and the surface is once in a while contrasted with sandpaper. They might be pink, red, dark, light, tan, or a combination of all of these, or they might be the same color as the skin around them. Actinic Keratoses (AKs) typically manifest as a white, scaly, and varying in thickness plaque surrounded by redness; When felt with a gloved hand, they have a texture similar to sandpaper. The skin around the lesion frequently shows signs of sun damage, such as yellow or pale patches with hyperpigmentation and noticeable changes in pigmentation; additionally, it has deep wrinkles, a coarse texture, purpura and ecchymoses, dry skin, and scattered telangiectasias. Photoaging prompts a collection of oncogenic changes, bringing about an expansion of transformed keratinocytes that can appear as AKs or other neoplastic developments. It is possible to develop multiple AKs in a single skin area after years of sun damage. Field cancerization is the name given to this condition. It is hypothesized that exposure to Ultra Violet (UV) light causes mutations in the epidermis's keratinocytes, facilitating their survival and proliferation. AKs have been linked to both UV-A and UV-B radiation. UV-A light, which has a wavelength of 320–400 nm, can penetrate the skin more deeply and cause reactive oxygen species to form. These oxygen species can then cause damage to cell membranes, signaling proteins, and nucleic acids. The formation of thymidine dimers in DNA and RNA by UV-B radiation (wavelength 290–320 nm) results in significant cellular mutations. In particular, mutations in the p53 tumor suppressor gene have been detected in between 30% and 50% of skin samples from AK lesions. Arachidonic acid and other molecules associated with inflammation have also been shown to be elevated in response to UV radiation. These alterations eventually result in the development of AKs over time. Actinic keratosis is typically diagnosed by a thorough physical examination that combines touch and visual observation. However, in order to ensure that the keratosis is not a skin cancer, a biopsy may be required if it is thick, has a large diameter, or is bleeding.

Actinic keratosis can develop into invasive Squamous Cell Carcinoma (SCC), but the two conditions can look similar on physical examination and can be hard to tell apart clinically. To distinguish AK from in situ or invasive SCC, a histological examination of the excision or biopsy lesion may be required. Seborrheic keratoses, basal cell carcinoma, lichenoid keratosis, porokeratosis, viral warts, erosive pustular dermatosis of the scalp, pemphigus foliaceus, inflammatory dermatoses like psoriasis, and melanoma can all be mistaken for AKs. UV exposure causes the accumulation of genetic lesions that make it easier for skin cancer to develop. Numerous pharmacologic agents are currently in development to enhance DNA repair and prevent DNA lesions from forming. Before being tested on humans, drugs must first be evaluated *in vitro*, which is currently carried out in cell culture systems. The diverse cellularity and architecture of intact human skin cannot be taken into account by current systems. Human skin is profoundly altered by ultraviolet radiation from the sun. Inflammatory erythema, injury-response pigmentation, and immunologic changes occur after an acute exposure. Negative changes in cutaneous structure and function and the neogenesis of the most common human cancers are both caused by chronic exposure. This article discusses photobiology, sunburn, skin pigmentation and types, immunologic changes, and the most common cancers caused by solar radiation.

Matrix Metallo Proteinase

The three most common types of skin cancer, Basal Cell Carcinoma (BCC), Squamous Cell Carcinoma (SCC), and

Melanoma, are individually addressed in this review of the history and epidemiological evidence of the link between sun exposure and skin cancer. There is strong evidence that sun exposure is the cause of BCC, SCC, and melanoma. The rates of BCC, SCC, and melanoma are higher in fair people who are more sensitive to the sun than in people with darker skin who are less sensitive to the sun. The risk is also higher as the amount of ambient solar radiation increases, with the highest densities on the parts of the body that are most exposed to the sun and the lowest densities on the parts of the body that are not Skin cancer incidence must be reduced if sun protection is to be effective. The epidemiological data suggest that implementing sun protection should avoid increasing intermittent exposure, that sun protection will have the greatest impact if implemented early in life, and that it will probably have an impact later in life, particularly in those who were exposed to a lot of solar radiation as children. Actinic keratosis is very common, accounting for about 14% of dermatology visits. It occurs more frequently in people with fair skin, and rates vary depending on age and location. The development may also be influenced by immunosuppression, certain phenotypic characteristics, and exposure to Ultra Violet (UV) radiation. To aid in diagnosing which AKs are more likely to progress into cutaneous or metastatic SCC, researchers are examining the role of novel biomarkers. Many different kinds of cancer have MMP (Matrix Metallo Proteinase) upregulation, and it has been found that SCC has higher levels of MMP expression and production in particular. Serpins, peptidase inhibitors, are also being studied for their function. SCC tumor progression *in vivo* was correlated with SerpinA1 upregulation, which was found to be elevated in the keratinocytes of SCC cell lines. Providers may be able to better assess prognosis and select the most effective treatment options for specific lesions with the assistance of additional research into particular biomarkers.