Idiopathic scrotal calcinosis: A rare scrotal skin disorder

Idiopatik skrotal kalsinozis: Ender görülen skrotal deri hastalığı

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Idiopathic scrotal calcinosis (ISC) is a rare benign disorder of scrotal skin which presents with multiple, asymptomatic nodules on the scrotum appearing in childhood or early adulthood (1). The main controversy is about the etiology of the entity, whether it is idiopathic or occurs due to a proceeding systemic or metabolic disorder (1). Because of its rarity and controversial nature of its development, we present a case of ISC in this report.

Case report

A 44-year-old man was referred to the outpatient clinic of urology with a twenty-year history of painless subcutaneous nodules on the scrotum. Physical examination revealed about twenty-three painless, well-circumscribed subcutaneous nodules in varying diameters on the scrotum (Fig. 1). There was no history of systemic, metabolic, endocrinologic, neoplastic or autoimmune diseases, scrotal trauma or inflammatory disorder of scrotum. Routine laboratory examinations, including serum calcium, phosphorus and parathyroid hormone showed no abnormality. The nodules were extirpated surgically under local anesthesia. The cut surfaces of the nodules were in a yellowish-whitish, chalky appearance macroscopically. His topathologic examination under light microscopy revealed amorphous calcified areas located in the dermis. Extensive fibrotic areas and foreign body reactions were also present within the lesions. No epithelial lining was noted (Fig. 2).

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Discussion

The disease was first described by Hutchinson in 1888 and was named as “Idiopathic scrotal calcinosis” by Shapiro et al. (2). Surgical excision of the lesions is the treatment of choice (1-3). The etiology has been disputed (1-3). No evidence of endocrinologic, metabolic, systemic disorder or any biochemical alteration had been shown to be the reason for ISC (1). There is still a controversy on the pathogenic mechanisms of ISC, whether it is idiopathic or develops upon a preexisting epidermal cyst (2-4). Absence of the epithelial lining within the lesions supports the theory of idiopathic etiology (1-3). Wright et al. presented nine patients with a conclusion that; the condition is truly idiopathic (2). In contrast, Saad et al. reported three patients with ISC and they concluded that, this disorder is not idiopathic and epidermal conclusion cysts play a major role in the pathogenesis of the disease (5).

Recent papers proposed the theory of dystrophic calcification of the epidermal cysts as a cause of ISC due to the appearance of squamous epithelial cells around these calcified areas (6,7). Swineheart et al. described this epidermal cyst theory in detail (8). According to them, calcification of epithelial cysts occurs after an inflammatory reaction, which triggers a degenerative process and eventually leads to loss of epithelial lining of these cysts. Many other authors supported their concept with relevant findings (6-8). The etiology of the dystrophic calcification is also a subject of controversy (6,7). According to the findings of Veress and Feinstein, minor trauma may play a role in the initiation process of this pathology, whereas in other studies dystrophic calcification of Dartos muscle was shown to be the basis of ISC (9,10). In a recent report of Pabuccuoglu et al. it is speculated that, Dartoic muscle degeneration and necrosis seem to be the most important factor in the pathogenesis of this disorder, and dystrophic calcification deposits eventually form in the disease due to this initiating process of muscular degeneration (11). To solve the dilemma about the etiology and terminology of scrotal skin calcifications, Dini et al. put forward an opinion that, the term idiopathic should be used for the cases without an undefined etiology rather than the whole cases (12).

In light microscopic examination of all the specimens we could not observe any epidermoid or pilar cysts and there was no epithelial lining around the lesions. According to these light microscopic findings, the etiology of the disease in our patient could not be ruled out definitely and the case was considered to be idiopathic. We thought that if we have made histological study of the lesions in the early stages of formation, associated with immunohistochemical investigations, we could have found the specific features of these dystrophic calcifications.
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References