Non germ cell tumors of testis- a 5 year data from a tertiary cancer center in South India with review of literature

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ABSTRACT:

Introduction: Testicular tumors of non-germ cell origin are very rare constituting 5 to 10 percent of all testicular neoplasms. This includes primary as well as secondary tumors. These tumors because of their diverse histomorphology pose diagnostic difficulty to pathologists. Hence, our study covers the spectrum of these lesions with emphasis on unusual morphological features of these lesions, their morphological similarities and diagnostic clues that may help us in diagnosis with the help of immunohistochemical markers.

Methods: All testicular tumors that were diagnosed and treated at our institute from 2017 to 2021 were collected retrospectively from the department registry. All germ cell tumors and hematolymphoid tumors which form the major bulk of testicular tumors were excluded from the study.

Results: Accordingly, twelve cases were triaged among which ten cases were primary testicular tumors and two were metastatic. Of the primary tumors, four cases were pure Leydig cell tumors, two cases of sertoli cell tumor, NOS, one case of neuroendocrine tumor, two cases of embryonal rhabdomyosarcoma and one case of undifferentiated pleomorphic sarcoma. Secondary tumors include two cases of prostatic adenocarcinoma metastasis.

Conclusion: Non germ cell testicular tumors pose diagnostic difficulties because of their diverse histomorphological presentation and lack of awareness. Extensive sampling with a panel of immunohistochemical markers might be necessary to arrive at a definitive diagnosis. Our study gives an overall view of these rare lesions.

Keywords: Testis, non germ cell tumors, metastasis

INTRODUCTION:

Testicular tumors of non-germ cell origin are very rare constituting 5 to 10 percent of all testicular neoplasms. This includes primary as well as secondary tumors. The primary tumors include sex cord stromal tumors, mixed sex cord stromal and germ cell tumors, mesenchymal as well as hematolymphoid tumors. The secondary tumors include metastatic tumors to testis or that shows infiltration into testis from nearby structures. It is important to diagnose these rarer entities as the biological behaviour and treatment of each of these entities are different. This study covers the presentation of these lesions with emphasis on difficulties that we face in diagnosing them with review of previous literatures.

MATERIAL AND METHODS:

All testicular tumors that were diagnosed and treated at our institute from 2017 to 2021 were collected retrospectively from the department registry. All germ cell tumors and hematolymphoid tumors which form the major bulk of testicular tumors were excluded from the study. Rest of the cases irrespective of the age group were included. Patient’s clinical information was collected from the medical record system. All the clinical and pathological data was compiled and analyzed. All H and E slides and IHC slides were retrieved from filing and studied.

RESULTS:

Accordingly, twelve cases were triaged among which ten cases were primary testicular tumors and two were metastatic. Of the primary tumors, four cases were pure Leydig cell tumors of which three were benign and one was malignant. Other tumors include two cases of sertoli cell tumor, NOS and one case of neuroendocrine tumor. Mesenchymal neoplasms include two cases of embryonal rhabdomyosarcoma and one case of undifferentiated pleomorphic sarcoma. Secondary tumors include two cases of prostatic adenocarcinoma metastasis. The detailed picture of each case is described as follows:

CASES 1-4:

4 cases of Leydig cell tumor was studied. All the cases have classical morphology. All 4 cases were tabulated with their clinical profile (Table 1).
<table>
<thead>
<tr>
<th>CASE NO.</th>
<th>AGE/SEX</th>
<th>CLINICAL PRESENTATION</th>
<th>GROSS</th>
<th>IHC (positive markers)</th>
<th>FINAL DIAGNOSIS</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>51/M</td>
<td>Asymptomatic Left testicular mass</td>
<td>8x5.5x3cm</td>
<td>CD 99, Calretinin</td>
<td>Benign leydig cell tumor</td>
</tr>
<tr>
<td>2</td>
<td>33/M</td>
<td>Asymptomatic Left testicular mass</td>
<td>6x6x5 cm</td>
<td>Calretinin, inhibin ,Melan A</td>
<td>Benign leydig cell tumor</td>
</tr>
<tr>
<td>3</td>
<td>34/M</td>
<td>Azoospermia Right testicular mass</td>
<td>1.5x1x1cm</td>
<td>Calretinin, inhibin ,Melan A</td>
<td>Benign leydig cell tumor</td>
</tr>
<tr>
<td>4</td>
<td>11 months/M</td>
<td>Precocious puberty, Right testicular mass</td>
<td>3x2.5x2 cm</td>
<td>Inhibin, melan A, CD 56</td>
<td>Malignant leydig cell tumor</td>
</tr>
</tbody>
</table>

