Letter to the Editor

Re: Nasal cartilage degeneration

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Editor.—

The nose is a common site for chronic actinic damage, actinic keratoses, and skin cancer. The histologic changes produced by ultraviolet energy on the epidermis and dermis include disorderly arrangement of keratinocytes, hyperchromasia of keratinocyte nuclei, dyskeratosis, and basophilic or elastotic change of the papillary and upper reticular dermis.[1] Changes in nasal cartilage due to solar exposure have not been reported, possibly due to the depth of the nasal cartilage within the skin and severity of sun damage required to elicit this response. We wish to describe the histologic findings in a nose lesion that showed prominent degeneration of the nasal cartilage. We speculate that these cartilaginous changes were induced by chronic ultraviolet energy and may be similar to those seen in some cases of chondrodermatitis nodularis chronica helicis.

A 67-year-old white man worked as a farmer and had a history of chronic ultraviolet light exposure. Two basal cell carcinomas had previously been diagnosed on the right cheek and forehead and were treated with electrodesiccation and curettage, and Mohs surgery respectively. There was no history of arsenic exposure, and the patient was otherwise in excellent health. Biopsy using a deep shave technique was done on a 4-mm pruritic, chronically ulcerated papule on the left nasal ala.

On routine hematoxylin and eosin staining, alternating focal parakeratosis and basket-weave hyperkeratosis were present. Focal acanthosis and moderate keratinocyte dysplasia with individual cell keratinization were seen laterally, characteristic of an actinic keratosis. In the center of the specimen, the epidermis was replaced by an inflammatory crust. Prominent basophilic degeneration of the papillary and upper reticular dermis was found at the lateral margins of the specimen, which contained abundant sebaceous lobules. The central portion of the tissue showed interlacing fascicles of fibroblasts and histiocytes, admixed with numerous, well-differentiated lymphocytes (*Fig. 1*). We believe this fibrohistiocytic proliferation represents a fibrous tissue reaction in a chronic ulcer. Beneath the inflammatory infiltrate separated by mucinous material, the reticular dermis showed increased fibroplasia. Additionally, there was fibrosis and basophilic degeneration of the perichondrium while the cartilage showed chondrocyte necrosis with eosinophilia and focal vacuolar basophilia (*Fig. 2*).

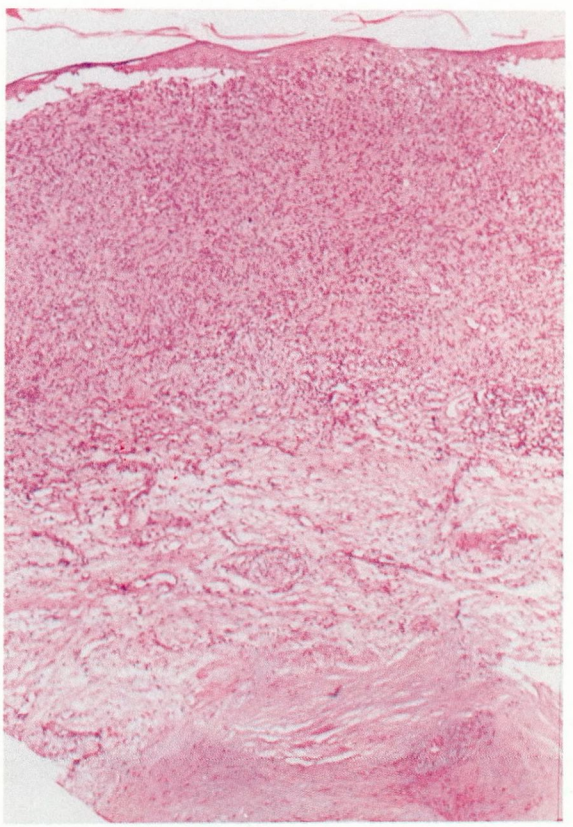

Fig. 1. Photomicrograph showing an epidermal erosion with underlying interlacing bundles of spindle cells and scattered lymphocytes. A clear space appears below the inflammatory cells and prominent fibroplasia of the reticular dermis is also seen. Perichondral fibrosis and degeneration of the cartilage is present (hematoxylin and eosin stain, original magnification × 40).

