Case Report

Primary Seminoma of Prostate: A Case Report

Hsing-Chia Mai [1], Chung-Hsien Chen [1], Yi-Ju Chen [2], Victor C. Lin [1,3]

We report a case of primary seminoma found in the prostate. A 69-year-old male patient visited our clinic for his painless gross hematuria. We found a prostate tumor with intravesical protrusion on cystoscopy and abdominal computed tomography (CT). Pathology from transurethral resection of bladder tumor (TURBT) and the prostate (TURP) showed seminoma inside the prostate and this diagnosis was confirmed by immunohistochemical staining. There was no evidence of disease found in other areas on physical and radiographic examinations. Therefore, we diagnosed this case as a primary seminoma of the prostate, and the patient was consequently treated with etoposide and cisplatin chemotherapy after operation. Based on current research, surgical resection of residual tumor masses after chemotherapy has proven to be beneficial to the patients with extragonadal seminomatous germ cell tumors.

Key words: primary seminoma, prostate, extragonadal germ cell tumor, germ cell tumors

Introduction

In males, germ cell tumors arise predominantly from the testis, however, sometimes they can also be found extragonadal in origin. Overall, around 5% to 10% of germ cell cancers are extragonadal, mostly found at the mediastinum and the retroperitoneum. Clinically, seminoma can be presented as a painless lump on the testicles. Current treatment options for seminoma include surgery, radiotherapy, and chemotherapy.[1] Extragonadal germ cell tumors (EGCTs) can be found in the mediastinum, the retroperitoneal organs, the thymus and the pineal gland, but seminoma originating from the prostate is extremely rare.[2] The clinical symptoms of EGCTs are nonspecific and vary according to the site of tumor growth. For example, the tumors arising from the central nervous system, the mediastinum, or the thymus, may have symptoms; but the tumors from the retroperitoneum usually have no symptoms.[3] If the tumor is presented in the

From the [1]Department of Urology, [2]Department of Pathology, E-Da Hospital, [3]School of Medicine for International Students, I-Shou University, I-Shou University, Kaohsiung, Taiwan.
Received: July 24, 2017 Accepted: December 25, 2017
Address reprint request and correspondence to: Hsing-Chia Mai, Department of Urology, E-Da Hospital, No.1, Yida Road, Jiao-Su Village, Yan-Chao District, Kaohsiung City 82445, Taiwan.
Tel: +886-7-6150011 ext. 252662, Fax: +886-7-9520011, E-mail: Simonmai701112@gmail.com

prostate, it may gradually cause lower urinary tract symptoms due to the tumor size. It is difficult to diagnose a primary prostate seminoma based on clinical symptoms only. A pathological examination is the most effective and accurate tool for its diagnosis.[2] EGCTs generally have a good prognosis and low potential for metastasis.[4]

Case Report

We describe this unusual case in whom gross painless hematuria was the first sign of a primary seminoma in the prostate. Cystoscopy of the patient revealed prostatic enlargement with lump dropping. His total prostate volume was 26 mL, and initial prostate-specific antigen (PSA) was 0.371 ng/mL. Digital examination revealed a walnut sized elastic firm lesion with smooth surface of the prostate. Abdominal CT showed enlarged prostate abutting the urinary bladder and a mass on the anterior bladder neck with intraluminal projection and adherence to the prostatic base (Fig. 1). Follow-up cystoscopy revealed tumor-like lesions (Fig. 2). Due to suspected malignancy, transurethral resection of the bladder tumor and prostate was performed two months later.

