CLINICOPATHOLOGIC REPORT

Well-Differentiated Squamous Cell Carcinoma of the Eyelid Arising During a 20-Year Period

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An 81-year-old man had a keratotic eyelid lesions for 20 years. He eventually sought treatment by ophthalmic plastic surgery. Clinically, the lesion resembled a keratoacanthoma. Findings from histologic examination of the excision biopsy specimen showed a squamous cell carcinoma. The lesion was completely excised. This case demonstrates the difficulty in making a correct clinical diagnosis of a keratotic eyelid lesion. Performing a histologic examination of nonregressed keratotic lesions is essential to exclude a squamous cell carcinoma.

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The relationship between keratoacanthoma and squamous cell carcinoma (SCC) is controversial. We report a case of an older man with a long-standing keratotic eyelid lesion, which probably represented a nonregressed keratoacanthoma that at some stage had undergone focal malignant degeneration to a well-differentiated SCC.

REPORT OF A CASE

An 81-year-old man complained that an eyelid lesion that was present for 20 years was interfering with his vision and that his family was embarrassed by his appearance. He had regularly trimmed the keratin horns growing from his right upper eyelid, but these continued to regrow, and the lesion enlarged steadily during the 20-year period. He reported no recent change in its growth pattern, no pain or bleeding, and was systemically well. An incisional biopsy specimen was obtained from the edge of the lesion by dermatologists and showed actinic keratosis. He was referred to the ophthalmic plastic surgery clinic for excision of the entire lesion.

He had severe right mechanical ptosis with compensatory bilateral frontalis muscle overaction (**Figure 1**). There was a multinodular mass with several keratotic horns arising from both the pretarsal and preseptal upper eyelid (**Figure 2**). The tumor was not tethered to the underlying tarsal plate or orbital structures. His visual acuity was 20/60 OU (when the eyelid was lifted off the visual axis), and ocular findings were normal.

A large and deep incisional biopsy specimen was obtained from one of the lesions by the ophthalmic plastic surgeons; findings from examination confirmed SCC. The patient declined micrographic surgery under local anesthesia. He underwent an excisional biopsy under general anesthesia, which included part of the underlying orbicularis oculi muscle and most of the eyelashes but spared the tarsal plate. A large anterior lamellar defect required reconstruction (**Figure 3**). This necessitated repair by the placement of free skin grafts from the ipsilateral postauricular area and from the contralateral upper eyelid. His recovery was satisfactory with no residual ptosis (**Figure 4**), and he experienced only mild lagophthalmos without exposure keratitis. There has been no recurrence of the lesion 1 year later.

Two specimens were excised for histopathological examination (**Figure 5**). The main lesion measured 3.5 × 2.5 cm in area, extending up to 1 cm in depth. Findings from histologic examination showed actinically damaged skin containing an ulcerated hyperkeratotic, well-differentiated, focally keratinizing, inva-

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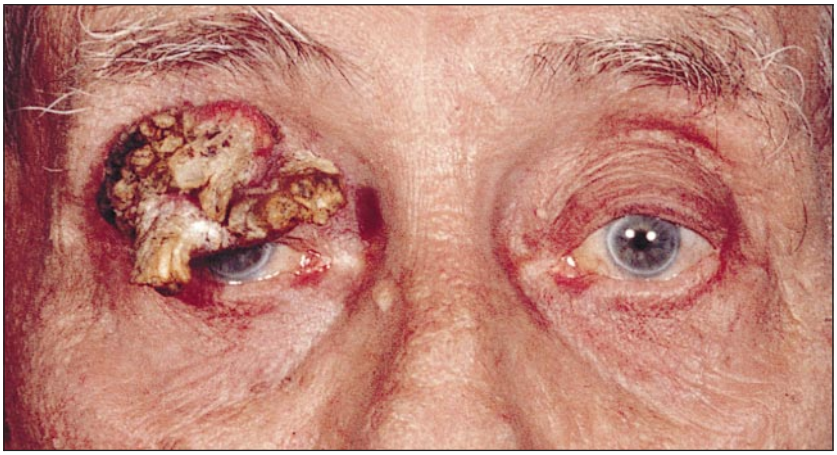

