Answer to Dermacase *continued from page 691*

2. Actinic keratosis

Actinic keratosis (AK), or solar keratosis, is one of the most common presentations in dermatology clinics. A precancerous lesion, AK has the potential to develop into invasive squamous cell carcinoma (SCC). The average rate of progression ranges from 0.025% to 16% per year; however, the exact rate is controversial.[1] The most important risk factors for developing AK are fair skin and cumulative sun exposure.[2] As such, it is seen almost exclusively in white people with Fitzpatrick skin phototypes I and II; prevalence increases with age, outdoor occupation, and geographic location closer to the equator.[3] Other risk factors include immunosuppression (eg, organ transplant patients) and genetic skin disorders, such as xeroderma pigmentosum.[2] Men are more frequently affected.

Typically, AK presents as rough, scaly, poorly demarcated erythematous papules or plaques on sun-exposed areas of the skin. Lesions can be skin-coloured or yellow-brown, although this is less common. A pigmented brown-black variant also exists. Scales are adherent, difficult to remove, and often felt before they can be seen. Over time, lesions can thicken or become hypertrophic.[4] Usually asymptomatic, AK might present (uncommonly) with tenderness, burning, or pruritus.[2]

Actinic keratosis occurs as multiple scattered or confluent lesions on the head, neck, and dorsa of the hands and forearms. On the face, the nose, cheeks, temples, and forehead are most frequently affected. In male patients, lesions might occur on the ears and scalp as well, particularly in the presence of androgenic alopecia. Actinic keratosis tends to arise on a background of dry, sun-damaged skin with wrinkles, yellowish discolouration, blotchy pigmentation, and telangiectasia.[2]

Diagnosis

Most often diagnosed clinically, the differential diagnosis includes seborrheic keratosis, Bowen disease (SCC in situ), SCC, superficial basal cell carcinoma, and lentigo maligna.[2,5] Seborrheic keratosis can be differentiated from AK by its well-defined margins and greasy "stuck on" appearance. Bowen disease and superficial basal cell carcinoma typically present with larger well-defined plaques. However, SCC is clinically indistinguishable from hypertrophic AK. Likewise, lentigo maligna cannot be differentiated with certainty from pigmented AK. Biopsy should be considered for thickened or pigmented papules.

Treatment

Actinic keratosis can regress, remain unchanged, or progress to SCC.[6] Up to 25% of AK spontaneously disappears within a year.[7] However, new lesions develop and patients still experience spreading of AK with age.

The rationale for treating AK is to prevent progression to SCC. As it is not possible to determine which papules will progress to SCC, treatment of all AK-related lesions is recommended.[8] Several highly effective treatments are available. Cryotherapy with liquid nitrogen is most common. Liquid nitrogen can be sprayed or applied with a cotton-tipped applicator. More intense, lengthy application might be required for hypertrophic lesions. Most AK will clear with cryotherapy, although several treatment sessions might be required. Disadvantages include pain and discomfort during treatment, dyspigmentation, redness, and blistering after treatment, which can take 2 to 3 weeks to heal.[9] Cryotherapy is the treatment of choice for patients with few lesions although it can be used to treat widespread AK as well. Curettage with or without electrodesiccation is also useful and effective for treating a small number of lesions.

Topical chemotherapeutic agents, such as 5-fluorouracil cream (5-FU), imiquimod, and, less common, tretinoin and diclofenac gel, are also used to treat AK but are generally reserved for patients with widespread lesions. Five percent 5-FU cream is applied twice daily to affected areas for 2 to 4 weeks. Within a few days, the lesions become erythematous, crusted, and eventually erode. Patients experience pain, burning, and pruritus.[9] Although effective, 5-FU causes substantial irritation and temporary cosmetic disfigurement. Patients should be warned about these side effects. Typically, 5% imiquimod cream is applied once daily, 3 times a week, for 16 weeks; however, other regimens have been developed to reduce discomfort and increase compliance. Side effects are similar to 5-FU but might be better tolerated.

Other treatments include topical 5-aminolevulinic acid photodynamic therapy, dermabrasion, chemical peels, laser surgery, and oral retinoids. Despite the many effective treatments available, the best treatment is prevention. Sun-protection measures, such as protective clothing and sunscreen, are recommended.[8]

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Competing interests

None declared

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