All were diagnosed in resections. Serum β hCG, AFP, LH, FSH and testosterone levels were normal in all 4 cases. The case of malignant leydig cell tumor shows features of capsular invasion, increased cytological atypia and mitosis- 18/10hpf. (Fig 1)

**Fig 1**: Malignant leydig cell tumor: H & E (a and b): x40 and x400 showing diffuse sheets of highly pleomorphic eosinophilic cells. IHC: c) Melan A positive d) Inhibin positive e) SALL 4 negative

**CASE 5:**
A 42 year male who was operated elsewhere and slides were submitted for review. Pre operative CECT showed 1.5x1x1 cm mass at one pole of left testis. Pre and post operative serum β hCG and AFP were normal. Microscopy showed a testicular neoplasm composed of cells arranged in sheets, clusters and nests. Individual cells are round to oval with ample granular eosinophilic cytoplasm, vesicular nuclei and prominent nucleoli. Mitosis was frequent. Many clusters of hyaline globules and stromal hyalinisation was noted. Few tubular structures were noted which simulated insitu neoplasia. Morphologically a possible diagnosis of yolk sac tumor, hepatoid variant was considered and IHC was done which showed diffuse positivity for Pan CK, CD 99, WT1 and β catenin. They were negative for SALL 4, inhibin, arginase, Glypican 3, OCT 4, CD 30, CK 5/6 and calretinin. Hence, a final diagnosis of sertoli cell tumor –NOS was made.

**CASE 6:**
It was in a 46 year male who presented with scrotal swelling. USG showed a 6x6x5 cm solid growth in the upper pole of right testis. His Serum β hCG, AFP and LDH were normal. Orchidectomy was done outside and we received the slides for review. Microscopy of this tumor was different from CASE 5. This tumor showed varying morphological patterns consisting of tumor cells arranged in lobules, tubules, cords, rosettes and in solid sheets. The cells display eosinophilic to clear cytoplasm, round to oval nuclei with moderate nuclear atypia. At places the cells appear spindled. Mitosis- 12-14/10 hpf. Necrosis was noted. Morphological differential include sertoli cell tumor, seminoma and neuroendocrine tumor. IHC showed diffuse strong positivity for vimentin and nuclear β
catenin. They were negative for inhibin, calretinin, CK, SALL 4, CD 99, WT 1 and Melan A. (Fig 2) A diagnosis of sertoli cell tumor was made based on morphology and β catenin positivity although inhibin, calretinin, CD 99 were negative.

*A diagnosis of sertoli cell tumor was made based on morphology and β catenin positivity although inhibin, calretinin, CD 99 were negative.*

*Fig 2: Sertoli cell tumor: a and b: H & E x40 and x400 showing tumor cells arranged in lobules with eosinophilic cytoplasm with focal spindling and rosette formation. c and d) IHC showing cells positive for β catenin (c) and vimentin (d).*

**CASE 7:**

A 31 year male presented with a testicular mass. CECT showed a 5x5x3cm mass with intrallesional foci of small stippled calcification. CT thorax done to rule out metastasis was normal. Serum β hCG and AFP were normal. High inguinal orchidectomy was done outside with a clinical suspicion of testicular germ cell tumor. Outside it was diagnosed as seminoma histologically and we received the slides and regrossed specimen. Testis measured 7x4.5x3cm and spermatic cord was 5 cm in length. External surface was unremarkable. Cut section showed a grey white homogenous firm mass in the lower pole of testis measuring 4.2x3.2x3cm. Tumor was confined to testis. Microscopy showed a neoplasm with cells arranged in cords, trabeculae and lobular pattern intervened by fibrous/hyaline septae. The cells were monomorphic, round with clear to eosinophilic cytoplasm displaying fine chromatin and prominent nucleoli. Mitosis was frequent with focal necrosis. Hence, a possibility of NET was made morphologically and IHC was done for confirmation. The tumor cells showed diffuse strong positivity for CD 56 and focally for synaptophysin. Pan CK showed dot positivity. They were negative for chromogranin, SALL 4, PLAP, OCT3/4, Glypican 3, CK, CK7, EMA, CD 99, NKX2.2, FLI1, WT 1, desmin, D2 40, inhibin and calretinin. Ki67 proliferation was – 18%. (Fig 3) Hence, a final diagnosis of neuroendocrine tumor, Grade 2 was given based on Ki 67 index and mitosis (WHO NET criteria). Extensive sectioning was done to rule out teratomatous component.

