Atypical Presentation of Seminoma in the Prostate — Case Report

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Seminoma is a very common germ cell tumor (GCT) of the testicle. It is a malignant neoplasm and is one of the most treatable and curable cancers, with a survival rate of over 95% if discovered in early stages. Primary extragonadal GCTs (EGCTs) are rare, and nearly two thirds are seminomatous tumors.1 As majority of the EGCTs originate from the midline structures, commonly reported locations include the mediastinum, thymus, retroperitoneal organs, and pineal gland.2,3 EGCTs have a good prognosis and a low rate of metastatic potential.4 They are different from primary GCTs with respect to distribution of histological subtypes, delayed presentation of EGCT, and nonseminomatous tumors are often associated with Klinefelter syndrome and hematological malignancies.5 In the absence of primary testicular involvement, seminoma originating from the prostate is extremely rare. Our extensive research yielded only 6 reported cases from all around the world.

We describe the case of a middle-aged male who presented with lower urinary tract symptoms suggestive of benign prostatic obstruction. Prostate-specific antigen (PSA) was normal and his symptoms remained unresponsive to alpha-blockers. However, histopathology confirmed the prostate as the primary location of seminoma following extensive imaging workup. Four cycles of Bleomycin, Etoposide, and Platinum chemotherapy were given postoperatively, and complete response was obtained.

CASE REPORT

We describe the case of a 54-year-old man who presented with voiding lower urinary tract symptoms for about 6 months, in 2014. Ethical approval was sought and written informed consent was taken from the patient. He was followed up at University Hospital from 2014 to date. He complained of increased frequency, nocturia, hesitancy, urgency, and poor stream. General physical examination and systemic review was normal. Inguinoscrotal examination was normal bilaterally. Digital Rectal Examination revealed a grossly enlarged prostate, indenting the rectal mucosa. Baseline work up was normal, with a PSA of 0.59 ng/mL; uroflowmetry showed a max flow rate ($Q_{\text{max}}$) of 10.5 mL/sec. An initial diagnosis of benign prostatic obstruction was made and patient was scheduled for Transurethral resection of prostate. Intraoperative findings showed a benign appearing prostate. Postoperative course remained uneventful and the patient was subsequently discharged. Histopathology revealed the diagnosis of primary seminoma of the prostate, as shown in Figure 1 A-E and subsequent imaging including ultrasound of the scrotum, Contrast computed tomography of the abdomen and chest confirmed the primary prostatic origin. The immunohistochemical stains CD117 and PLAP were also performed on the prostatic biopsy, and both were positive, however, PSA, cytokeratin (CK), CAM 1, and CK, AE1/AE3 were negative. Based on these immunohistochemical findings and reviewing the morphology, the histopathologist concluded that this is seminoma.

Lactate dehydrogenase, serum alpha fetoprotein, beta human chorionic gonadotropin levels were sent, and normal values indicated pure seminoma. He received four cycles of Bleomycin, Etoposide and Platinum chemotherapy. Post-treatment, he developed gross left-sided hydronephrosis and hydroureter with a moderate rise in creatinine levels. A left double J stent was inserted and resolution was observed on follow-up ultrasound. The stent was removed after 6 months. The patient remained well for 1 year, but again developed moderate right, and gross left-sided hydronephrosis. Cystoscopic examination revealed suspicious macular lesions in the bladder, and biopsies showed nephrogenic adenoma, which is a rare benign condition mostly due to chronic inflammatory insults and is of no serious consequences.2,6 Currently, at 4 years from the initial diagnosis, the patient is alive and healthy with no urinary symptoms and no recurrence has been identified on 2 annual follow-up positron emission tomography scans.

DISCUSSION BY M. HAMMAD Aither, MD

GCTs are broadly classified into seminomatous and nonseminomatous, and usually occur in the gonads (ovary or...
testis). EGCTs are rare and 60% of these have been reported to be seminomas. EGCTs can occur in the mediastinum, thymus, pineal gland, and retroperitoneal organs; primary prostatic seminoma is a rare entity. Furthermore, primary seminoma of the prostate is extremely rare. Only 6 cases have been reported so far (Table 1). All cases demonstrate an enlarged prostate, and 3 of them were invading the bladder neck.

CD117 staining is typically cytoplasmic, with stronger accentuation along the cell membranes. It is primarily used in the diagnosis of GISTs. It is also used to differentiate seminoma from nonseminomatous GCTs. Placental alkaline phosphatase (PLAP) is an allosteric enzyme that in humans is encoded by the ALPP gene. It is a tumor marker used in the diagnosis of seminoma and ovarian cancer. CK and CD30 stains may be useful in separating seminoma from embryonal carcinoma. Seminomas typically do not demonstrate much CK positivity, especially with the AE1/AE3 antibody. The combination of a diffusely positive placental alkaline phosphatase and CD 117, negative CK, AE1/AE3 and positive OCT 3/4 stains provides good evidence that a tumor is seminoma. Seminoma may be negative to focally positive with CK cocktail AE1/AE3 and CAM 5.2. Stains for AFP are negative in seminoma. CD 30 is also negative in seminomas but positive in embryonal carcinomas.