The pathologic findings showed that the tumor structure had a diffused nuclear reactivity for OCT3/4, membranous positivity for placental-like alkaline phosphatase (PLAP) under immunohistochemistry study, and sheets of large round to polyhedral cells bear-

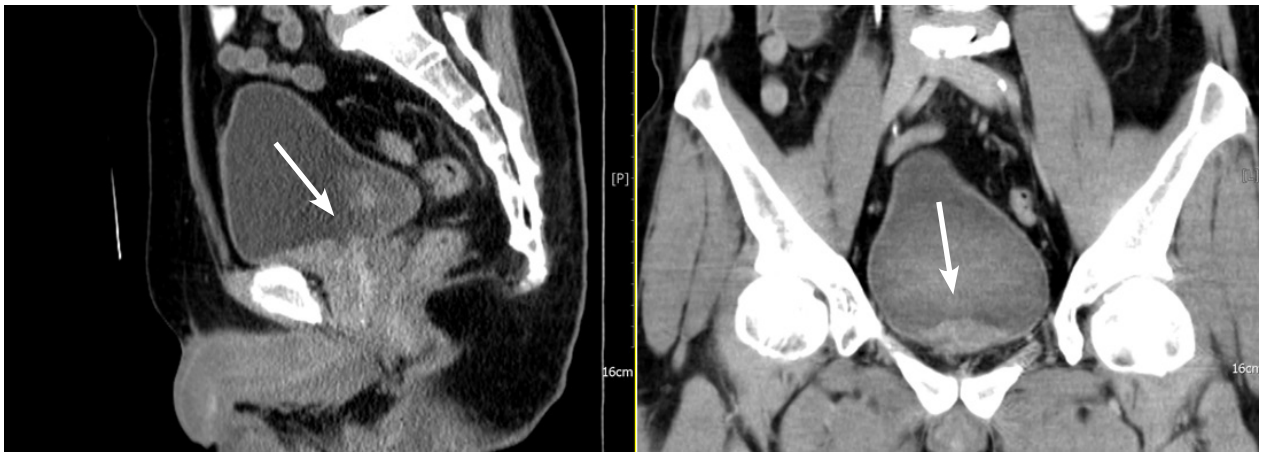

Fig. 1 Contrast-enhanced CT study with sagittal and coronal reconstruction views revealed a mass on the anterior bladder neck with intraluminal projection and adherence to the prostatic base.

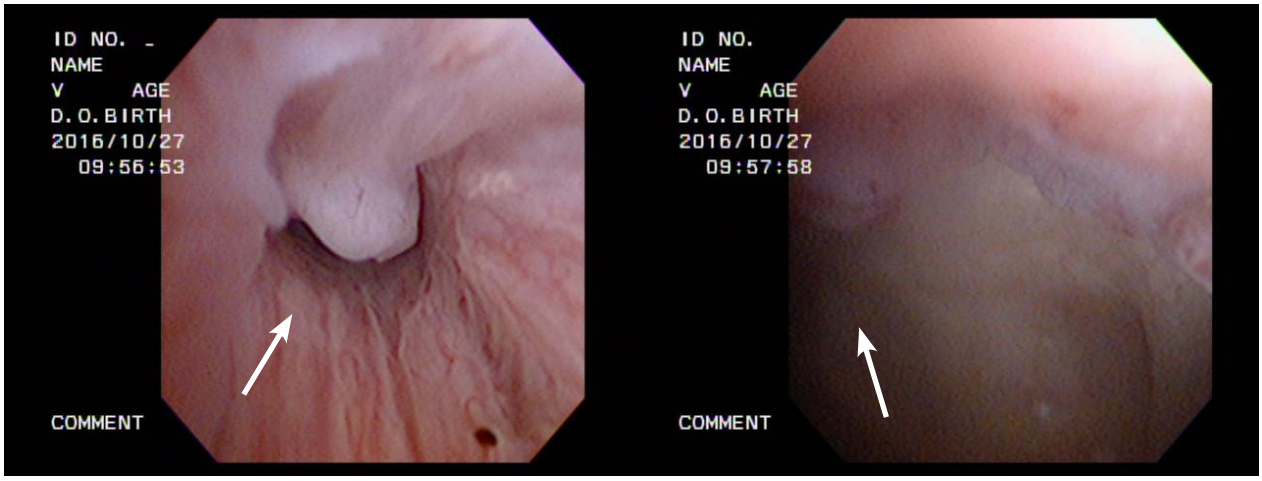

Fig. 2 Tumor-like lesions over 12 o'clock position of prostatic urethra extending to the bladder neck and anterior wall.

