

Smooth muscle hyperplasia of the epididymis

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ABSTRACT

Benign smooth muscle cell proliferation commonly involves a variety of body organs, yet is a rare finding in the spermatic cord or paratesticular tissue of the male genital tract. Here we discuss a case of smooth muscle hyperplasia of the epididymis which presented as an intrascrotal mass. This is a very rare condition and should be considered in the differential diagnosis of any paratesticular mass.

INTRODUCTION

Benign smooth muscle cell lesions are frequently found in a diversity of body organs, such as the breast, gastrointestinal tract, kidney, bladder, and skin (1), but they are extremely rare in the spermatic cord or paratesticular tissue of the male genital tract. They are described as hypertrophy, hyperplasia or hamartomatous proliferations in the few published reports (2). They are however a largely unrecognised non-neoplastic cause of intrascrotal masses. In this article, we report a rare case of an intrascrotal epididymal mass, diagnosed as smooth muscle cell hyperplasia.

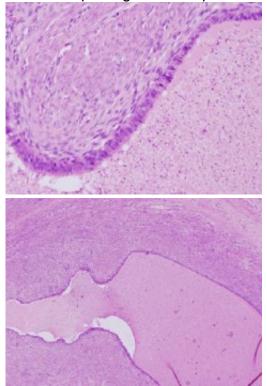
CASE REPORT

A 55-year-old Caucasian male presented with a two-year history of a firm, tender lump in his scrotum, which was also increasing in size. The patient was otherwise fit with no significant past medical history including no vasectomy history, exposure to TB or recent foreign travel. Physical examination revealed a 3cm irregular hard mass in the upper pole of his left epididymis, which was completely separate from a normal feeling testis and from the scrotal wall. The rest of his genitalia were normal as was his abdominal examination. The serum level of Alpha-Fetoprotein and ß-Human Chorionic Gonadotropin were within normal limits. On ultrasonography both testes appeared normal and a small varicocoele was noted in the right hemi scrotum. There was an irregular, hypoechoic cystic mass in the upper epididymis with internal echoes measuring up to 2cm in diameter and slightly prominent left testicular veins. A staging CT of the chest, abdomen and pelvis excluded the possibility of metastases or another primary. After discussion at the Multidisiplinary Team meeting an inguinal orchidectomy was advised. Sectioning the orchidectomy specimen revealed a well circumscribed, predominantly multicystic, mass measuring 17 mm in the tail of epididymis (Figure 1). Further sectioning showed the lesion extending towards the epididymal head and measured maximally 34mm. Both proximally and distally the lesion was less well demarcated from the normal epididymal structures. The background testis and spermatic cord were unremarkable.



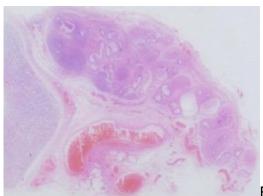
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Microscopic examination of the lesion showed cystic spaces and ducts lined predominantly by flattened and, to a lesser extent, by columnar epithelium, identical to that seen within the normal epididymis. Some of theses spaces contained spermatozoa (*Figure 2*). Parts of the epithelium lining the ducts were thrown up into low papillary folds. A prominent layer smooth muscle was found lining these cystic spaces and was also identified within the interstitium between them (*Figure 3*). The lesion merged with tubules in the epydidymal tail and head (*Figure 4*). The rete testis showed extensive cystic dilatation as did the tubules in the epididymal head. There was an occasion sperm granuloma present. No abnormality was seen within the testis or cord.





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Based on the histopathological findings a diagnosis of smooth muscle hyperplasia of the epididymis was made.

DISCUSSION

Hyperplastic and hamartomatous extratesticular intrascrotal lesions are exceptionally rare and may simulate neoplasia on clinical examination (2). There are only a handful of reports in the literature, (2-6). The largest series was published by Barton et al (1999), (7). He described 16 testicular adnexal lesions in males aged between 46 and 81, mean 63 years. Ultrasonography was the imaging modality of choice, followed by orchidectomy in all cases except a few where only epididymis was surgically excised. Nine of the 16 cases were in the epididymis; of the remaining three were in the spermatic cord and one each in the tunica vaginalis and tunica albuginea. Grossly, the masses ranged in size from 6mm to 70mm (mean 26mm). Microscopically all cases showed smooth muscle hyperplasia in a periductal, perivascular, interstitial or mixed pattern. Follow up was available for 13 patients and this was uneventful. The periductal pattern as described by Barton is similar to the pattern of proliferation in our case. When considering the normal anatomy of the epididymis, the tubules in the tail have a prominent smooth muscle coat compared with those in the head. In addition the presence of spermatozoa in the cystic spaces and the overall preservation of the epididymal architecture further support the theory that this is a hyperplastic process. What we have is an overgrowth of muscle normally present in this area. As Barton points out it is debatable if these lesions are better classified as hamartomas (8), but we agree hyperplasia better describes the proliferation. The cause of this proliferation is unknown. In other body systems, such as the gallbladder, the adenomyomatous change is related to chronic inflammation. The possibility of obstruction of the epididymal or vas deferens ducts has been considered (7). However in our case, as in the cases described by Barton there is no clear relationship to previous injury, surgery or an inflammatory disorder. The proximal ectatic changes in the epididymal head and rete testis are presumed a secondary phenomenon related to pressure affect in the area of smooth muscle hyperplasia. In summary we have presented a case of smooth muscle hyperplasia of the epididymis, a very rare and benign lesion, the cause of which is unknown. It should be included in the differential diagnosis of epididymal masses. In addition, more careful examination of the epididymis in all orchidectomy specimens may reveal coincidental subclinical smooth muscle hyperplasia, which would otherwise have gone undetected. This may improve of understanding of these benign lesions.

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