

MULTIPLE SCROTAL SWELLINGS; A HISTOLOGICAL SURPRISE

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Abstract: Scrotal calcinosis (SC) is a rare and benign disease of the scrotum wall. It is defined as the existence of multiple calcified and asymptomatic nodules of the scrotal skin. The main controversial point, concerns the pathogenesis of the scrotal calcinosis. Indeed, if some authors think that SC is the result of dystrophic calcifications of dartoic muscles. It is also unclear whether scrotal calcinosis originates from inflammation of epidermal cysts, others did not find any evidence of pre-existing cystic structures and believe this condition to be idiopathic. Histopathologically, SC is characterized by the presence of calcium deposits within the dermis surrounded by a foreign body—type granulomatous reaction. Despite the controversy about the origin of this entity, surgery is the treatment of choice and provides excellent results

Introduction

Scrotal calcinosis (SC) is a rare benign entity characterized by calcium deposits within the dermis of the scrotal skin. The nodules may range from one to a hundred in number and from 1 mm to several centimetres in size they may occupy the whole scrotum also. These nodules are confined to the scrotum and are mostly asymptomatic. SC may not cause any abnormalities in parathyroid hormone, calcitonin, 25-OH vitamin D and phosphorus/calcium levels. However, few cases of scrotal calcinosis have been reported in the literature and there is still a controversy about the pathogenesis of this rare condition. It is not known whether it is idiopathic or not [1]

CASE REPORT.

We report a 49 year old Indian male presenting with multiple scrotal swellings since childhood and the swellings have increased for the past five years. He had discomfort due to the swellings. He consulted few doctors earlier for excision but was deferred. VDRL and HIV tests were negative with normal blood parameters. Patient had no associated medical co-morbidities. Laboratory examinations, including serum calcium, phosphorus, and parathyroid hormone levels, were normal

On examination, middle aged patient was moderately built and moderately nourished. The scrotal examination revealed multiple, subcutaneous, dark brown, firm, non-tender, scrotal swellings, swellings measuring about 0.5 cm to 1 cm each. With a Diagnosis of multiple scrotal epidermoid cyst (Strawberry Scrotum) he was subjected for subtotal scrotectomy and primary closure. He withstood the procedure well and was discharged on post operative day 4. His final histopathology report revealed calcinosis cutis of scrotal wall. No recurrence was observed for 6 months follow up and a good aesthetic result was obtained.

RESULTS AND DISCUSSION

Scrotal calcinosis, first described by Lewinski, usually appears in men aged 20 to 40 years. The youngest and oldest patients reported in the literature were 9 and 85 years old, respectively [2]. Scrotal calcinosis consists of nodules within the dermis of the scrotal skin varying in size and number and develops slowly over many years. Although they are mostly asymptomatic, with a feeling of heaviness in the scrotum, discharge and itching are the most frequently encountered complaints [3]. For diagnosis of scrotal calcinosis, Ito et al. performed an immunohistochemical study using anti bodies against CEA, epithelial membrane antigen (EMA), and gross cystic disease fluid protein-15 (GCDFP-15) to describe dystrophic scrotal calcinosis originating from eccrine cysts. They found a positive reaction for CEA and EMA in the luminal cells and in the contents of a large cyst and ductal structures, and positive GCDFP-15 staining in the latter [4]. Dini and Colafranceschi used antibodies against low molecular weight cytokeratin CAM 5.2, a cocktail of cytokeratin AE1/AE3, CEA, collagen type IV and laminin of the basement membrane and only observed a slight positivity for cytokeratin AE1/AE3 within the amorphous calcified mass, which was probably due to dystrophic calcification

