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Scrotal calcinosis: is it idiopathic or dystrophic?*

Esra Canan Kelten[1], Metin Akbulut[1], Nagihan Çolakoglu[1], Hatice Bayramoglu[2], S. Ender Duzcan[1]

[1]*Department of Pathology, Pamukkale University School of Medicine, Denizli, Turkey*
[2]*Department of Pathology, Dr. Zekai Tahir Burak Hospital, Ankara, Turkey*

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Background: Scrotal calcinosis is an uncommon lesion characterized by multiple calcific deposits within scrotal skin and often misdiagnosed clinically as epidermal cyst.
Case: In this article, we described a scrotal calcinosis in a 19-year-old man in whom calcified nodules without evidence of epithelialized lining were seen both in dermis and within the bundles of dartos muscle. Additionally, these calcified nodules were surrounded by a few number of mononuclear lymphocytes, mast cells, and hyalinization.
Conclusion: We concluded that, dystrophic calcification of dartos muscle, may be the basic mechanism of scrotal calcinosis in some cases.

Key words: scrotal calcinosis, dystrophic calcification, dartos muscle degeneration, mast cells

Introduction

Scrotal calcinosis was first described by Lewinsky in 1883 as a subtype of calcinosis cutis. Calcinosis cutis is generally used to describe accumulation of calcium salts within dermis. Calcification of the skin occurs in three main forms: dystrophic; metastatic; and idiopathic.[1]

It had been suggested that there were a number of histopathologic mechanisms in the formation of scrotal calcinosis. The main dispute about the cause of this condition is whether the calcium is deposited at the site of preexisting structures; including epidermal cysts, calcification of ecrine sweat ducts or degenerated dartoic muscle or whether the calcified nodules are truly idiopathic. To our knowledge, dystrophic calcification of dartoic muscle is the most unfavorable hypothesis in the literature.[2]

Case

We report a 19-year-old man, who presented with slowly growing, painless nodule on his scrotal skin, that had been present for one year. He had no systemic symptoms and there was no history of trauma, scrotal infection, any systemic disease or family history of a similar complaint. Laboratory investigations, which included serum calcium and phosphorus, and parathyroid hormone levels were within normal limits. The physical examination disclosed a nodule, easily movable beneath the skin surface. No drainage and similar lesions were present elsewhere. The testes were normal and the remaining physical examination revealed no other physical abnormality.

The excised specimen was consisted of a polypoid lesion covered with skin, measuring 1,5x1x0,5 cm. The cut surface revealed multiple firm nodules containing yellowish chalky material. On histologic examination, the epidermis was hyperplastic and the dermis was widely infiltrated by multifocal calcified nodules of varying size surrounded by a few mononuclear inflammatory cells and prominent hyalinization (Fig. 1). Similar small calcified nodules, and surrounding hyalinization were seen within the fibres of dartos muscle. A careful histological examination showed degenerative changes in the dartoic muscle bundles and dystrophic calcification of muscle bundles, adjacent to morphologically comparable vital muscle bundles (Fig. 2). Small calcospherites are associated with a few mononuclear cell infiltration in contrast with prominent fibrosis. The calcified material stained deeply basophilic with the hematoxylin-eosin stain and black with the von Kossa's method for calcium (Fig. 3).

