axilla and pubis and is likely to be related to an autoimmune pathogenesis (4, 6). Recently, a new autoantigen related to APS I, tyrosine hydroxylase, has been identified in patients affected by alopecia areata (9).

Vitiligo, observed in 8–13% of APS I patients, appears in childhood and is typically associated with the presence of autoantibodies directed against the SOX9 and SOX10 autoantigens (1, 10).

The high incidence of chronic mucocutaneous candidiasis in APS type I, as first presenting feature of the syndrome, and its absence in APS types II and III underline the relevance of chronic mucocutaneous candidiasis as early diagnostic criteria for APS type I. In light of this view, and related to the subsequent onset of the different signs that complete the syndrome, patients affected by chronic mucocutaneous candidiasis in early childhood should have a long-term monitoring dermatological and endocrinological follow-up in order to detect whether, other than the immunological defects commonly associated with this infection, an early diagnosis of APS type I can be suspected and a correct therapy can be established (5, 8).

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A Case of Localized Acne Following Radiation Therapy

Ji youn Song, Seog Jun Ha, Chung Won Kim and Hyung Ok Kim

Department of Dermatology, College of Medicine, The Catholic University of Korea, 505, Banpo-Dong, Seocho-Gu, Seoul, 137-040, Korea. E-mail: knderma@cmc.cuk.ac.kr

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Sir,

Acute and chronic radiodermatitis are well-known, frequent complications of radiation therapy. However, acne eruption localized to the radiation field is an unusual late sequelae of radiation therapy. We describe here a case of localized acne eruption on the neck of a patient who received radiation therapy for laryngeal carcinoma.

CASE REPORT

A 51-year-old man presented with an acne eruption on his neck for 15 days. Four months previously he noticed hoarseness. Laryngoscopic examination showed an exophytic mass on the right true vocal cord and subsequent endoscopic biopsy revealed squamous cell carcinoma of the larynx. The patient received three courses of cisplatin-sodium thiosulfate chemotherapy and concurrent radiotherapy. Initially, 6 MV radiation therapy in a total dose of 5000 cGY was applied to the entire neck, followed by a booster irradiation of 2200 cGy to the glottic region. A total dose of 7200 cGy in 40 fractions over 74 days was therefore applied on a 12×7 cm rectangular area of his neck. He did not have a past history of acne and had not been using steroid or any topical preparation.

On physical examination, there was a sharply defined area of acne eruption consisting of erythema, follicular papules, pustules and black comedones in the anterior neck. The lesion corresponded to the portal field of radiation (Fig. 1) and the density of acne was more compact on the area of the booster irradiation. Biopsy showed open and closed comedones containing keratin plugs with flattened follicular epithelial lining and a few atrophic sebaceous glands. He was treated with retinoic acid gel 0.01% for 2 months and showed improvement.

DISCUSSION

Radiation-induced acne eruptions have been reported following several types of radiotherapy, including super-

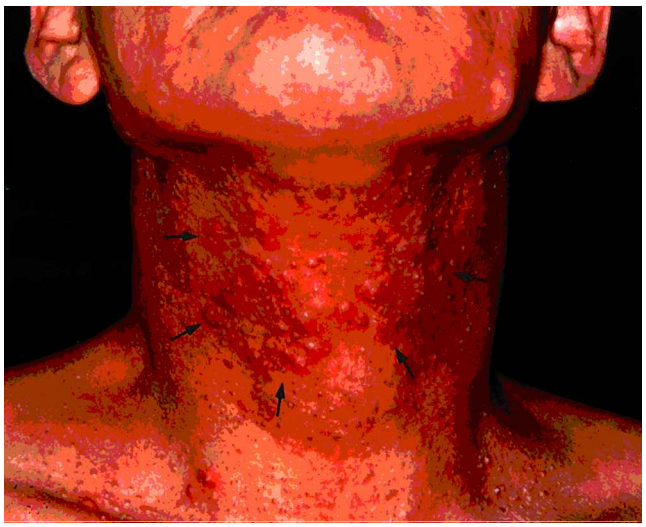

Fig. 1. Acne eruption localized to the radiation field, characterized by pustules, papules and comedones. Anterior view (arrow = the area of booster irradiation).

ficial X-ray therapy, deep cobalt therapy and megavoltage therapy. Despite the skin-sparing advantages of deep cobalt irradiation, skin reactions have been observed in approximately a quarter of the patients treated (1). Most radiation-induced acne eruptions have been developed after superficial X-ray therapy. Little was found in the literature on acne eruptions after deep radiation therapy and only three cases have been reported after megavoltage radiotherapy (2, 3). In our case, acne eruption developed more compactly on the booster irradiation site, and this coincides with the fact that the higher the radiation dose, the greater the cutaneous effect.

The etiology of radiation-induced acne eruption is unknown. Trunnell et al. (4) reported two cases of acne lesions after cobalt irradiation, and found horn plugs in the hair follicle ostia upon skin biopsy. Only remnants of the pilosebaceous apparatus remained, and these were engulfed by foreign-body giant cells. It was therefore suggested that the pilosebaceous remnants act as foreign bodies, leading to an inflammatory reaction that clinically appears as acne. Stein et al. (5) suggested a multifactorial etiology comprising a predisposition to acne vulgaris, concurrent drug therapy such as systemic steroids, and radiation. Engels et al. (6) postulated a difference in sensitivity to radiation between the pilosebaceous duct and glandular elements which leads to an alteration in their secretions, resulting in cell debris and secretions entering narrowed duct openings. In our case, biopsy revealed open and closed comedones containing keratin plugs with flattened follicular epithelial lining and a few atrophic sebaceous glands. We consider radiation to be a direct causative agent in this case, because acne eruptions developed on a confined portal area of radiation and showed compact density on areas of booster radiation.

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