Figure 1. *Right mechanical ptosis caused by large keratotic eyelid tumor.*

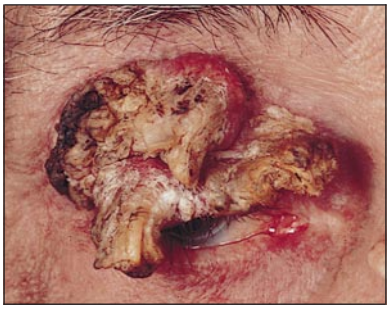

Figure 2. *Keratotic tumor; right upper eyelid.*

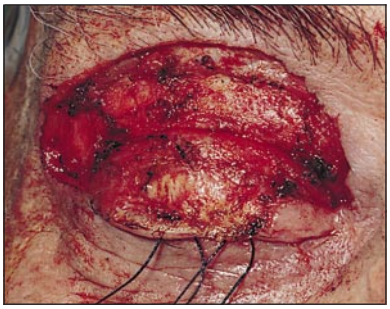

Figure 3. *Extent of perioperative defect after excision biopsy.*

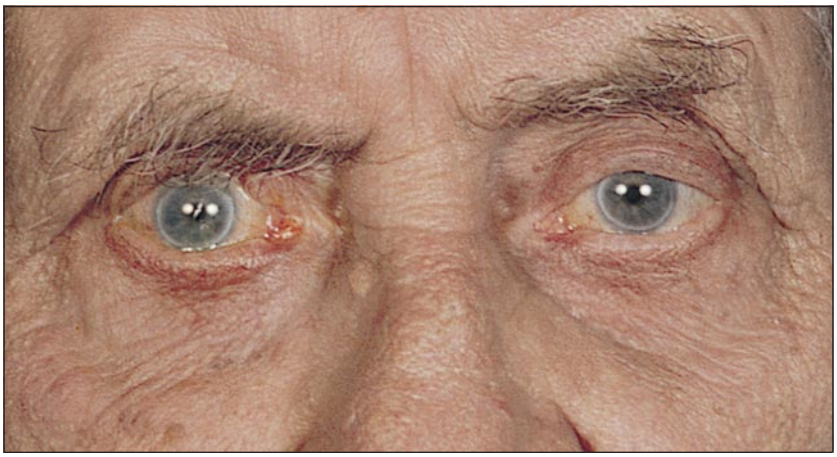

Figure 4. *Appearance 6 months after excision of tumor and reconstruction.*

sive SCC, which was completely excised. There was an associated marked inflammatory response and a focal giant-cell reaction in relation to the keratin. The second smaller specimen was of an involved area superomedial to the main lesion. This measured 1.2 × 0.5 cm in area and 0.2 cm deep. Results of histologic examination showed prominent actinic damage with focal severe dysplasia amounting to the intraepidermal carcinoma, which was completely excised.

COMMENT

This patient had a long-standing keratotic eyelid lesion. Clinically, it appeared to be a keratoacanthoma (KA), but findings from histologic examination revealed SCC. The chronicity suggested that this lesion may have initially been a KA that failed to regress and underwent focal malignant degeneration to a well-differentiated SCC, perhaps many years ago.

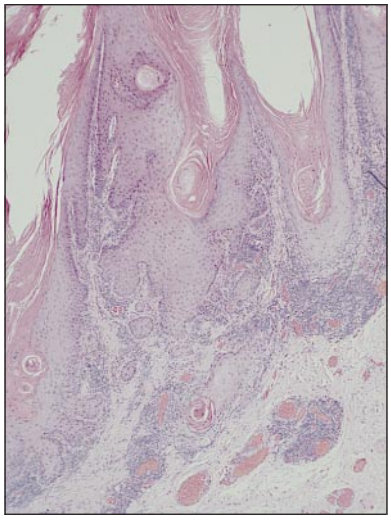

Figure 5. *Low-power photomicrograph showing hyperkeratotic dysplastic squamous epithelium and islands of invasive focally keratinizing squamous cell carcinoma (hematoxylin-eosin, original magnification ×4).*

Periocular SCC is an aggressive neoplasm, accounting for between 2.4% and 30.2% of malignant eyelid lesions but for fewer than 2% of all eyelid lesions.[1] Cutaneous SCC has several histopathological subtypes including adenoid/acantholytic, mucin producing, and verrucous. The prognosis is based on location, histologic characteristics, cause, and clinical features.[2]

Periocular SCC commonly arises in cases of solar keratoses. Keratoacanthoma is an epithelial tumor bearing a close clinical and histological resemblance to SCC but has the potential to regress spontaneously. Both KA and SCC appear in fair-skinned individuals who have a history of chronic sun exposure. The exact relationship between KA and SCC is controversial. Some authors consider KA to be a variant of SCC, both falling within a spectrum of varying malignancy.[3-5] The lack of notably different immunohistochemical findings between KA and well-differentiated SCC supports this.[6]

The treatment of keratotic lesions is controversial because of the difficulty in making a precise clinical diagnosis. Options include observation, surgical excision with histologic control of the excision

margins, radiotherapy, and cryotherapy.[7] Whereas KA is capable of spontaneous regression, periocular SCC has an aggressive malignant potential, with prognosis correlating with local recurrence and metastasis rate. These are influenced by treatment modality, location, depth of tumor, histological differentiation, perineural involvement, precipitating factors other than UV light, host immunosuppression, and desmoplastic changes.[8]

Excisional biopsy of keratotic eyelid lesions is recommended rather than observation to see if the lesions regress. In this case, the lesion was large; therefore, the physician removed a large incisional biopsy specimen from the center of the lesion for histologic examination prior to an intended micrographic excision. If an incisional biopsy specimen is obtained, this must include both the edge and base of the tumor and be large enough for the physician to detect focal invasion.[9] Frozen section control, such as micrographic surgery, is preferred for the control of the margins where the tumor has not extended to bone or orbital fat.[1]

Our case highlights the difficulty in making a precise clinical diagnosis of SCC and supports the case for definitive treatment by surgical excision of all suspected KA and SCC. Nonregressed keratotic lesions should be regarded as SCC until proven otherwise. An incisional biopsy specimen from the edge of the lesion may not correctly represent the main diagnosis when there is focally invasive carcinoma, as in our case.

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100 Years Ago in the ARCHIVES

A look at the past . . .

The number of microscopical examinations of eyes with syphilitic disease is not large. Besides some short reports by J. Hutchinson, Bader, Klebs, Furstner, and Pagenstecher, I have found the following accounts in the literature: Edmunds and Brailey make the following summary statement as to syphilis of the interior of the eye: The choroidal vessels seem not to be altered, while those of the retina, both in acquired and congenital syphilis, are thickened, probably from inflammatory processes.

Reference: Nagel G. The examination of two cases of old specific chorioretinitis. *Arch Ophthalmol*. 1900;29:514.