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PSEUDOTUMORS AND REACTIVE PROLIFERATIONS OF TESTIS AND PARATESTICULAR STRUCTURES

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ABSTRACT
Non-neoplastic masses or pseudotumors (tumor-like proliferations) and non-neoplastic cellular proliferations are rare in the testis and paratesticular tissues. These lesions (infarction; testicular abscess; ectopic tissues and hyperplastic lesions) may present as a true neoplasm either macroscopically or microscopically. Their incidence varies according to different series from 6 to 30%. Hence an attempt is being made to distinguish benign mimickers and pseudotumors from true neoplasia on morphologic criteria, so as to prevent unnecessary orchiectomy. We summerize main tumor like lesion and benign mimickers with emphasis on morphologic criteria for the differential diagnosis from true neoplasia.

KEYWORDS: Testis, Paratesticular structures, Pseudotumors, Benign mimickers.

INTRODUCTION
The pseudoneoplastic or nonneoplastic lesions are the lesions that can simulate a neoplasm in the testis or paratesticular tissues.[1] These lesions can be divided broadly into two groups - that only macroscopically imitate neoplasia (vascular lesions; inflammatory lesions – nonspecific and specific infectious lesions and non-infectious inflammatory lesions; cysts – testicular, epididymal, spermatic cord cysts; ectopic tissues – spleno-gonadal, adrenal cortical rests; Testicular appendages – adenomatoid tumor; miscellaneous other lesions) and those that microscopically imitate neoplasia, regardless of whether or not they form a macroscopic mass (testicular i.e. inflammatory-reactive lesions, sertoli cell hyperplasia, intersticial cell hyperplasia, hyperplasia of rete testis; epididymis – adenomatoid hyperplasia; tunica albuginea-vaginalis – mesothelial hyperplasia; spermatic cord – vasitis nodosa, inflammatory pseudotumor (funiculitis proliferans) and miscellaneous other lesion.[1] It is more problematic for the pathologist to classify the latter group correctly.

The lesions that macroscopically imitate a neoplasia affect both testicular and paratesticular tissues and are therefore difficult to establish if one or both are affected. Therefore, the tumor like lesions were classified according to their etiology. Patient age and location (intratesticular vs paratesticular) are two of the most important determining features.[1]

MATERIAL AND METHOD
The present study was conducted in the department of pathology, SGT Medical college, Budhera, Gurgaon for a period of 1 year. Fifteen testicular and paratesticular lesions were received including inflammatory lesions – abscess (20%); 4 cases were non-inflammatory lesions - infarction, adenomatoid hyperplasia (26.6%) and 8 cases were miscellaneous lesions - spleno-gonadal fusion, hydrocele (53.3%). The gross features of all the submitted specimen were noted. Sections were taken and processed for histopathological observetion for light microscopic studies, using haematoxylin and eosin (H&E) stain. Immunohistochemistry on appropriate sections were done wherever required.

RESULT
Out of 15 cases, 3 cases (20%) were inflammatory in nature, 4 cases (26.6%) were non-inflammatory lesions and 8 cases (53.3%) were miscellaneous lesions.

Table 1: Distribution pattern of cases

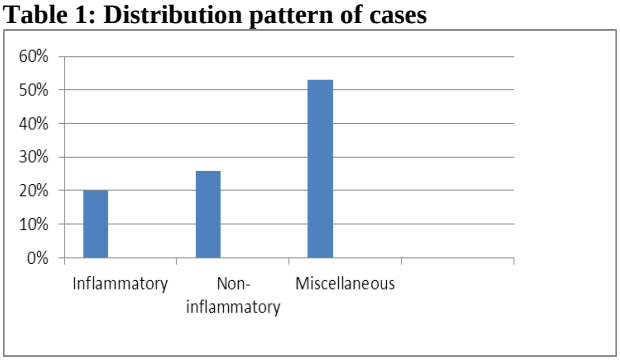
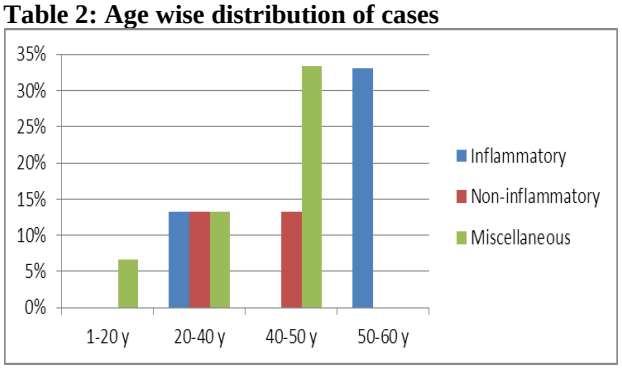