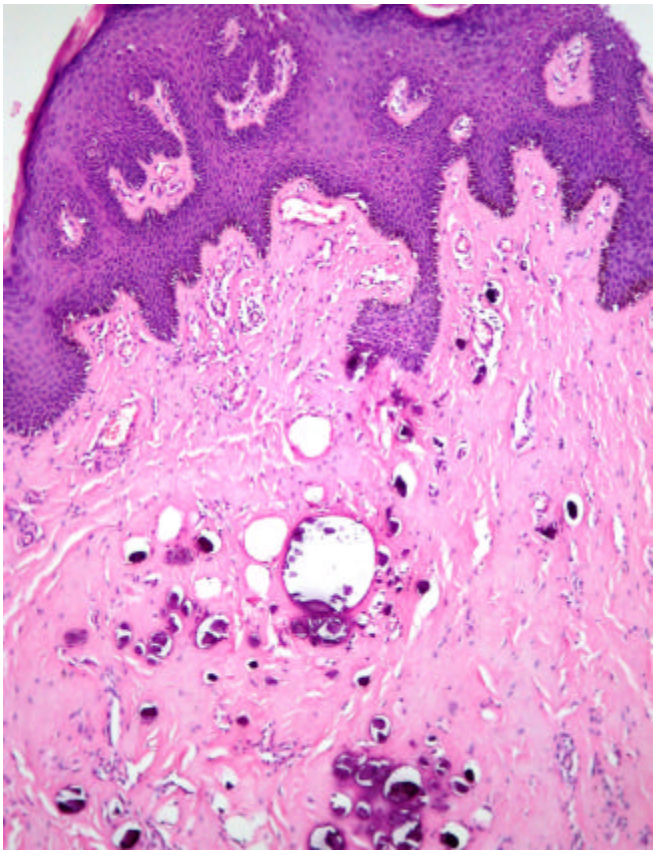

Figure 1. Acanthotic epidermis and several calcified globulles in the superficial dermis (H&E, x4).

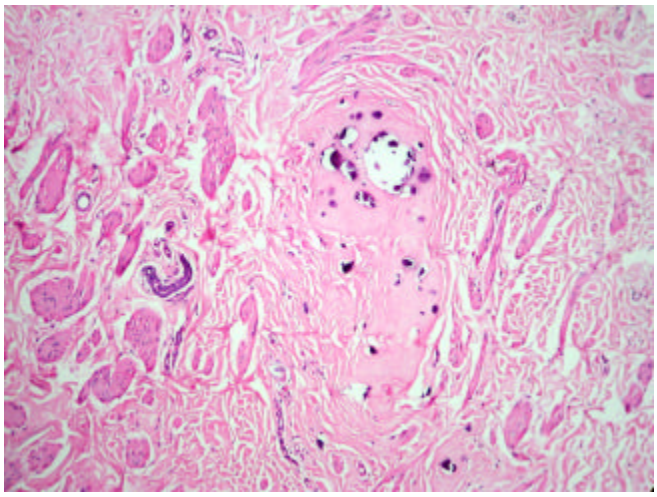

Figure 2. Small calcified globulles surrounding prominent hyalinization within the dartos muscles bundles (H&E, x10).

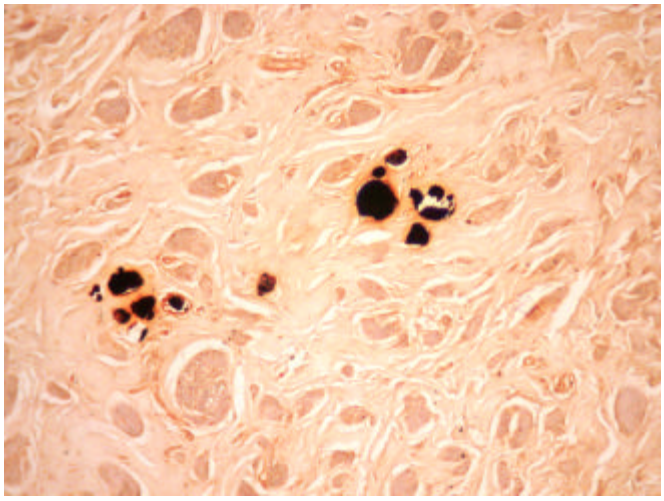

Figure 3. Small calcified globulles within the dartos muscle bundles (Von Kossa, x20).

Despite a careful search no keratinous material, cystic spaces and preexisting duct-like structures or predisposing cause of calcification could be demonstrated inside the calcifications. Immunohistochemistry for CEA (Dako, USA, ready to use), EMA (Neomarker, GP 1, 4, 1/750), and cytokeratin (Dako, USA, AE1/AE3, 1/1000) failed to show any epithelial lining or evidence of keratin in the dermal tissue immediately adjacent to the calcium deposits. Additionally, many mast cells were determined by Giemsa staining closed to the dartos muscle bundles.

