

Answer to Dermacase continued from page 985

4. Keratoacanthoma

Keratoacanthoma (KA) is a neoplasm of epithelial cells, and typically affects fair-skinned, middle-aged to older individuals; it has a slightly higher predominance in males.¹⁻³ It usually appears as a solitary, nontender, flesh-coloured or pink nodule with a central-crust, keratotic plug on hair-bearing, sun-exposed areas, primarily the face, neck, and hands.¹⁻³ The lesion is characterized by rapid growth and achieves an average diameter of 2.5 cm within 6 to 10 weeks.^{1,3} In approximately 50% of cases, spontaneous resolution of the tumour occurs within 4 to 9 months of achieving maximal size, although an atrophic and pigmented scar usually remains.^{1,3,4}

The etiology of KA is unknown, but it is thought to derive from hair follicles.¹⁻³ Additionally, several factors have been postulated to be involved in its development: Poor immunocompetence, UV radiation, mineral oil, cigarettes, and chemical carcinogens, such as tar and pitch, all might play an etiologic role.^{1,2} Not uncommonly, KA forms in areas of trauma, such as surgical sites or areas subject to laser resurfacing.¹⁻³ Certain types of human papillomavirus have also been implicated¹ and DNA related to the virus has been found in some KA tumours.²

Keratoacanthoma might present with clinical and pathological findings similar to those of squamous cell carcinoma (SCC), and many SCC lesions resemble KA lesions.^{2,3} Classification of KA is therefore a source of controversy; it could be a distinct entity, but also has been proposed as a variant of cutaneous SCC.^{1-3,5} Because spontaneous involution of the tumour has been described, some consider KA lesions to be benign; however, their potential for evolution into invasive SCC lesions that might metastasize, as well as their potential for recurrence following excision, could suggest otherwise.¹⁻⁵

Diagnosis and management

Upon presentation of the lesion, KA can usually be diagnosed based on its distinctive clinical history; however, because of a lack of features distinguishing KA from SCC in appearance, it is suggested that KA be considered as potential SCC and handled accordingly.^{1,2,5} A biopsy is typically performed to rule out SCC and a sample of sufficient depth is required.^{1-3,5} Recent studies have suggested chromosomal differences in the 2 conditions, which can be detected using comparative genomic hybridization^{1,2}; however, cytogenetics is not currently a standard investigation for KA.



A variety of treatment modalities are available for KA. Intralesional 5-fluorouracil, topical imiquimod, cryosurgery, electrodesiccation and curettage, wide local excision, radiation therapy, and laser therapy have all been successfully employed to treat small, solitary lesions.¹⁻⁴ In areas of cosmetic importance, or if the lesions are large or invasive, referral for Mohs micrographic surgery is preferred.^{1,2} Should a patient or physician seek a nontreatment approach, regular follow-up sessions with a dermatologist who can observe and photograph the lesion from presentation to complete resolution is recommended.^{1,4} Even in cases where the KA is expected to regress on its own, treatment is a reasonable option for anxiety reduction and cosmetic purposes.¹⁻⁴

It is also important to reinforce conservative measures, such as minimizing contact with those aforementioned factors that might play a role in the development of KA (chemical carcinogens, cigarettes, etc). Excessive sun exposure should be avoided and sunscreen should be applied regularly to reduce scarring and prevent recurrence.¹

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Competing interests

None declared

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