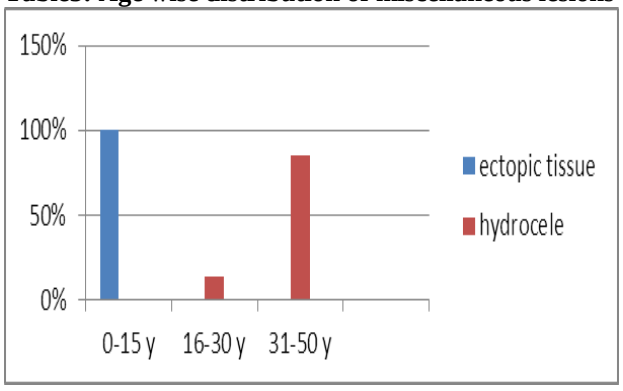
Table 2: Age wise distribution of cases



2 cases (13.3%) of inflammatory lesions were between 20-40 years. 1 case (6.6%) of inflammatory lesion was between 50-60 years; 4 cases (26.6%) of non-inflammatory lesion were between 20-50 years; 1 case (6.6%) of miscellaneous lesion was between age group 0-20 year; 2 cases (13.3%) were between 20-40 years and 5 cases (33.3%) were between 40-50 years.

Out of 3 cases of inflammatory lesions 1 case was of acute inflammation, 1 case of acute on chronic inflammation with granulation tissue and 1 case of tubercular abscess.

Table3: Age wise distribution of miscellaneous lesions



Miscellaneous lesions categorises 1 case of spleno-gonadal fusion between 0-15 years and 7 cases of hydrocele between 16-50 years.

There were 3 cases of testicular abscess between age group 25-60 years. On examination, scrotum was swollen, indurated and tender. On haematological examination, there were polymorphonuclear leucocytosis (90% were neutrophils). Urine microscopy showed a field full of pus cells. Ultrasound of scrotum showed thickening with decreased vascularity and evolving abscess. The scrotal exploration was performed under spinal anesthesia, testicular tissue was removed. On microscopic examination tissue was necrotic and acute inflammatory infilterate was present; ulcerated stratified squamous epithelium with acute on chronic inflammation and subepithelial granulation tissue was observed. Tubercular abscess showed ill defined granuloma around foci of necrosis surrounded by marked acute on chronic inflammatory infilterate.

There were 7 cases of hydrocele between age group 25-50 years, clinically presented with gradually increasing scrotal mass and scrotal tissue wall was firm and fibrous. Ultrasound examination was not specific. On exploration, thick fibrous wall of hydrocele sac was removed. On microscopic examination, fibrocollagenous of hydrocele sac showed chronic non-specific inflammatory infiltrate of lymphocytes. In one case focal areas of calcification was seen.

The 3 cases of testicular infarction were seen varying between age groups 25-50 years of age, presenting with acute onset of testicular pain. Ultrasound examination showed avascular parenchymal lobules, the remaining area of testis exhibited normal echogenicity and vascularity. Since the underlying cause of testicular pain was unclear, surgical exploration was performed. The microscopic examination of the specimen revealed areas of coagulative necrosis and infarction surrounded by an inflammatory reaction including foreign body type granulomatous reaction. Fibrosis and lipofuscin deposits were identified.

A 45 year old patient, with Adenomatoid tumor presented with gradually increasing swelling in right scrotum since 2 months. Ultrasonography revealed normal scan of testis. Clinical impression was of an epididymal cyst. Surgical excision was performed and histopathological examination along with immunohistochemistry confirmed the diagnosis of adenomatoid tumor of the epididymis. Microscopic examination showed unencapsulated tumor, focally entrapping epididymal tubules. The tumor was composed polygonal, cuboidal, flattened or vacuolated epithelioid cells arranged in tubular/glandular structures or solid cords and a hypocellular fibrous stroma interspersed with occasional lymphocytes. The tumor cells were epithelioid showing mild cytologic atypia with ample acidophilic finely granular or vacuolated cytoplasm. The low nucleocytoplasmic ratio and mild anisonucleosis was present. The large round to ovoid nuclei were located either centrally or eccentrically, with evenly distributed chromatin, smooth nuclear membranes and single central nucleoli. No mitosis was seen. At the periphery lymphoid aggregates were seen. Immunohistochemistry revealed positive staining for calretinin and negative staining for epithelial membrane antigen (EMA). On the basis of histomorphological and immunohistochemical study, final diagnosis of adenomatoid tumor of epididymis was given.

Speno-gonadal fusion was found in an 8 year old boy. Specimen submitted in department grossly showed long tubular grey white soft tissue 8X2X1 cm, cut section showed areas of haemorrhage. Microscopically splenic tissue with areas of haemorrhage and congested blood vessels in the capsule was seen. Morphologically, the ectopic splenic tissue was in close relation to the head of the epididymis.