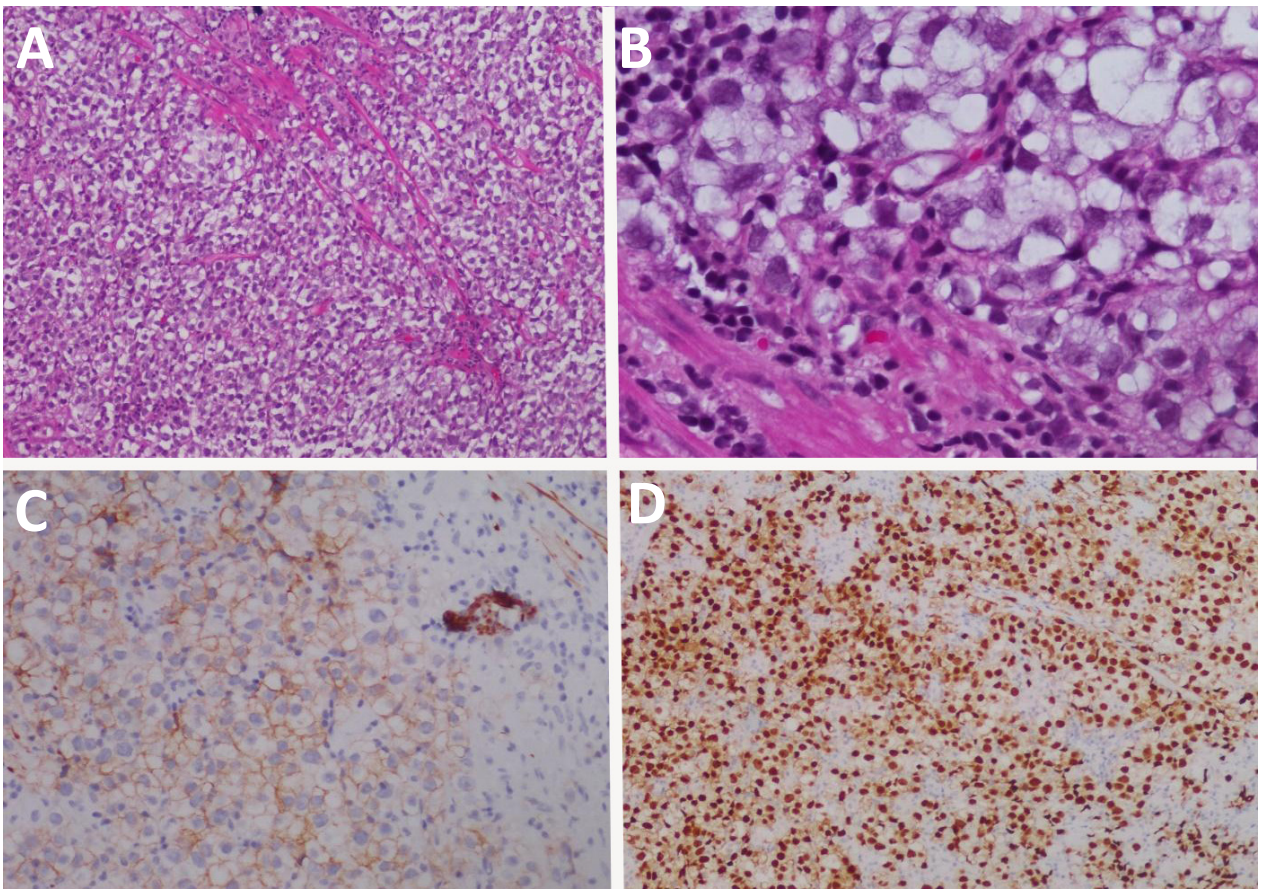

Fig. 3 Histologic and immunohistochemical studies on prostate seminoma. A: Sheets of large round to polyhedral cells bearing abundant clear cytoplasm and distinct cell membranes. (H&E, 100X) B: Delicate fibrous septa containing lymphocytes within the tumor. (H&E, 400X) C: Membranous positivity for PLAP. D: Diffuse nuclear reactivity for OCT3/4

ing abundant clear cytoplasm and distinct cell membranes under hematoxylin and eosin (H&E) stain, which are in agreement with the diagnosis of prostate seminoma (Fig. 3). The tumor involved the wall of the bladder, but no tumor mass was present in the testicles on testicular sonography examination, and no evident abnormality was identified in the mediastinum, the retroperitoneum, and the pineal gland. Therefore, the diagnosis of prostate seminoma was confirmed.