of epidermoid cysts [5]. A firm diagnosis can only be made with histological examination as in this case. In the literature, scrotal calcinosis has rarely been reported and there is an ongoing debate about the pathogenesis of this rare condition. In fact, it is still arguable whether it is idiopathic or not [1]. In their series of 14 cases, Shapiro et al. reported that calcified nodules without epithelial linings were idiopathic [6]. However, King et al. claimed that the lesions showed dystrophic calcifications of the dartoic muscle [7]. Dini and Colafranceschi noted that inflammation and rupture of epidermoid cysts constituted the main pathological mechanism in SC [5]. Two studies suggested that the epithelial lining may be obscured in the course of time by inflammation of epidermal cysts followed by calcification, rupture of the cyst wall and granulomatous proliferation [8,9]. Swinehart and Golitz thought that scrotal calcinosis resulted from inflammation and calcification. Furthermore, Song et al. examined 51 nodules excised from a patient with SC [9]. They demonstrated that epidermal cysts were affected by mild to moderate inflammation and that mononuclear cell or foreign body granuloma formation was followed by resorption of cyst walls and keratinous material until the calcified deposits remained. One of the most important observations was the resorption of the cyst wall and this was a rapid stage of the sequence. As a result, histopathological findings change depending on the age of cysts and this causes long term cysts to have fewer or no epithelial lining cells. The last theory, proposed by Ito et al. is that that SCs originate from eccrine epithelial cysts. Matrix debris is deposited following discharge. Antibodies against sulphated mucopolysaccharides and immunohistochemical studies using CEA and EMA give positive reactions [4]. In our case which is discussed although there was no epithelial lining, the dermis showed granules and globules of amorphous basophilic extracellular calcium deposits. Testicular tumours such as teratomas, gonadoblastomas, and Leydig cell tumours may show calcification or ossification [2]. Recurrent asymptomatic lesions proving to be calcinosis by biopsy may be observed safely because of carrying no risk of malignancy. Therefore, recurrent asymptomatic lesions may be followed up due to their clinical circumstance [2]. In fact, SC is of interest to multiple disciplines such as urology, plastic and general surgery and pathology. Because most patients with scrotal calcinosis are asymptomatic, they usually seek medical advice for cosmetic reasons. Therefore, biopsy of these lesions at an advanced stage can be late and only shows dermal calcium deposits [2]. In such cases, the patients should be assured that they do not necessarily have a malignant condition, but that only a histological examination of the material removed through surgery can confirm it. They should also be reminded that the disease may recur, though rarely, and that the most appropriate treatment is surgery. During ultrasonographic (US) examination, calcification in or adjacent to epididymis may be found and this is usually due to chronic epididymitis. Granulomatous disease should always be considered in these circumstances. Haematoma and sperm granulomas (sperm extravasation with granuloma formation) may produce a solitary echogenic area within the epididymis. The appendix epididymis and appendix testis may calcify and these are recognized by their characteristic position and shape. These lesions are related to previous inflammatory diseases of the epididymis [10]. Subtotal excision of the scrotal wall is recommended for the treatment of massive calcinosis [11]. In the case presented here, there was a massive occurrence involving the whole of the scrotum and local resection was not possible, a subtotal scrotectomy was done with a primary closure of the defect with mattress sutures. At the end of a 6month follow-up, we achieved satisfactory cosmetic results and there was no relapse.

CONCLUSION

Although scrotal calcinosis is a rare diagnosis by itself, the pathogenesis and the origin is quiet controversial but the gold standard of treatment seems to be surgical approach and excision depends on the extent of the involvement of the scrotum.

REFERENCES

- 1. Saladi RN, Persaud AN, Phelps RG, Cohen SR: Scrotal calcinosis: is the cause still unknown? J Am Acad Dermatol 2004, 51(2 Suppl):S97-S101
- 2. Saad AG, Zaatari GS: Scrotal calcinosis: is it idiopathic? Urology 2001, 57:365.
- 3. Shah V, Shet T: Scrotal calcinosis results from calcification of cysts derived from hair follicles: a series of 20 cases evaluating the spectrum of changes resulting in scrotal calcinosis. Am J Dermatopathology 2007, 29:172-175
- 4. Ito A, Sakamoto F, Ito M: Dystrophic scrotal calcinosis originating from benign eccrine epithelial cysts. Br J Dermatol 2001, 144:146-150.
- 5. Dini M, Colafranceschi M: Should scrotal calcinosis still be termed idiopathic? Am J Dermatopathol 1998, 20:399-402.
- 6. Shapiro L, Platt N, Torres-Rodríguez VM: Idiopathic calcinosis of the scrotum. Arch Dermatol 1970, 102:199-204.
- 7. King DT, Brosman S, Hirose FM, Gillespie LM: Idiopathic calcinosis of scrotum. Urology 1979, 14:92-94.
- 8. Swinehart JM, Golitz LE: Dystrophic calcification of epidermoid cysts. Arch Dermatol 1982, 118:985-988.
- 9. Song DH, Lee KH, Kang WH: Idiopathic calcinosis of the scrotum: histopathologic observations of fifty-one nodules. J Am Acad Dermatol 1988, 19:1095-1101.
- 10. Bushby LH, Miller FN, Rosairo S, Clarke JL, Sidhu PS: Scrotal calcification: ultrasound appearances, distribution and aetiology. Br J Radiol 2002, 75:283-288.
- 11. Ruiz-Genao DP, Ríos-Buceta L, Herrero L, Fraga J, Aragüés M, GarcíaDíez A: Massive scrotal calcinosis. Dermatol Surg 2002, 28:745-747

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