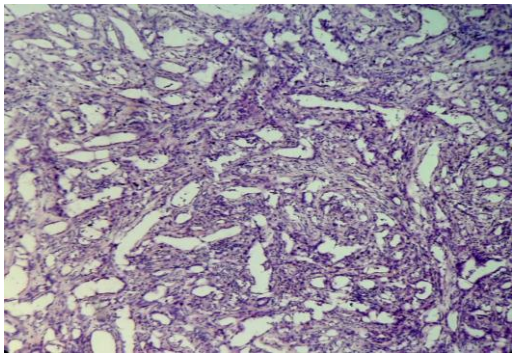


Fig 1: Adenomatoid tumor: epithelial-like cells and fibrous stroma (H&E stain 100X)

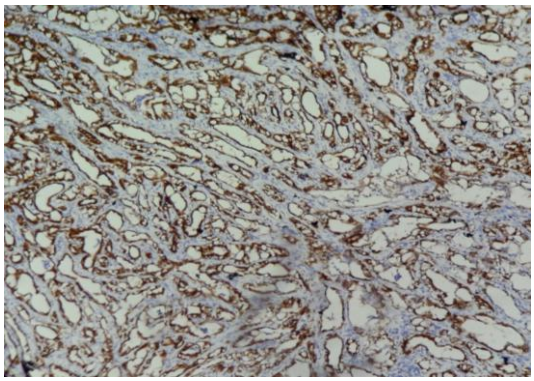


Fig 2: Adenomatoid tumor with positive immunoreactivity to calretinin.

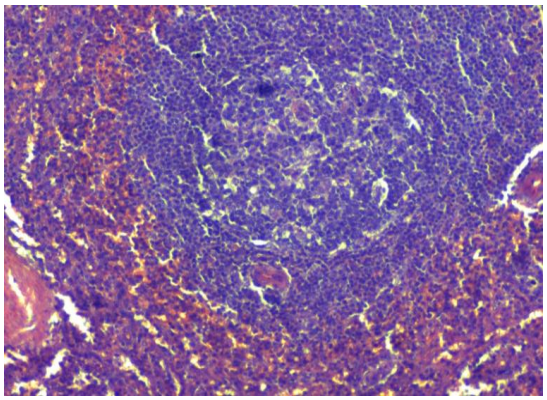


Fig 3: Spleno-gonadal fusion(H&E 100X)

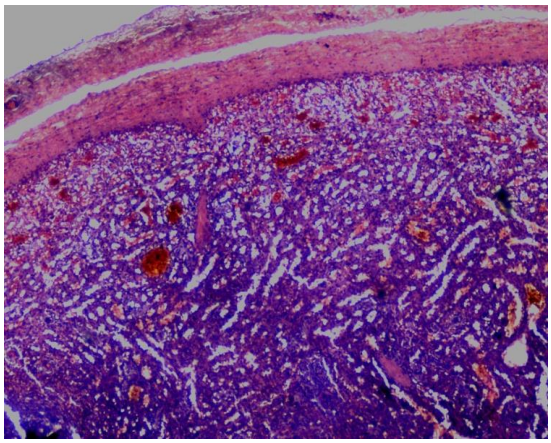


Fig 4: Spleno-gonadal fusion (H&E 40X)

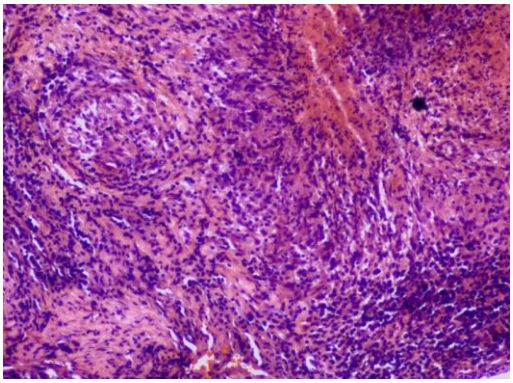


Fig 5: Tubercular abscess showing granuloma (H&E 100X)

DISCUSSION

In the male gonad majority of vascular disorders are not confused with a tumor because, in addition to causing acute symptoms, they usually affect the entire gonad. However, in the rare situation that is when vascular lesion is segmental, it can simulate neoplasia.[1] Testicular infarction is uncommon. It frequently presents with acute scrotal pain and may be confused clinically and radiologically with a testicular tumor or torsion.[2] The exact etiology of segmental infarction of testis is not clear. Several authors have implicated various local and systemic pathology as possible etiological factors for the development of segmental infarction within the testis. Acute epididymitis, spermatic cord torsion, inguinal trauma such as varicocelectomy and herniorrhaphy, cholesterol embolisation, protein S deficiency and sickle cell trait are all implicated in the pathogenesis of segmental testicular infarction. Recently diabetic microangiopahty and human chorionic gonadotropin are identified as possible etiological factors in the development of focal testicular infarction. The other etiological factors attributed to this pathology are vasculitis, fibroplasias of spermatic artery tunica media, polyarteritis nodosa, polycythemia, thromboangitis obliterans and strenuous exercise.[3]