*Fig 3: Neuroendocrine tumor of testis: a and b: H & E x40 and x400 showing monomrophic cells arranged in organoid pattern with foci of necrosis. c: CD 56 positive, d: Synaptophysin positive, e: Pan CK -Dot positive, f- SALL 4 negative*
CASE 8:
A 16 year old male who presented with left testicular mass since 6 months. CECT showed a heterogenous enhancing mass with retroperitoneal lymph node enlargement and L1 to L4 vertebral lytic lesions. Serum β hCG and AFP were normal. Intraoperatively testis was found adherent to scrotum at one end. High inguinal orchidectomy was done and we received the specimen. Grossly testis measures 11x8x7 cm with spermatic cord 6 cm in length. External surface was lobulated without any breech. Cut section showed a 9.6x7x6cm mass occupying entire testis. External surface of spermatic cord shows a firm nodule measuring 2x2x1.5cm which was separate from the testicular mass. Microscopy showed a malignant tumor arranged in diffuse sheets composed of monomorphic to highly pleomorphic cells with high nuclear cytoplasmic ratio in a myxoid stroma. Rhabdoid differentiation was noted. Mitosis-4/hpf. Tumor involves the seminiferous tubules, tunica and adjacent soft tissue. Epididymis and resected margin were involved by tumor. Lymphovascular invasion was noted. Differentials include teratoma with RMS differentiation or pure rhabdomyosarcoma and metastasis. Extensive sampling was done to rule out teratomatous component and was not found. With this a diagnosis of poorly differentiated malignant neoplasm with rhabdoid differentiation was made and IHC was done. IHC showed strong diffuse positivity for Myo D1 and desmin and myogenin was positive in the rhabdoid cells. They were negative for CD 30, SALL4, OCT ¾ and INI 1 was retained. Hence, a final diagnosis of embryonal rhabdomyosarcoma (anaplastic variant) was made.

CASE 9:
A 23 year old male who presented with cough and breathlessness. CECT thorax showed multiple nodules in the lung. Patient had history of orchidectomy 6 months back. On reviewing the orchidectomy slides, the tumor showed highly pleomorphic round cells with few spindle and rhabdoid cells in a myxoid background which showed diffuse positivity for desmin and myogenin and negative for Pan CK, SALL 4. INI 1 was retained. Here, we would like to highlight that our cases were the 3rd and 4th case in the literature to present with distant metastasis in vertebra and lung respectively.

CASE 10:
Our case was a 67 year old male who presented with a left inguinal swelling measuring 5.7x4.5 cm. FNAC done from the mass showed a high grade malignant tumor and was suggested for a biopsy. Trucut biopsy revealed cores of tumor tissue composed of highly pleomorphic epithelioid to rhabdoid cells with abundant eosinophilic cytoplasm. Brisk mitosis was noted on a background of loose stroma and haemorrhage. With this a diagnosis of poorly differentiated malignant neoplasm was made with morphological differentials of RMS, epithelioid sarcoma and melanoma. Patient had a previous history of scrotal swelling for which he had undergone high inguinal orchidectomy 1 year back outside. On reviewing the orchidectomy slides, the morphology of tumor was similar to that of inguinal swelling with highly pleomorphic cells arranged in patternless pattern with brisk mitosis and wide areas of necrosis. IHC done in both slides showed patchy SMA positivity and negative for CD 34, CD 117, desmin, S100, myogenin and myo D1.

His preoperative CECT abdomen showed a soft tissue density lesion in left testis extending to subcutaneous plane of left hemiscrotum measuring 8.46.5x9.5 cm. Serum β hCG and AFP levels were normal preoperatively. Hence, a final diagnosis of recurrent/ metastatic undifferentiated pleomorphic sarcoma was made. On reviewing the previous literature we found that dedifferentiated liposarcoma is more common at this location than UPS and can show UPS like areas. Extensive sampling might be required to look for well differentiated component.