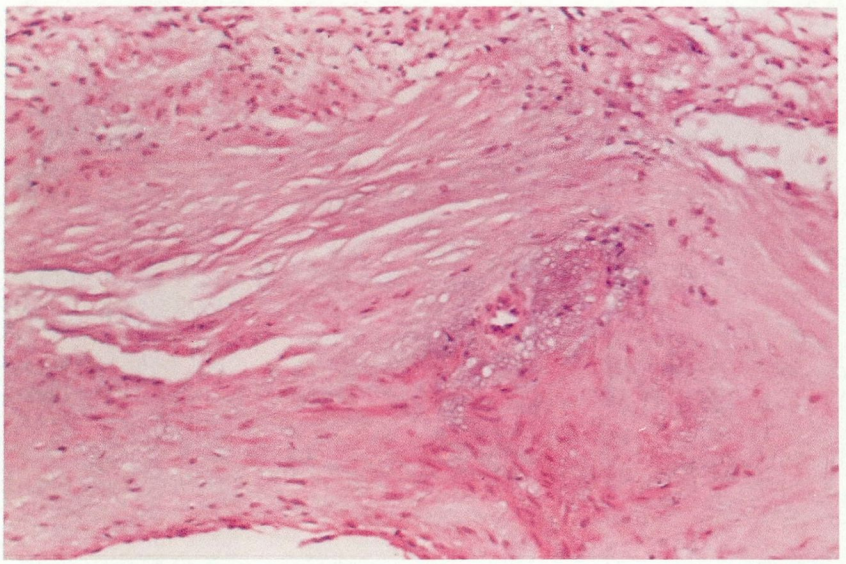

Fig. 2. Higher-power photomicrograph showing perichondral fibrosis, cell necrosis of chondrocytes, and basophilic degeneration of cartilaginous matrix (hematoxylin and eosin stain, original magnification × 100).

Verhoeff van Gieson's stain for elastic tissue displayed marked, focal aggregates of positive-staining material in the upper dermis, representing elastotic material. Colloidal iron stain for mucin showed increased staining diffusely throughout the dermis that corresponded to the mucinous material seen on routine hematoxylin and eosin stain. Periodic acid-Schiff staining was noncontributory to the diagnosis.

The changes seen in the cartilage from the nose in actinically damaged skin from our patient consisted of pyknosis of chondrocyte nuclei, basophilic and mucinous changes of ground substance, and perichondral fibrosis. In view of the patient's history of skin cancer, his occupation, and the anatomic location, this change may be related to chronic exposure to ultraviolet energy. However, aging, dystrophic changes from the overlying fibrous tissue reaction, or other electromagnetic radiation cannot be eliminated as possible etiologic cofactors. In fact, chondrocyte nuclear and matrix changes have been described that were attributable to senescence alone.[2]

A common cutaneous disorder that has features similar to our case is that of chondrodermatitis nodularis chronica helicis (CNCH). CNCH appears as a painful, crusted nodule on the ear that most frequently occurs on the helix of elderly men with a history of prolonged ultraviolet radiation exposure.[2–4] Proposed etiologic factors include chronic trauma, compromised vascular supply to the ear, and ultraviolet radiation injury.[2–4] Although perichondritis or degenerated cartilage may be seen in CNCH, the cartilage may often be normal.[2–7] Recent studies suggest that CNCH is one of the disorders that shows transepidermal elimination.[5–7] Although degeneration of the dermal connective tissue appears to be the predominant pathologic alteration in CNCH, it is possible that the pathologic changes seen in the cartilage in some cases of CNCH are secondary changes due to prolonged, chronic ultraviolet energy.

Further studies are needed to determine the etiologic factors responsible for cartilaginous degeneration. Although we propose chronic ultraviolet energy as one mechanism, other factors may play a role. However, knowledge of this pathologic change in cartilage may provide further information on the pathogenesis of other cutaneous disorders, such as CNCH.

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