Inflammation that simulates a neoplasia has atypical clinical features. Nonspecific infectious inflammatory lesions are lesions with a tumorlike presentation and are frequently chronic processes causing fibrosis, which may clinically or sonographicaly stimulate neoplasia. Rarely a testicular, epididymal, or vas deferens abscess can look like a neoplasia. Very occasionally, testicular neoplasia can clinically imitate an acute inflammatory process. Specific infectious inflammatory lesions that are most often have been confused with neoplasias are granulomatous inflammation in tuberculosis, brucellosis, syphillis, fungal infections and parasite diseases. These lesions does not cause problem for the pathologist.[3] Genitourinary tuberculosis is the second most common site of involvement among extrapulmonary TB. Isolated genital involvement is seen in about 28% of the patients. The most common site is epididymis; isolated testicular tuberculosis is rare.[4]

Testicular abscess is an uncommon complication affecting 4-5% of clinically diagnosed males with severe epididymoorchitis. The abscesses complicating acute epidiymo orchitis can be divided into those sexually transmitted (Chlamydia trachomatis, Neisseria gonnorhea) or non sexually transmitted (E.coli, Enterococcus, Staphylococcus, Streptococcus). Testicular abscess can also result from bacterial infection of a pre existing haematoma due to previous trauma or testicular infarction secondary to torsion or mumps. Ultrasonography of the scrotum is the modality of choice for diagnosing the conditition. However in untreated cases, secondary scrotal testicular atrophy can develop.[5]

Hydrocele is the term given to the collection of serous fluid within the tunica vaginalis sac. Acquired and congenital forms exist, the former being associated with inflammatory processes of the scrotal contents. The lining of the sac is made up of mesothelial cells. This condition should be distinguished from spermatocele of epididymal origin, which is identified by the presence of sperm in the cyst fluid. Sections from hydrocele and spermatocele specimens may contain sloughed 'small blue cells' probably artifactually and squeezed from the rete testis which may mimic small cell carcinoma.[6]

Splenic–gonadal fusion is the fusion of spleen and gonad. It is more frequent on the left side, with about 148 published cases.[9] Morphologically, the ectopic spenic tissue may be in close relation to the head of epididymis or the upper pole of the testis or separate from it. There may or may not be structural continuity between the normal spleen and the ectopic tissue.[7] In about 30% of cases it is associated with complex malformations such as micrognathia, peromelia, or phocomelia (absence of upper portion of the limb).[8,10,11] Hepato-gonadal fusion is reported.[12]

Adenomatoid tumor are benign masses usually located within the genital tract organs of males (epididymis, testicular tunica, spermatic cord, testicular parenchyma) and females (uterus, fallopian tubes and ovarian area).[13,14,15] Extragenital sites are heart, lymph nodes, adrenal glands, intestinal mesentery, omentum and retroperitoneum.[16] It is a nonhormone dependent tumor of mesothelial origin that is usually localized in the epididymis.[19] Paratesticular tumors account for less than 5% of all intrascrotal masses and adenomatoid tumors comprises about 30%.[17] Beccia et al.[18] reported 256 benign epididymal tumors out of a total of 341 non-testicular tumors. Among these epididymal tumors, adenomatoid tumor (73%) was most frequent, others were leiomyoma (11%) and papillary cystadenoma (9%). The remaining benign entities (7%) included angioma, lipoma, dermoid cysts, fibroma, hamartoma, teratoma and cholesteatoma. Pediatric cases are considered very rare with only few reported cases.[21,22] Immunohistochemically, an adenomatoid tumor is positive for markers, such as CK (AE1/AE3) EMA, Cam5.2, CK 5/6, CK7, calretinin, vimentin, WT1 and HBME-1. Other tumor markers, such as AFP, LDH, CEA, and b-HCG, when measured, are negative, being helpful for the exclusion of malignancy.[20,23,24,25,26]

CONCLUSION

The testicular and paratesticular described constitute a large heterogeneous group, without etiological or pathogenic relations among them or with true neoplasias. The differential diagnosis of these lesions includes malignant testicular and paratesticular tumors and inflammatory conditions. A scrotal ultrasound should be done for patients when the initial diagnosis of painless scrotal tumor has become questionable. A through clinical and radiological assessment is of paramount importance and timely intervention can provide the maximum testicular preservation for these patients.

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