Secondary Ossification Associated with Seborrheic Keratosis Adjacent to Basal Cell Carcinoma

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Sir,

An 80-year-old female was referred to our hospital, complaining of a facial tumor which had existed for last 8 years. She denied either prior local trauma or infection at the site. Physical examination showed a relatively well-circumscribed erythematous plaque with infiltration on the left cheek, and in addition, a brownish keratotic slightly-elevated nodule was located adjacenty [Figure 1]. A biopsy specimen showed well-circumscribed basaloid tumor nests in the upper to mid-dermis. The tumor cells were uniform in size with oval nuclei and scant cytoplasm with peripheral palisading, and the tumor nests were surrounded by myxomatous stroma [Figure 2a], which was positively stained by alcian blue, suggestive of basal cell carcinoma. A total resection including the adjacent keratotic tumor was performed. Histological features of the keratotic nodule showed reticular epidermal proliferation containing pseudocysts, extending to the upper dermis [Figure 2b]. Of note, a lamellar bony structure of homogeneous materials containing osteoblastic cells was found in the surrounding stroma [Figure 2c and d].

Secondary ossification is sometimes observed in several skin tumors and inflammatory skin disorders. Among skin tumors, basal cell carcinoma is the most common. The present case developed basal cell carcinoma and seborrheic keratosis in a close location, however, secondary ossification (osteoma) was observed just beneath the seborrheic keratosis, but not associated with basal cell carcinoma. Benign skin tumors showing secondary ossification are most common in melanocytic nevus and pilomatrixoma, whereas other benign tumors are rare, i.e., apocrine hidrocystoma, lipofibroma, neurofibroma, pyogenic granuloma, and keratoacanthoma. To the best of our knowledge, only one case of seborrheic keratosis with secondary ossification was reported; however, details are unknown because it was reported in a large review of a single institute.

Figure 1: Clinical appearance of the nodule on the cheek presenting with an erythematous plaque (arrowhead) and keratotic nodule adjacenty (×).
The mechanism of secondary ossification is still unknown. Osteoblast plays a central role in the bone formation. Osteoblast secrets several inducing factors such as bone morphogenetic protein (BMP)-2, BMP-4, β-catenin, osteopontin, osteonectin and osteocalcin, which exert an effect on the precursor cells derived from mesenchymal cells. BMPs are members of transforming growth factor (TGF)-β, and transform the fibroblasts, primitive mesenchymal cells, or progenitor cells in the bone marrow into osteoblasts. BMPs interact with the extracellular matrix, which may contribute to the transformation of primitive mesenchymal cells into osteoblasts forming bony tissues. In addition, previous studies suggested that TGF-β and connective TGF may stimulate primitive mesenchymal cells or displaced embryonic cells in the stroma to differentiate into osteocytes. Our results of the immunohistological study showed that BMP-4 was detected in the sebaceous glands (data not shown). Sebaceous glands are abundant in the face, which may explain why the majority of skin tumors with secondary ossification occur on the face, but the reason why the frequency of secondary ossification is low still remains unknown.

**Declaration of patient consent**
The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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**Conflicts of interest**
There are no conflicts of interest.

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Aquagenic Syringeal Acrokeratoderma: Report of a Case with Dermoscopic Findings

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Sir,

Aquagenic syringeal acrokeratoderma (ASA) is a rare disorder that is characterized by symmetrical, edematous, white and translucent papules and plaques. It develops after brief exposure to water. The diagnosis of ASA is generally made based on patient's history, clinical and histopathological examination. Dermatoscopic findings of ASA have been reported in only two cases previously. We report ASA in a teenage girl with dermoscopic features.

A 15-year-old girl presented with a 1-year history of asymptomatic, symmetrical, and white lesions located on her palms. These lesions occurred within minutes of exposure to water or sweat and resolved within 20 min after drying her hands. The patient also reported hyperhidrosis of the palms. On dermatological examination, her palms were macerated, when she soaked her hands in water for several minutes, small white papules coalescing into plaques appeared. She had no history of drug use, contact, or atopic diathesis. On dermoscopic examination, sweat duct pores became larger when compared with a normal-appearing area on the thenar region. Furthermore, tripe-like structures were seen on involved areas. After immersion in water, a punch biopsy was performed on the right palm. Histopathologic examination revealed hyperkeratosis, acanthosis, and dilatation of the eccrine ducts in the epidermis. Based on the clinical, dermoscopic, and histopathological findings, we made the diagnosis of ASA. Her symptoms were regressed by 19% aluminum hydroxychloride cream application for 3 months.

The exact pathogenesis of ASA remains unclear. Several hypotheses have been proposed to explain the pathogenesis - structural or functional defects of stratum corneum during adolescence, primary disease of the sweat ducts, increased sodium concentration in the skin, thereby increasing the water-retention capacity of stratum corneum or a reaction to drugs such as rofecoxib, celecoxib, and aspirin. The reported associations included cystic fibrosis (CF), asthma, allergic rhinitis, urticaria, palmar erythema, and malignant melanoma. In our patient, she had no history of CF, so we did not perform sweat chloride test. Palmar/plantar hyperhidrosis has also been described in a subset of cases with ASA. In our patient, there was no associated disorder except palmar hyperhidrosis.

Treatment modalities include 20% aluminum chloride solution, botulinum toxin injections, iontophoresis, antihistamines, pomade containing 5% salicylic acid, a mixture of mometasone furoate and petroleum jelly, and a cream containing 20% urea. Our patient used 19% aluminum hydroxychloride cream for 3 months. Her lesions regressed after 3 months, but when she stopped the use of the cream her symptoms recurred. Dermatoscopic features of the lesion have been defined in two previous reports as per our knowledge. In these reports, comparison of marked enlargements of the sweat duct puncta and unaffected palmar regions were demonstrated by dermoscope. These findings reflect the...