Blood samples were checked after surgery for this unusual pathological finding. Lactate dehydrogenase (LDH) tumor marker level was slightly increased (224 IU/L). PSA and β-human chorionic gonadotropin levels were normal. Alpha-Fetoprotein (AFP) (13.43 ng/mL) was slightly increased, which may be the effect of hepatitis C instead of being tumor related. We searched in the literature for treatment regimen for primary seminoma in the prostate and planned to give the patient four cycles of bleomycin, etoposide, and cisplatin (BEP) combination but we only gave etoposide and cisplatin to this patient due to his old age. Unfortunately, after three cycles of chemotherapy, the patient expired four months later due to post-chemotherapy neutropenia and sepsis.

Discussion

Gonadal germ cell tumors usually occur in the ovaries and testicles and are more common in young males. Extragonadal germ cell tumors represent 5 to 10% of all germ cell tumors in the United States (USA).[3]

Since cases are rare, there is no standard treatment for primary prostate seminoma. The treatment strategy can be similar to that of testicular seminoma. Besides surgical treatment, extragonadal seminoma is sensitive to radiotherapy and chemotherapy, which can also achieve significant positive effects on the outcome. Currently, the common medication for chemotherapy treating seminomatous germ cell tumors (SGCT) and non-seminomatous germ cell tumors (NSGCT) in mediastinal germ cell tumors are cisplatin, etoposide, and bleomycin.[5] In our case, we only administered etoposide and cisplatin due to the old age of our patient.

Testicular seminoma is sensitive to radiotherapy and chemotherapy, which can reach curative effect. The curative rate can be as high as 80% when used in good combination of chemotherapy with cisplatin.[6] Although in the short-term, outcome of chemotherapy with cisplatin is effective, the long-term effects are still unclear.[7] Prognosis is usually good, with a five-year survival rate around 85%, and low rate of recurrences or distant metastases. Common sites of metastasis include the lungs, the liver and the brain which are all sensitive to chemotherapy and radiotherapy.[5,6] The patient presented by Zheng et al. received chemotherapy with cyclophosphamide following pelvic exenteration and orchidectomy and has been disease free after 10 years of follow up.[8] The patient in Hayman et al. received chemotherapy (BEP four courses) and radiotherapy (40 Gy) also has survived after 13 years of follow up.[9] In our case, further surgical intervention can be considered if there is residual mass noted after cisplatin based chemotherapy.

Approximately 85% of extragonadal non-seminomatous germ cell tumors are associated with elevated levels of either AFP or β-hCG, or both. Approximately 50% of the patients with disseminated seminoma have a slightly increased serum β-hCG. In yolk-sac tumors and embryonal carcinoma, elevated serum AFP is common, but not in pure seminomas or pure choriocarcinomas. Pregnancy, hepatocellular carcinoma, cirrhosis, and hepatitis also may be associated with increased levels of serum AFP.[10] Our patient has a history of hepatitis C (anti-HBs positive 21.84 mlU/mL, anti-HCV positive 12.94 S/CO) and fatty change of liver parenchyma shown on abdominal CT. These can induce elevated levels of

