Commentary

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Foundational Microorganism use in a Patient with an Ischemic Foot Ulcer: A Contextual Analysis

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Description

Actinic Keratosis (AK) sometimes called solar keratosis or senile keratosis is a pre-cancerous area of thick, scaly, or crusty skin. Actinic keratosis is a disorder of epidermal keratinocytes that is induced by Ultraviolet (UV) light exposure. These growths are more common in fair-skinned people and those who are frequently in the sun. They are believed to form when skin gets damaged by UV radiation from the sun or indoor tanning beds, usually over the course of decades. Given their pre-cancerous nature, if left untreated, they may turn into a type of skin cancer called squamous cell carcinoma. Untreated lesions have up to a 20% risk of progression to squamous cell carcinoma, so treatment by a dermatologist is recommended. Actinic keratoses characteristically appear as thick, scaly, or crusty areas that often feel dry or rough. Size commonly ranges between 2 and 6 millimeters, but they can grow to be several centimeters in diameter. Notably AKs are often felt before they are seen and the texture is sometimes compared to sandpaper. They may be dark, light, tan, pink, red, a combination of all these, or have the same color as the surrounding skin.

Squamous Cell Carcinoma Based on Clinical Examination

Given the causal relationship between sun exposure and AK growth, they often appear on a background of sun-damaged skin and in areas that are commonly sun-exposed, such as the face, ears, neck, scalp, chest, backs of hands, forearms, or lips. Because sun exposure is rarely limited to a small area, most people who have an AK have more than one.

If clinical examination findings are not typical of AK and the possibility of in situ or invasive Squamous Cell Carcinoma (SCC) cannot be excluded based on clinical examination alone, a biopsy or excision can be considered for definitive diagnosis by histologic examination of the lesional tissue. Multiple treatment options for AK are available. Photodynamic Therapy (PDT) is one option the treatment of numerous AK lesions in a region of the skin, termed field cancerization. It involves the application of a photosensitizer to the skin followed by illumination with a strong light source. Topical creams, such as 5-fluorouracil or imiquimod, may require daily application to affected skin areas over a typical time course of weeks. Cryotherapy is frequently used for few and well-defined lesions, but undesired skin lightening, or hypopigmentation, may occur at the treatment site. By following up with a dermatologist, AKs can be treated before they progress to skin cancer. If cancer does develop from an AK lesson, it can be caught early with close monitoring, at a time when treatment is likely to have a high cure rate.

Photo-aging Leads to an Accumulation of Oncogenic Changes

Actinic Keratoses (AKs) most commonly present as a white, scaly plaque of variable thickness with surrounding redness; they are most notable for having a sandpaper-like texture when felt with a gloved hand. Skin nearby the lesion often shows evidence of solar damage characterized by notable pigmentary alterations, being yellow or pale in color with areas of hyperpigmentation; deep wrinkles, coarse texture, purpura and ecchymoses, dry skin, and scattered telangiectasias are also characteristic.

Photo-aging leads to an accumulation of oncogenic changes, resulting in a proliferation of mutated keratinocytes that can manifest as AKs or other neoplastic growths. With years of sun damage, it is possible to develop multiple AKs in a single area on the skin. This condition is termed field cancerization. The lesions are usually asymptomatic, but can be tender, itch, bleed, or produce a stinging or burning sensation. AKs are typically graded in accordance with their clinical presentation: Grade I (easily visible, slightly palpable), Grade II (easily visible, palpable) and Grade III (frankly visible and hyperkeratotic).

The most important cause of AK formation is solar radiation, through a variety of mechanisms. Mutation of the p53 tumor suppressor gene, induced by UV radiation, has been identified as a crucial step in AK formation. This tumor suppressor gene, located on chromosome, allows for cell cycle arrest when DNA or RNA is damaged. Dysregulation of the pathway can thus result in unchecked replication of dysplastic keratinocytes, thereby serving as a source of neoplastic growth and the development of AK, as well as possible progression from AK to skin cancer. Other molecular markers that have been associated with the development of AK include the expression of p16ink4,

p14, the CD95 ligand, TNF-related apoptosis-inducing ligand (TRAIL) and TRAIL receptors and loss of heterozygosity.

Evidence also suggests that the Human Papillomavirus (HPV) plays a role in the development of AKs. The HPV virus has been detected in AKs, with measurable HPV viral loads (one HPV-DNA copy per less than 50 cells) measured in 40% of AKs. Similar to UV radiation, higher levels of HPV found in AKs reflect enhanced viral DNA replication. This is suspected to be related to the abnormal keratinocyte proliferation and differentiation in AKs, which facilitate an environment for HPV replication. This in turn may further stimulate the abnormal proliferation that contributes to the development of AKs and carcinogenesis is a pigment in the epidermis that functions to protect keratinocytes from the damage caused by UV radiation; it is found in higher concentrations in the epidermis of darker-skinned individuals, affording them protection against the development of AKs. Fair-skinned individuals have a significantly increased risk of developing AKs when compared to olive-skinned individual and AKs are uncommon in dark-skinned people of African descent. Other phenotypic features seen in fair-skinned individuals that are associated with an increased propensity to develop AKs include.

Physicians usually diagnose actinic keratosis by doing a thorough physical examination, through a combination of visual observation and touch. However a biopsy may be necessary when the keratosis is large in diameter, thick, or bleeding, in order to make sure that the lesion is not a skin cancer. Actinic keratosis may progress to invasive Squamous Cell Carcinoma (SCC) but both diseases can present similarly upon physical exam and can be difficult to distinguish clinically. Histological examination of the lesion from a biopsy or excision may be necessary to definitively distinguish AK from in situ or invasive SCC. In addition to SCCs, AKs can be mistaken for other cutaneous lesions including seborrheic keratoses, basal cell carcinoma, lichenoid keratosis, porokeratosis, viral warts, erosive pustular dermatosis of the scalp, pemphigus foliaceus, inflammatory dermatoses like psoriasis, or melanoma.