Earliest stage treatment of actinic keratosis/non melanoma skin cancer.

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Introduction

Depending on age, lifestyle, and skin type, squamous cell carcinoma (SCC), a non melanoma skin cancer (NMSC) may develop in sun-exposed skin. Actinic keratosis (AK) represents an early or in situ SCC [1-4]. Pathogenesis of AK can be explained by potentially carcinogenic UV light interacting with keratinocyte DNA where DNA repair mechanisms fail. AK evolve slowly in the basal layer until they become thicker and clinically evident as coarse erythematous patches in early stages which may become hyperkeratotic later on (Figure 1) [1,4-6]. Topical imiquimod has been shown to be useful in clearing AK lesions [7-11]. Imiquimod as a toll-like receptor 7 (TLR-7) agonist induces cytokines, starting an inflammatory skin reaction directed primarily against malignant or virus-infected cells, but has virtually no effect on normal skin.

Imiquimod 5% cream is licensed in the USA (FDA) and Europe (EMA) for the treatment of external genital warts, superficial basal cell carcinoma, and AK, and is being experimentally used in various other dermato-oncological conditions [12-14]. A lesser concentration of imiquimod 3.75% cream is available for the treatment of AK on face and scalp [15].

ABOUT THE STUDY

Two case reports on the treatment of facial uv-exposed skin shall open the discussion if subclinical actinic keratoses can be detected by the use of imiquimod cream in uv-exposed areas even if no lesions can be found clinically.

A 87-year old female showing small scaly AK lesions on her right cheek was treated with imiquimod 3.75% cream. Inflammatory reaction developed from day three onwards and showed field cancerisation, the lesions healed without scarring.

A 59-year old female without obvious clinical signs of uv-damage on the face experimentally applied imiquimod 3.75% cream twice daily on the entire face for two weeks. At the end of the treatment phase distinct signs of inflammation appeared, and then taking two weeks for healing without sequelae.

According to the FDA drug safety data report: Imiquimod revealed no evidence of mutagenic or clastogenic potential based on the results of five in vitro genotoxicity tests and three in vivo genotoxicity tests [16,17].

Depending on the country one course of treatment using 28 sachets of imiquimod 3.75% cream will cost approximately US $ 150. This seems comparatively economical in contrast to the cost of surgical interventions when the disease has come to an advanced stage [18,19] (Figure 1).

CONCLUSION

Taking into account that actinic keratoses as a result of proliferating uv-damaged keratinocytes develop quietly from the dermoepidermal basal cell layer in both directions upwards to the skin surface and invasively into the dermis, earliest stage treatment can prevent non melanoma skin cancer formation.

My personal approach as a clinical dermatologist aims primarily at the „public health factor“. As therapists we are trained to focus on health and happiness of our patients. Successful prevention of diseases categorized as malignant could be named the utmost success in medical treatment. In this case we reach this excellent goal at very low cost and minimal suffering.

These results open the discussion if the use of imiquimod 3.75% cream could be recommended preventively in uv-exposed skin areas to obviate a later development of actinic keratoses AK/squamous cell carcinoma SCC/non melanoma skin cancer NMSC.

References


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