CASE 11 and 12:
Two cases of prostatic carcinoma metastasis to testis was diagnosed one of which had ductal type morphology with intratubular growth pattern resembling primary malignancy of testis. The details are tabulated below (Table 2)

### TABLE 2: Presentation of prostatic adenocarcinoma metastasis to testis

<table>
<thead>
<tr>
<th>S.NO</th>
<th>AGE</th>
<th>Clinical presentation</th>
<th>Gross</th>
<th>Laterality</th>
<th>Microscopy</th>
<th>IHC</th>
<th>Final diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>54</td>
<td>3 months later of initial diagnosis</td>
<td>6.5x6x5cm-right solitary 0.4x0.3x0.3cm-left solitary</td>
<td>bilateral</td>
<td>Undifferentiated cells</td>
<td>PanCK, AMACR positive, LCA, SALL4, TTF1, PSA, Synaptophysin, chromogranin ve, Ki67-60%</td>
<td>metastatic acinar adenocarcinoma prostate with dedifferentiation</td>
</tr>
<tr>
<td>2</td>
<td>63</td>
<td>6 months later</td>
<td>2x1x0.6 cm solitary</td>
<td>Unilateral – left testis</td>
<td>Ductal adenocarcinoma with intraductal component</td>
<td>AMACR positive PSA- negative</td>
<td>Metastatic ductal adenocarcinoma prostate</td>
</tr>
</tbody>
</table>

### 2.3 DISCUSSION:

#### 2.3.1 SEX CORD TROMAL TUMORS:

Sex cord stromal tumors constitutes around 2-5% of all testicular tumors in adults and 25% in children, among which leydig cell tumors are the most common ones followed by sertoli cell tumors.[4]

**A) LEYDIG CELL TUMOR:**

Pure leydig cell tumors (LCT) account for 1-3% (adults) and 4% (prepubertal children) of all testicular tumors.[1] They usually present as asymptomatic testicular mass in adults whereas children present with precocious puberty due to increased testosterone levels.[2] which was concordant with our study. Majority are benign with malignant being <5% in incidence.[2] The features of malignance include tumors >5cm, infiltrative margins, vascular invasion, cytological atypia, mitosis >3/10hpf and necrosis.[2] The pathologists role lies in diagnosing these entities and differentiating benign from malignant preoperatively in biopsies or in frozen sections as testicular sparing surgeries have been tried in recent years for benign tumors. These tumors presents with classic morphology in most cases. Lesions >0.5 cm has to be differentiated from leydig cell hyperplasia. Cells with clear cytoplasm may mimic seminoma at times.

**B) SERTOLI CELL TUMOR, NOS:**

Although sertoli cell tumors are the 2nd most common sex cord tumors of testis, they constitute <1% of all testicular tumors.[3] Sertoli cell tumors of testis are more common than ovary.[4] They usually present as asymptomatic small masses confined to the testis. They are typically unilateral except the large cell calcifying type which is typically bilateral.[4]

A larger literature review done by Josias Grogg et al in evaluating the outcome of 435 cases have shown that most common histological variant was NOS (325/435, 75%), followed by large cell calcifying SCTs (99/435, 23%) and intratubular large cell hyalinizing sertoli cell neoplasia (10/435, 2%).[5] These tumors may occur throughout life but are rare in the first decade and have a peak frequency in the range of 35 to 50 years of age (mean age 45 years).[1] They rarely show metastasis and risk factors for metastatic disease included age, tumor size, necrosis, tumor extension to the spermatic cord, angiolympathic invasion, and mitotic index.

These tumors always possess a diagnostic difficulty to pathologists because of their rarity and their diverse morphological patterns mimicking primary as well as metastatic tumors. The tubular form, the classic pattern has to be differentiated from sertoli cell nodule which is usually a microscopic lesion seen commonly in undescended testis.[4] The rarer less well differentiated diffuse pattern particularly if found with clear cells admixed with inflammatory cells mimics a seminoma. A focal tubular pattern and the presence of stromal component has to be searched for differentiating both the entities.[3] Presence of hyaline globules and basement membrane like material have been described in the study by Robert H Young as a non specific finding in sertoli cell tumor NOS.[4] This may mimic a yolk sac tumor particularly when they have a microcystic/ retiform pattern. Tumors contained osteoclast-like giant cells and cells with abundant eosinophilic granular cytoplasm mimicking leydig cell tumor have also been reported.