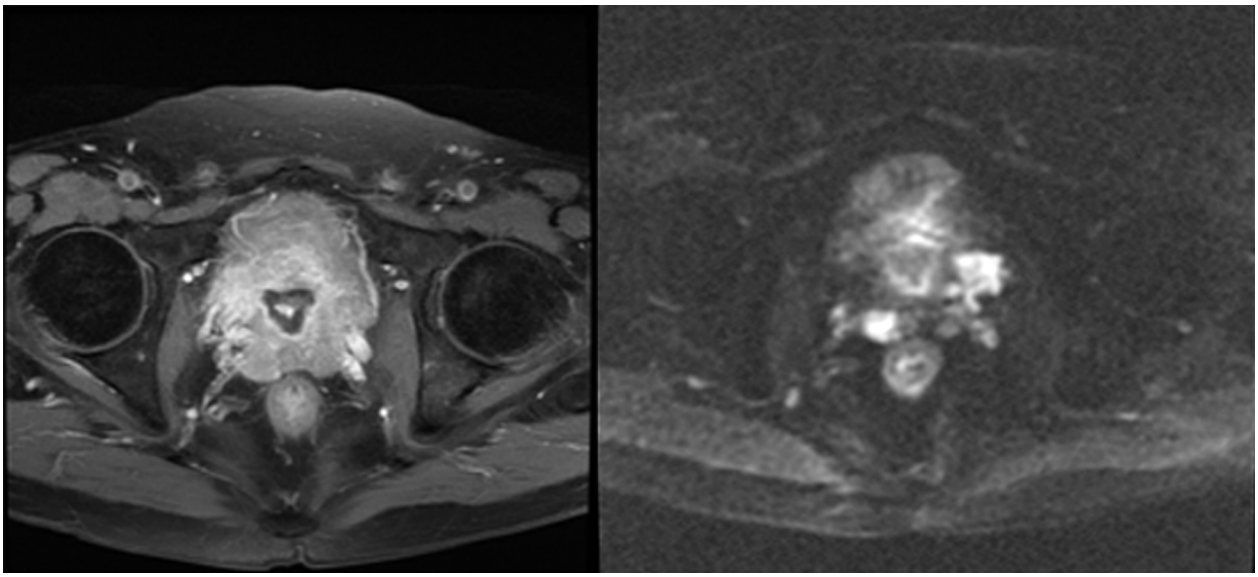

Fig. 4 Pelvic MRI before chemotherapy: status post TURBT of urinary bladder and TURP of the prostate. Wall thickening and heterogeneous enhancement are noted in urinary bladder. Also an abnormal soft tissue on the left pelvic floor, which is suspected to be a residual tumor or metastasis.

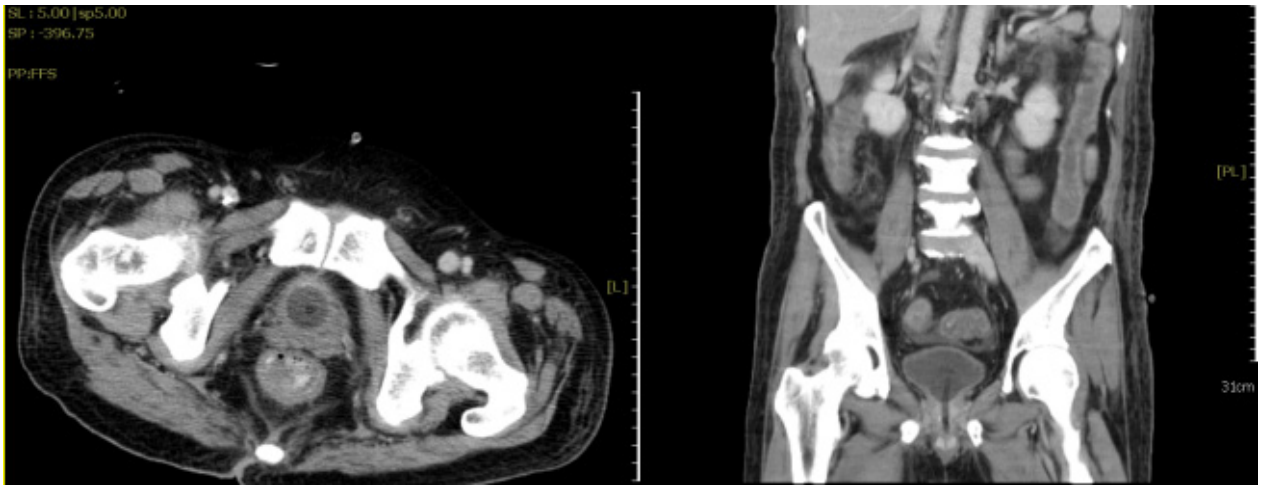

Fig. 5 Abdominal CT post-surgery and chemotherapy: no obvious local abnormal mass is noted.

serum AFP. On the other side, normal β-hCG is unusual in NSGCT, therefore, we consider this case to be an SGCT case.

Pelvic magnetic resonance imaging (MRI) from our case has revealed residual tumor or metastasis on the wall of urinary bladder base and left pelvic floor prior to chemotherapy (Fig. 4). During abdomen CT follow-up after three cycles of EP treatment regimen, this tumor mass appears to have dissolved (Fig. 5). The current research studies support the use of cisplatin based chemotherapy as beneficial to the patient. We would suggest cisplatin based chemotherapy in this SGCT case, and radiotherapy can be considered if there is also retroperitoneal lymphadenopathy. Surgical resection can be done if residual tumors are found after chemotherapy.

Our patient received first cycle of etoposide (80 mg/m^2) 130 mg and cisplatin (20 mg/m^2) 30 mg as initial dose, without side effect during hospitalization. But absolute neutrophil count (ANC) dropped to 145 mm^3 three days later after discharge. During the second cycle of chemotherapy two weeks later, the prophylaxis granulocyte colony-stimulating factor (G-CSF) was given to avoid post-chemotherapy neutropenia, but patient returned to emergency room due to urinary tract infection episode three days later, and another episode of post-chemotherapy neutropenia with absolute neutrophil count decreased to 108 mm^3 occurred several days later. Unfortunately, the patient suffered from severe neutropenia after the third cycle of chemotherapy. The ANC dropped to 13.8 mm^3 with septic shock and leading to death. Even the efficiency of treatment is important, one must reach an equilibrium between the side-effect and treatment potency.

Conclusion

We report this extremely rare case of primary seminoma of the prostate. The number of cases reported is small and no standard treatment exists for this type of case currently. Based on the treatment regimen for prostate tumors, surgical intervention followed by chemotherapy and radiotherapy according to patient's response seems to be the reasonable option for this unusual case.

References

1. Majewski W, Majewski S, Maciejewski A, et al: Adverse effects after radiotherapy for early stage (I,IIa,IIb) seminoma. Radiother Oncol 2005;76: 257-63.
2. Hashimoto T, Ohori M, Sakamoto N, et al: Primary seminoma of the prostate. Int J Urol 2009;16:967-

70.
3. Albany C and Einhorn LH: Extragonadal germ cell tumors: clinical presentation and management. Curr Opin Oncol 2013;25:261-5.
4. Stein ME, Charas T, Drumea K, et al: Spermatocytic variant of classic seminoma: a report of five cases and a brief review of the literature. Rambam Maimonides Med J 2014;5:e0021.
5. Dueland S, Stenwig AE, Heilo A, et al: Treatment and outcome of patients with extragonadal germ cell tumours-the Norwegian Radium Hospital's experience 1979-94. Br J Cancer 1998;77:329-35.
6. Gerl A, Clemm C, Lamerz R, et al: Cisplatin-based chemotherapy of primary extragonadal germ cell tumors. A single institution experience. Cancer 1996;77:526-32.
7. Powles T, Robinson D, Shamash J, et al: The long-term risks of adjuvant carboplatin treatment for stage I seminoma of the testis. Ann Oncol 2008;19:443-7.
8. Zheng W, Wang L, Yang D, et al: Primary extragonadal germ cell tumor: a case report on prostate seminoma. Oncol Lett 2015;10:2323-6.
9. Hayman R, Patel A, Fisher C, et al: Primary seminoma of the prostate. Br J Urol 1995;76:273–4.
10. Peterson ML, Ma C, Spear BT: Zhx2 and Zbtb20: novel regulators of postnatal alpha-fetoprotein repression and their potential role in gene reactivation during liver cancer. Semin Cancer Biol 2011; 21:21-7.