
The tumor showed homogeneous staining for K14 (figure 1E). There was no staining for K10 (figure 1F), involucrin (figure 1G), or filaggrin in the basaloid tumor cells. Positive staining for these markers was observed only in the upper squamoid non-tumor cells. The secretory gland epithelium markers K7 (figure 1H) and epithelial membrane antigen (figure 1I) were detected exclusively in nests of sebocytes and ducts. SESD, which also has been referred to as reticulated acanthoma with sebaceous differentiation [1] and acanthomatous superficial sebaceous hamartoma [2], is a histologically distinct benign neoplasm characterized by a superficial plate-like proliferation of basaloid cells with foci of sebaceous differentiation [2-4]. The additional histological features of SESD include a verruciform structure, pigmentation of the basal layer and lymphoid cell infiltration in the upper dermis. Differential diagnoses from other cutaneous neoplasms with sebaceous differentiation include sebaceoma, sebaceous adenoma, trichoblastoma with sebaceous differentiation, apocrine poroma with sebaceous differentiation and basal cell carcinoma with sebaceous differentiation. However, SESD can be easily differentiated from these other neoplasms histologically by the superficial plate-like proliferation of monotonous small basaloid cells, which is also a histological characteristic of seborrheic keratosis.

The pathogenesis of sebaceous differentiation in SESD is not yet known. Therefore, we examined the expression of keratinocyte differentiation marker molecules in SESD to evaluate the differentiation stage of SESD. The observed staining pattern suggested that the tumor cells do not differentiate towards interfollicular epidermis but rather towards sebfollicular epithelium. This may explain why the overall plate-like configuration and the histological features of basaloid cell proliferation are similar to those seen in seborrheic keratosis, which is a tumor derived from the follicular infundibulum [3].


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Comedogenic acne following radiation therapy

Different types of radiation may induce acneiform eruptions. Favre-Racouchot syndrome, which is characterized by yellow atrophic plaques with large comedones, wrinkles and furrows on sun-exposed areas, is the most commonly seen form of radiation-induced acne [1]. There are also a few case reports in the literature of comedone formation after exposure to ionizing radiation, especially deep cobalt radiation [2-6]. Here, we emphasize this very rare condition and present a case with comedogenic acne after megavoltage radiotherapy.
A forty-year-old male patient was admitted to our outpatient clinic with recently appearing black spots on his face. He had a mass lesion on his right buccal mucosa which had been diagnosed as squamous cell carcinoma 10 months previously and this region had been treated with 50 Gy megavoltage radiotherapy. He claimed that there were no comedones, papules or pustules on his face before and he noticed the lesions 4 weeks after initiating radiation therapy. He did not use any systemic or topical agents that could cause acneiform eruptions and had no history of acne in his adolescence. On dermatological examination there was a hyperpigmented area on his right cheek, chin, periorificial region and the tip of the nose, with sharp margins, which contained open comedones, closed comedones and milia-like cysts (figure 1A, B). Histopathological examination of these lesions revealed large, thin-walled, open and closed comedones with abundant keratin in the upper dermis, but solar elastosis was not seen (figure 1C). The patient was diagnosed as having comedogenic acne following radiotherapy, according to the clinical and histopathological findings. Treatment with topical tretinoin was offered but he declined to use any medicine.

Acneiform reactions to radiation were first defined by Blufarbin in 1947 and only a few similar cases under the names of acneiform reaction, radiogenic acne, comedones or Favre-Racouchot-like disease, have been reported until now [2-6]. We used the term ‘comedogenic acne’ due to the lack of inflammatory acne lesions and elastosis. Open and closed comedones and milia-like cysts limited to the exposure areas are common clinical findings in these cases. Inflammatory acne lesions have rarely been described [3-5]. Radiation-induced acne can occur on any area of the body but it is most commonly seen on the scalp, face and neck [5]. In all reported cases the eruptions were limited within the radiotherapy exposure area and usually developed about 2 weeks to 6 months after completion of radiotherapy [2-6]. It has been reported to occur after several types of radiotherapy, including superficial X-ray therapy, deep cobalt therapy and megavoltage therapy. Deep cobalt therapy is the most reported type of radiotherapy [5]. Our patient had 50 Gy, 6 Megavoltage therapy.

In contrast to its acneigenic effect, radiation itself has been used to treat acne in the past for its ability to suppress pilosebaceous gland activity [5]. The pathogenesis of radiation-induced acne is not clear but it was postulated that, after irradiation, the sebum production and content decrease, pilosebaceous cells are damaged and cellular debris advances in the narrowed openings of the sebaceous glands, and thus comedones occur. The other claimed mechanism is that the radiation initiates chronic follicular inflammation and follicular hyperkeratosis, resulting in the formation of follicular cysts and comedones. Furthermore, deep irradiation leads to the destruction of the hair-sebaceous apparatus, remnants then cause foreign body reactions and induce acneiform lesions [4-6]. The use of some agents such as systemic steroids along with radiotherapy may also act as a facilitator for comedogenesis [4]. The treatment of radiation-induced acne is not different from comedogenic acne. Keratolytics such as benzoyl peroxide and topical or oral retinoids, comedo extraction, curettage and, rarely, excision are the treatments available for this condition [3, 5].


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Figure 1. A) Open and close comedones and milia-like cysts with sharp margins on the radiotherapy induced area. B) Left side of the face which was not radiated. C) Open and closed comedones containing keratin in the upper dermis.

Pulmonary cryptococcoma in a patient with Sézary syndrome treated with alemtuzumab

Sézary syndrome (SS), a severe form of cutaneous T-cell lymphoma (CTCL), may be complicated by life-threatening infections [1]. Cryptococcosis, an opportunistic mycosis


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