The urogenital system grows between the third and seventh week of fetal life. The prostate develops as an adnexal organ. Urothelium near the mesonephric duct invaginates, setting a base for the endoderm to grow into the prostate. One theory suggests that a primary seminoma of the prostate occurs due to missed/faulty migration from the yolk sac to the gonadal ridge, and the ectopic germ cells in the prostate then have a tendency to develop into a tumor. Another hypothesis postulates that pluripotent stem cells in the prostate have the potential to convert neoplastic cells to germ cells in certain individuals.

GCTs metastasizing to the prostate are also very rare. Metastases are usually testicular via the para-aortic lymphatics, and may occur from migration of tumor cells in a retrograde fashion.

The standard treatment and prognosis of patients with EGCTs remains controversial because of the rarity of the tumor in different extragonadal sites, specifically the prostate. Chemotherapy seems to be the first treatment in most patients with extragonadal seminoma. However when this disease recurs locally, surgery is still one of the treatment options, while additional chemotherapy and/or radiation are possible as well.

**CONCLUSION**

We report this rare case of a primary seminoma of the prostate. The number of reported cases is small, and chemotherapy according to the regimen for a testicular tumor seems to be the best option for this unusual case. The process of treatment and the 5-year follow-up record of this patient may serve as a valuable reference and theoretical basis for the treatment primary prostatic seminoma.
<table>
<thead>
<tr>
<th>No.</th>
<th>Year</th>
<th>Author</th>
<th>Age (yrs)</th>
<th>Symptoms</th>
<th>Initial Diagnosis</th>
<th>Findings</th>
<th>Treatment Given</th>
<th>Follow Up</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>1988</td>
<td>Arai et al</td>
<td>51</td>
<td>Malaise</td>
<td>Adenocarcinoma Prostate</td>
<td>Large prostate, retroperitoneal mass</td>
<td>Chemotherapy 4 cycles BEP</td>
<td>Alive at 05 months</td>
</tr>
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<td>2.</td>
<td>1993</td>
<td>Kandekar et al</td>
<td>58</td>
<td>Hematuria, urgency</td>
<td>Large prostate</td>
<td>Mass at bladder neck</td>
<td>Chemotherapy 2 cycles, BEP</td>
<td>Alive at 10 months</td>
</tr>
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<td>3.</td>
<td>1995</td>
<td>Hayman et al</td>
<td>31</td>
<td>Hematospermia, dysuria, hematuria, wt. loss</td>
<td>Prostatitis, prostatic abscess</td>
<td>Large prostate, iliac lymphadenopathy</td>
<td>Chemotherapy 4 cycles BEP, radiotherapy (40 Gy)</td>
<td>Alive at 13 yrs</td>
</tr>
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<td>4.</td>
<td>1995</td>
<td>Kimura et al</td>
<td>27</td>
<td>Hematuria, flank pain</td>
<td>Rhabdomyosarcoma</td>
<td>Large prostate, embryonal cell carcinoma of the left testis with pulmonary metastasis, 3 yrs after TURP</td>
<td>Surgery, chemotherapy 02 cycles PVB</td>
<td>Alive at 05 yrs</td>
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<td>5.</td>
<td>2003</td>
<td>Han et al</td>
<td>24</td>
<td>Lumbago, hematuria</td>
<td>Primary prostatic tumor</td>
<td>Yolk sac tumor with focal seminoma</td>
<td>Chemotherapy 4 cycles (cisplatin, peplomycin, doxorubicin, 6. MMC, cy7, clophosphamide, dacarbazin, etoposide)</td>
<td>Dead at 08 months</td>
</tr>
<tr>
<td>6.</td>
<td>2009</td>
<td>Hashimoto et al</td>
<td>54</td>
<td>Dysuria</td>
<td>Advanced CaP</td>
<td>Large prostate, invading the bladder</td>
<td>Chemotherapy, 3 cycles BEP</td>
<td>Alive at 28 months</td>
</tr>
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<td>7.</td>
<td>2014</td>
<td>Current case</td>
<td>47</td>
<td>Frequency, urgency, poor stream</td>
<td>Grossly enlarged prostate, BPE</td>
<td>Primary seminoma prostate</td>
<td>TURP, chemotherapy 4 cycles BEP</td>
<td>Alive at 04 yrs</td>
</tr>
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BEP, Bleomycin, Etoposide and Platinum.
References