A negative inhibin and calretinin cannot rule out sertoli cell tumor and this was supported by Kommos et al. who reviewed 66 cases of sertoli cell tumors and showed only a minority of them were positive in contrast to all the leydig cell tumors.[6] Positive nuclear staining of beta-catenin is specific for SCT-NOS, SSCT, and Sertoli-stromal cell tumor among testicular sex cord-stromal tumors but has limited sensitivity (63%).[7]
2.3.2 TESTICULAR CARCINOID:

Primary testicular neuroendocrine tumors (NETs) are rare, constituting 0.23% of all testicular tumors. Testicular carcinoid tumors can be divided into three subgroups: primary pure testicular carcinoid tumors, carcinoid tumors associated with teratoma, and carcinoid metastasis to the tests. In 1954, Simon et al. reported the first case of primary testicular carcinoid. A systematic review and meta-analysis was done by Majed et al. where they have reviewed 139 cases of testicular neuroendocrine tumors. Pure primary testicular neuroendocrine tumors were found with higher incidence (76.52%) and they are usually of prepubertal type followed by NETs with teratomas (16.67%) and secondary NETs that metastasize to testis from various other sites (6.82%). The common primary site was from GIT (88.89%). The histogenesis of pure testicular carcinoid has not been well established.

Extensive sampling and search of teratomatous components has to be done in all cases of NET to rule out NET arising from teratoma. Metastasis from other sites has to be ruled out before labelling it as primary pure intratesticular NET. This is of utmost importance because treatment differ for each of the 3 entities. Radical inguinal orchectomy with close follow-up is the treatment of choice for testicular neuroendocrine tumors. However, treatment for tumor associated with teratoma is identical to that of testicular teratoma. The indications of retroperitoneal lymph node dissection (RPLND) in pure primary testicular neuroendocrine tumor remains unclear. Somatostatin analogues were reported to be effective in patients with carcinoid syndrome and can stabilize metastatic disease progression.

2.3.3 TESTICULAR SARCOMAS:

Testicular sarcomas constitute only 1% of all testicular tumors. The most common histology was rhabdomyosarcoma (35.3%), followed by leiomyosarcoma (26.5%), and well-differentiated liposarcoma (23.5%). Intraprostatic sarcomas are very rare and mostly associated with a germ cell tumor.

A) INTRATESTICULAR RHABDOMYOSARCOMA:

Primary intratesticular rhabdomyosarcoma (ITRMS) is very rare, with just 21 cases reported in the literature till date. A largest review of ITRMS was done by Jun et al. in 2014 where they have reviewed 19 cases of primary ITRMS. Since, then we found 2 more cases in the literature. Both were embryonal subtype. According to their study, the mean age of presentation is 20 years. Embryonal RMS is the most common histological subtype accounting for 77.27% (17/22), followed by pleomorphic RMS in 13.63% (3/22), alveolar RMS in 4.5% (1/22) and 1 case of undetermined RMS. Less than 5% of sarcomas present with retroperitoneal lymph node metastasis. All the cases presented with normal serum β hCG and AFP levels except in two cases where serum AFP was mildly elevated which then were found to be mixed germ cell tumor with RMS differentiation. Hence, serum markers may help in excluding the possibility of mixed germ cell tumor with RMS differentiation. ITRMS is extremely aggressive and develops rapidly, with metastasis often occurring within a 2-year period. But, compared to paraaortic RMS they have a better prognosis.

With the advent of chemotherapy, prognosis has improved. Orchidecomy with RPLND and adjuvant chemotherapy remains the mainstay of treatment.

B) UNDIFFERENTIATED PLEOMORPHIC SARCOMA:

Testicular undifferentiated pleomorphic sarcoma (UPS) is very rare with only 5 cases reported so far among which 2 are in english literature. The prognosis of UPS is very poor wherever the site may be. Out of reported cases so far, around 21% cases had presented with recurrence. Our case presented with recurrence and lymph node metastasis. Hence, adequate surgical resection with wide negative margin and adjuvant therapy is required. Chemotherapy is required in metastatic disease with agents – doxorubicin and ifosfamide and have showed response rates ranging from 17 to 33% as single-agent therapy and from 55 to 66% when used in combination.

2.3.4 METASTATIC TUMORS:

Metastasis to testis is usually seen by hematolymphoid neoplasms. Solid tumor metastasis to testis are extremely rare compared to other visceral organs. The reason being testis is considered as a sanctuary site and the blood testis barrier formed by sertoli cells prevents the entry of foreign cells. But, even then tumors do metastasize to testis rarely through various routes. The incidence of testicular metastasis varies accordingly in different studies from 0.06% to 3.6%. The common sites of primary includes prostate (33.8%), followed by GIT (14.4%), kidney (9.8%), lung (9.6%) and melanoma (5.7%). Tumors are usually unilateral and solitary while bilaterality is seen in 13-20% cases. The mean age is 60 years. Histological patterns include interstitial (40%), destructive (26%) and nodular (21%). Many times secondary tumors in testis poses diagnostic difficulty because of the following reasons: their presentation as solitary, unilateral masses, undiagnosed primaries, intratubular growth pattern rather than intertubular particularity in prostate, bladder, and kidney tumors (due to spread of tumors through vas deferens), and presence of vacuolated cells which may mimic sertoli cell tumor. Morphological overlap occurs with sertoli cell tumors. Also metastatic tumors that have glandular differentiation has to be differentiated from glandular pattern of yolk sac tumor, primary adenocarcinoma of rete testis or adenocarcinoma arising as a somatic malignancy in a teratoma. Certain clues that may help in differentiating include absence of germ cell neoplasia insitu (GCNIS), intactubular growth pattern, presence of lymphovascular invasion and a history of primary tumor.

