

Management of Actinic Keratosis

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A large number of the UK population leave Britain on vacations to sun-drenched lands and over-expose themselves to long hours in strong sunlight. They lie over long on beaches and the decks of cruise-ships with resultant sunburn and skin damage. This can present as Actinic keratosis (AK). This is a commonly occurring condition in general practice. Returning travellers and those repeatedly exposed to the sun present to travel health clinics and GP surgeries. It occurs particularly among fairer skinned people and on ageing skin, presenting as rough erythematous plaques on the skin which can become thickened and horny. They commonly present on chronically sun-exposed skin of the face and back of hands. They are precancerous cutaneous lesions which rarely occur on their own and can be a precursor to cutaneous malignancy. One study in the UK reports 34% of men and 18% of women over the age of 70 have had an actinic keratosis lesion and worldwide prevalence is estimated at between 11-25%.¹

Once they appear they can progress to persistent lesions that do not ultimately alter into squamous cell carcinoma, or may resolve spontaneously. Some do however progress into invasive squamous cell carcinoma (SCC).² Health professionals advising potential travellers in travel health clinics should educate their clients, particularly the fair, the aged and immunocompromised on the importance of skin protection and the avoidance of exposure to the sun when at its strongest in the middle of the day or by the seaside.

The rate of malignant transformation of AKs may be as low as 1000 per annum but some authorities consider AKs to be at one end of a spectrum of carcinogenesis, with as many as 10% progressing to squamous cell carcinoma, especially if the individual is immunocompromised.³ This difference of opinion results in dubiety as to whether all lesions must be treated. The European Forum Guidelines recommend treatment of all AKs as it cannot be predicted which will progress to malignancy.

Diagnosis

Histologically the main feature is epidermal dysplasia. Diagnosis is primarily clinically based on observation. AKs are typically demarcated, keratotic, with a varying degree of erythema and less than 1 cm in size.

Differential diagnosis.

This will include superficial basal cell carcinoma and seborrheic keratosis and melanocytic lentigines. If the risk clinical doubt as to diagnosis then a skin

biopsy should be requested and possible referral to a dermatologist.

Management

If AKs are treated, the risk of developing squamous carcinoma in the surrounding skin is much reduced. Field directed therapy is the objective rather than just targeting specific lesions in line with the concept of field cancerisation - the local regional changes occurring when a nearly of tissue has had long-term exposure to a carcinogen.

Emollients

Topical emollients can be effective in controlling clinical symptoms of the AK such as itching and scaling. 5 fluorouracil (5FU) is an anti-metabolite cytotoxic agent that works by disrupting DNA and RNA synthesis. It provides useful therapy for patients with multiple AKs. About half patients treated with this ointment can expect complete clearance and 90% total reduction in the lesion numbers. Treatment is lengthy; the ointment must be applied twice daily for three weeks and it can cause erythema, pain and sometimes skin ulceration. In two thirds of patients the treatment will be required again in a year. Imiquimod acts as an immune response modifier working as a toll type receptor 7 agonist.

Treatment is three times weekly for four weeks followed by a one month break with a further month of treatment if required. There is an almost 50% complete clearance rate but the side effects are similar to 5FU.

Diclofenac gel available as Solaraze is an anti-inflammatory working primarily by inhibiting cyclo-oxygenase. Typical treatment is twice daily for two or three months. Studies show a 64% reduction in the number of lesions compared to placebo. This long length of treatment requires good compliance for efficacy and there are few side-effects. A shorter mode of treatment is the use of ingenol mebutate given for two or three days only. Side-effects are local erythema, pain, crusting and pustulation.

Cryotherapy

This is commonly used for solitary lesions. It damages the tissue surface through vaporisation of liquid nitrogen which freezes the lesion. The freezing and thawing results in tissue necrosis. Quick and simple it can be practised in the GP surgery. Side-effects include pain, hyperpigmentation and blistering and 75% of the lesions will be clear three months after treatment.

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Dynamic therapy requires the topical application of photosensitiser 5 aminolevulinic acid. After appropriate wavelength treatment this selectively destroys the rapidly dividing keratinocytes. One treatment session is usually adequate. Side-effects include local erythema, pain and crusting of the treated lesion. This is a form of field directed therapy as multiple lesions can be treated in one session.

An atypical lesions not responding to other therapies the dermatologist will use curettage and surgical excision.

Conclusion

Many foreign travellers expose themselves to the sun and damage the skin, resulting in actinic keratosis. Prevalence of actinic keratosis is increasing with an ageing population where many travel abroad and reside in sunny countries for prolonged periods. Actinic keratosis is a pre-cancerous lesion with possible progression to squamous cell carcinoma.

Multiple treatments are available in management with most having good efficacy: cryotherapy, photodynamic therapy, curettage and surgical excision may be required

References

- 1 Guidelines for the management of the actinic keratosis. British Journal of Dermatology 2007; 156: 222-230
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- 3 Malignant transformation of solar keratosis to squamous cell carcinoma Lancet 1988; 1 – 8589.



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