Read Item - Keratoacanthoma And Benign Self-Healing Epithelioma

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Publisher/Journal: **Keywords:** Keratoacanthoma Benign Self-healing epithelioma

Abstract: Doctor's resource on Keratoacanthoma And Benign Self-Healing Epithelioma

Keratoacanthoma And Benign Self-Healing Epithelioma

Introduction

A keratoacanthoma (KA) is a rapidly evolving skin nodule that is histologically malignant but biologically benign. It occurs predominantly in an elderly balding population in response to solar radiation.

It has been suggested that keratoacanthoma arise from hair follicles and the growth phase corresponds to the anagen phase of hair growth and the involution and apoptosis that accompanies resolution may correspond to catagen. While this hypothesis is attractive, the occurrence of keratoacanthomas on non-hair bearing sites such as the mucous membranes, palms and soles and the sub-ungual region confounds this theory.

Clinical Features and Investigation

Less than 5% of all keratoacanthomas occur on the scalp. They appear suddenly and grow rapidly to a size of 1-2 cm in about 6 weeks. Initially the lesions are non-discript papules resembling molluscum contagiosum of a viral wart. By 6 weeks they are characteristic solitary, symmetrical, flesh-coloured, dome shaped nodules with a central keratin plug. In contrast to squamous cell carcinoma (SCC) there is no spreading inflammation at the base.

The lesions remain unchanged for about 6 weeks before undergoing spontaneous involution. Resolution begins with softening of the lesion and expulsion of the central keratin plug. The rim then flattens and leaves behind only a puckered scar.

Clinical variants include giant keratoacanthoma, which may grow to 5 or more centimetres, and multiple keratoacanthomas of the Ferguson-Smith type or the Grzybowski type. Occasionally multiple lesions may be a feature of the Muir-Torre syndrome. The multiple lesions are very rare, occur in younger people and are often inherited by autosomal dominant transmission. Many of the multiple type KAs do not have the central keratin plug and are identical histologically to SCC. These lesions can only be differentiated from SCC by their biological behaviour.

The histology of keratoacanthoma shows a symmetrical exo-endophytic lesion in the upper dermis and extending no deeper than the sweat gland coils. A central mass of keratin is surrounded by columns of acanthotic epidermis that forms a colarette. The cells show some cytological atypia, but atypical mitoses are not common. The keratinocytes have an eosinophilic cytoplasm and there is a lymphocytic infiltrate at the base of the lesion with scattered neutrophils and eosinophils. Distinction from a well-differentiated SCC can be difficult at times, however the architecture and cellular morphology allow most to be distinguished.

Management

On the basis of the clinical and histological features, a prediction of biological behaviour can be made. In the presence of a normal immunological system a KA may be left alone to resolve spontaneously. If however there is immunodeficiency (for example post-organ transplantation) then the lesions tend not to involute and may metastasise, albeit rarely. Such lesions should be removed, as should any lesion that threatens to cause local destruction to the nose or eyelids.

KAs that may leave an disfiguring scar after healing, that are still enlarging or that are difficult to diagnose with certainty clinically, should also be excised. In practise this encompasses the majority of keratoacanthomas and only a relatively small percentage are best left to regress spontaneously. A shave biopsy followed by curettage and cautery is usually sufficient. Alternatively an elliptical excision has a slightly lower rate of recurrence. Cryotherapy is less effective and is not recommended for treatment however, radiotherapy may be successful.

Key Points

Between 1 and 5% of keratoacanthomas occur on the scalp. Classically they enlarge for about 6 weeks, plateau for 6 weeks and involute spontaneously over 6 weeks.