The postoperative course was uneventful and the cosmetic result was excellent. On follow-up

examination, seven years after the surgery, our patient was asymptomatic.

Conclusion

Scrotal calcinosis, also described as idiopathic calcinosis, is a rare benign lesion and its pathogenesis still remains unclear and somewhat controversial. These asymptomatic lesions tend to enlarge slowly within years. They may be seen as a solitary or multiple, painless, firm nodules varying from 1–2 mm to 2cm in diameter and usually appear during childhood or early adulthood. Microscopically, multiple calcified globules surrounded by lymphocytic infiltration, histiocytes and hyalinization were observed.[1] In most cases, surgical removal of the nodules is effective and histologic examination will confirm the diagnosis. Although recurrence is unusual, recurrent asymptomatic nodules may occur because of microscopic foci.[3,4]

The etiology of scrotal calcinosis is uncertain. Many authors proposed that scrotal calcinosis represent dystrophic calcification of preexisting structures including; epidermal cysts, eccrine duct milia, eccrine epithelial cysts and degenerated dartoic muscle.[2,3,5–10] In the dystrophic form, calcium and phosphorus levels are normal, and there is a local favoring condition that predisposes the calcinosis. It may be observed in connective tissue diseases like scleroderma, dermatomyositis, SLE and secondary to trauma and inflammation.[1] Calcifications that are not associated with tissue damage or metabolic disorders are called idiopathic scrotal calcinosis.[1] In some cases of dystrophic calcinosis cutis, the underlying connective tissue disease may be mild and can be overlooked if not searched carefully. Therefore, such cases may be misdiagnosed as idiopathic calcinosis cutis. In our patient, there was no history of any connective tissue disease and trauma, and his serum values of calcium and phosphorus were normal. And no existing inflammation or epithelial lining was found around the calcified nodules microscopically in the present case. Also Pabuççuoglu et al. indicated that they did not observe any residual cyst and epitelial lining around the calcified globules and cystic or duct-like spaces in their cases.[9]

Armijo, Fischer, and King detected calcified globules in dartos muscle fibres and close to the muscle. They supposed that scrotal calcinosis was followed by dystrophic calcification of dartos muscle.[8,9,10] Also, recently, Pabuççuoglu et al. concluded that degeneration and necrosis of the dartoic muscle were the initial events in the pathogenesis of the disease.[9] Calcification of smooth muscle has also been observed in uterine leiomyomas.[11] Minor trauma to the scrotal skin thought to be an important role as the starting point of dystrophic calcification.[12] Fuzesi supposed that degenerative changes caused alterations in chemical microenvironment leading to deposition of calcium and phosphorus.[13]

Fukaya and Ueda reported a case with vulvar calcinosis and noticed a number of mast cells around calcified globules in dermis by toluidine-blue staining. They had also shown similar metachromatic staining like mast cells in the calcified globules and they thought mast cells might play a role in pathogenesis of the calcinosis based on their findings.[14] Similarly, we observed calcium globules in the dartos muscle fibres and there were a lot of mast cells between the dartos muscle fibres. Secretion products, especially metachromatic granules of the mast cells were reported to be capable of chelating calcium.[15]

Most cases with scrotal calcinosis have been asymptomatic and painless, therefore, biopsy of these lesions is a late event. Although dystrophic calcifications of cysts, foreign bodies, or degenerated dartos muscle are found in some cases in earlier stages of scrotal calcinosis but their absence at advanced stage of the disease and presence of only dermal calcium deposits may be due to prominent inflammation, fibrosis and hyalinization. In small number of cases an idiopathic origin of the disease could be still proposed, if there was no history of local or systemic favoring factors, and without evidence of epithelial cyst lining or an adnexal tumor even with immunohistochemical studies.[16]

In the present case, our findings strongly support the hypothesis that degenerated dartos muscle fibres may constitute the basic abnormality. Inflammatory reaction and calcium chelasion triggered by mast cells, may be the basic mechanism of scrotal calcinosis in some cases.

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