Prostate carcinoma metastasis are detected either incidentally after therapeutic orchietomies for prostate carcinoma or during autopsies. Incidence of testicular metastases from primary prostate carcinoma ranges between 0.18% and 0.5%.
We hereby provide a summary of various entities that provide diagnostic dilemmas in routine practice because of their overlapping morphological features along with certain diagnostic clues that may aid us in arriving at a diagnosis with the help of immunohistochemical markers. (Table 3).

**Table 3: Summary of entities with differentials, overlapping features and diagnostic clues.**

<table>
<thead>
<tr>
<th>S.N O</th>
<th>ENTITY</th>
<th>CLINICAL PRESENTATION</th>
<th>MORPHOLOGIC DIFFERENTIALS</th>
<th>OVERLAPPING FEATURES</th>
<th>DIAGNOSTIC CLUES</th>
<th>IHC</th>
</tr>
</thead>
</table>
| 1     | Leydig cell tumor | Asymptomatic, Precocious puberty, Gynecomastia, Increased testosterone | a) Leydig cell hyperplasia  
b) Granular cell tumor  
c) Seminoma | Similar morphology  
Cells with abundant eosinophilic granular cytoplasm.  
Leydig cells with clear cytoplasm | Size <0.5 cm  
Absence of reinke crystals, PAS D +  
GCNIS +, Fibrous septa with lymphoplasmacytic infiltrate, Squared nuclei | Similar Profile  
CD 68, S100 +  
SALL 4, OCT 34, CD 117 + |
| 2     | Sertoli cell tumor | Asymptomatic, Normal testosterone and AFP | a) Sertoli cell nodule  
b) Leydig cell tumor  
c) Seminoma  
d) Yolk sac tumor | Similar morphology  
Cells with abundant eosinophilic cytoplasm  
Diffuse sheeting  
Hyaline globules, reticular pattern | Smaller size, multifocal  
Absence of tubules  
GCNIS+,lymphoplasmacytic infiltrate, squared nuclei with high mitosis.  
Increased AFP, varying other patterns | β catenin +  
Inhibin, Calretinin, SF 1 +  
SALL 4, CD 117 +  
SALL 4, Glypican 3 + |
| 3     | Neuroendocrine tumor | a) Metastatic NET  
b) Teratoma associated with NET  
c) Granulosa cell tumor | Organoid pattern | Clinical history  
Extensive sampling to find teratoma component  
Call exner bodies, Varied patterns. | Synaptophysin, Chromogranin +  
SF1, inhibin + |
| 4     | Rhabdomyosarcoma | Teratoma with rhabdomyoblastic differentiation | Rhabdoid cells, Strap cells | Presence of teratoma elements | Myogenin , Myo D1 + |
| 5     | Undifferentiated pleomorphic sarcoma | a) Dedifferentiated liposarcoma | Pleomorphic cells | Presence of well differentiated liposarcoma component | MDM 2, CDK 4 + |
| 6     | Prostate adenocarcinoma metastasis | History of prostate carcinoma/ incidental | a) Sertoli cell tumor  
b) Adenocarcinoma of testis  
c) Adenocarcinoma arising in a teratoma  
d) Glandular pattern of yolk sac tumor | Glandular pattern, Intratubular growth, unilateral. | Clinical history, Serum PSA. IHC-AMACR +  
Cuboidal cells in solid pattern  
Other teratoma elements | β catenin, inhibin+  
SALL 4, Glypican 3+ |
3. CONCLUSION:

Hence, non germ cell testicular tumors although very rare one has to keep these entities in their differentials. They pose diagnostic difficulties because of their diverse histomorphological presentation and unawareness of these rare entities. Clinical and hormonal correlation may aid in diagnosis. Extensive sampling with a panel of immunohistochemical markers might be necessary to come to a definitive diagnosis.

REFERENCES: