Burned-out testicular seminoma that metastasized to the prostate

Mehmet Yucel[1], Ugur Saracoglu[2], Soner Yalcinkaya[2], Namik Kemal Hatipoglu[2], Sahin Kabay[1], Galip Dedekarginoglu[2]

[1]Dumlupinar University Faculty of Medicine, Department of Urology Kutahya, Turkey
[2]Okmeydanı Educational Hospital, Department of Urology, Istanbul, Turkey

KEY WORDS

seminoma ◗ burned-out tumour ◗ prostate ◗ metastasis ◗ prostate specific antigen

ABSTRACT

A 49-year-old man came with complaints of macroscopic hematuria. Ultrasonography revealed an irregular cystic change of the right epididymis and prostatic enlargement. The MRI demonstrated a 5.5 x 6 cm. mass arising from an enlarged prostate and involving the bladder base and seminal vesicles. Needle biopsy of the prostate and transurethral resection of prostate (TUR-P) were performed. Because of the malignant germ cell tumor of the pathological findings classical pure seminomatous infiltration, right radical orchiectomy was performed. Pathological examination revealed a burned-out testicular tumor. Remember that there is the risk of metastases in the area of the secret primary intratubular scar (Burned-out).

INTRODUCTION

We report a case of a burned-out seminoma of the testicle with metastasis to the prostate. Seminomas metastasize predominantly to the retroperitoneal lymph nodes and subsequently to the lungs. Hematogenous spread and visceral metastases occur less often [1]. Clinically manifesting metastatic involvement of the prostate by solid tumors is very uncommon. Melanomas and lung and gastrointestinal carcinomas constitute most of the malignancies that metastasize to the prostate [2].

CASE REPORT

A 49-year-old man was referred to our clinic with complaints of macroscopic hematuria and lower urinary tract symptoms. His complaints were increasing continuously for one month. Physical examination revealed that an induration was minimally sensitive in the region of the end of right epididymis palpated in the lower pole of the right testicle approximately 1 x 1 cm in diameter. Digital rectal examination revealed a markedly nodular, firm and fixed bulging prostate. Seminal vesicles could not be palpated. The other systems examined were normal.

Urine microscopy showed sterile pyuria and hematuria. Minimal increase (313 U/l) in lactate dehydrogenase (LDH) and an increase in serum prostate specific antigen (PSA) level (7.85 ng/ml) were found in the blood biochemistry. The sedimentation rate was normal. The beta-human chorionic Gonadotropin (β-HCG) and alpha-fetoprotein (AFP) were within normal limits. A routine radiograph of the chest was normal.

Bilateral testicular ultrasound revealed only an irregular cystic change of the right epididymis. Evidence of a tumor was not found. The left testicle and epididymis were normal. Upper and lower urinary tract ultrasound showed normal kidneys bilaterally as well as prostatic enlargement (150 g). The prostatic mass invaded the vesicle neck and the right pelvic side wall. An increased post void residual volume and a lymph node in the left parailiac region, approximately 3 x 2 cm in diameter, were noted.

Intravenous pyelography revealed that bilaterally kidneys were normal. The abdominal magnetic resonance imaging (MRI) dem-

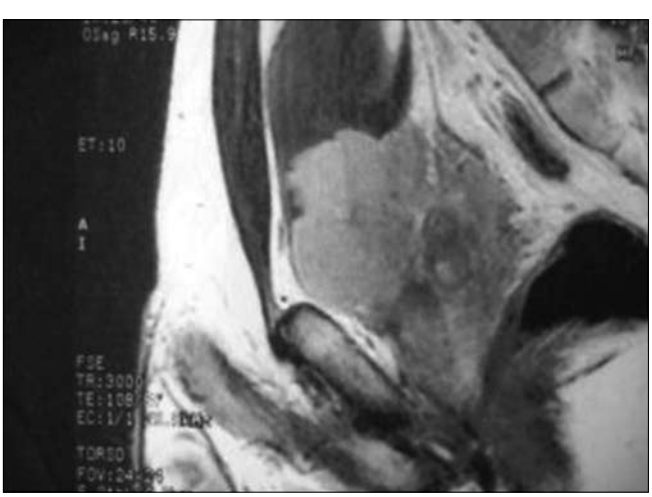

Fig. 1. The abdominal MRI demonstrated a 5.5 x 6 cm mass arising from an enlarged prostate and involving the bladder base and seminal vesicles. A 2 x 2 cm left obturator lymph node invading the bilateral neurovascular bundle can be seen as well.

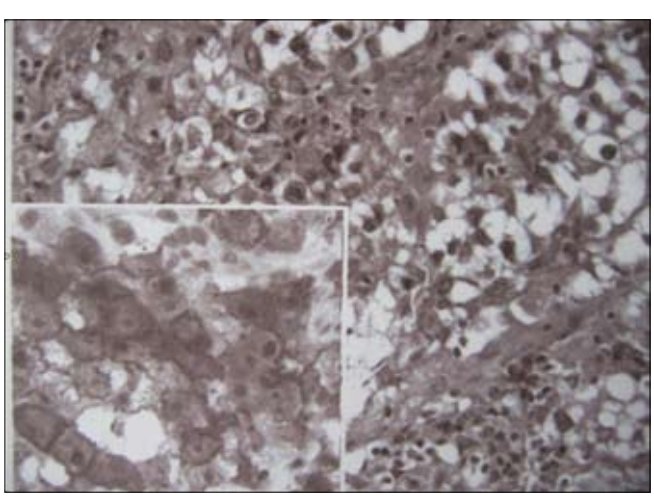

Fig. 2. Histological specimen of prostatic biopsy shows malignant germ cell tumor of pure classical seminomatous infiltration.

onstrated a 5.5 x 6 cm mass arising from an enlarged prostate and involving the bladder base and seminal vesicles as well as a 2 x 2 cm left obturator lymph node invading the bilateral neurovascular bundle (Fig. 1).

Transrectal ultrasound (TRUS) revealed an enlargement of the prostate (150 cc) that involved the bladder base, right ureter, and both seminal vesicles. A mass having a neoplastic character and showing polypoid enlargements into the periprostatic and perirectal regions of echogenic nature with irregular contour was detected. TRUS guided prostate biopsies were performed. Eight biopsies were taken and demonstrated no definite malignancy, but nuclear atypical findings were found. An immunohistochemical examination had been done for the differential diagnosis, but histological examination was not able to identify definitive malignancy. Cystoscopy showed large lateral lobes of prostate, especially the right lobe, in close proximity to the prostatic urethra with invasion of the bladder neck. Mucosa of the bladder was hemorrhagic and trabeculated. Following these investigations a transurethral resection of prostate was performed. Histopathological examination was performed again. Placental alkaline phosphatase, an immunohistochemical substance, was used to investigate the resected prostatic material. Histological examination revealed a malignant germ cell tumor of pure classical seminomatous infiltration (Fig. 2).

Due to embryologic development, primary prostatic seminoma is theoretically impossible; we returned to the right testicle, which possessed an irregular cystic change of the right epididymis, and then decided to perform a right radical orchiectomy. In microscopic evaluation of the orchiectomy specimens, a yellow colored necrosis in the soft regions (1.0 x 1.0 x 1.0 cm diameters), histiocytes and histiocytic giant cells, as well as calcification of the peripheral areas were detected. Furthermore, apart from these areas of diffuse interstitial fibrosis in the testicle parenchyma, tubular atrophy, hyalinization, germ cell aplasia, and focal small spermatogenesis were observed (Burned-out testicular tumor) (Fig. 3). The term "burned-out" tumor of the testis describes a spontaneously and completely regressed testicular tumor with no treatment. Tumoral structures were not observed in the testicle, epididymis, or spermatic cord. The left testicle was normal in ultrasonography and MRI, for this reason we did not proceed with biopsy.

Thoracic Computed Tomography (CT) was performed and no pathology was observed. Bone scan was normal.

The patient initially received five courses of PVB (cisplatin, vinblastine, and bleomycin) chemotherapy followed by external beam radiotherapy (total 3060 cGy) delivered to the para-aortic, mediastinal, and bilateral pelvic lymph nodes during an 8-week-period. Then the BEP (cisplatin 40 mg/day i.v., etoposide 190 mg/day i.v., and bleomycin 30 mg/day i.v.) treatment protocol was applied for two days. After two weeks, cisplatin 40 mg/day i.v. and etoposide 180 mg/day i.v. for three days. In all, five courses of chemotherapy were administered.

The patient's complaints decreased after radiotherapy. Follow-up CT scan demonstrated almost complete regression of both the prostatic mass and the involved nodes.

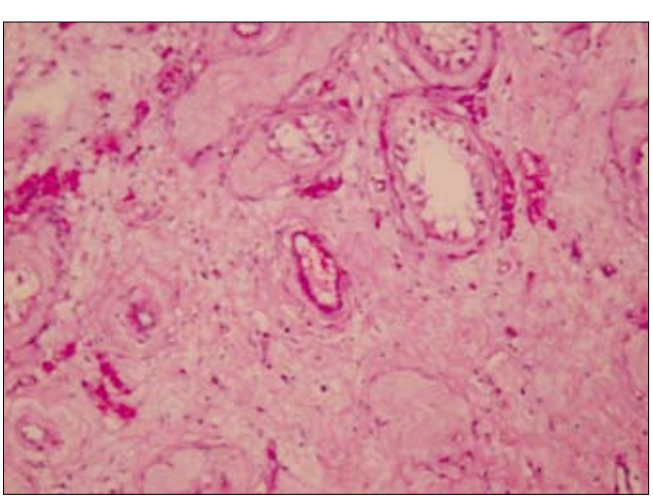

Fig. 3. Histological specimen of the testis shows large hyalinized areas, tubular hyalinization, interstitial fibrosis, and focal Leydig cell hyperplasia (Burned-out germ cell tumor).

DISCUSSION

Seminomatous metastasis of testis usually occurs via the lymphatic system, especially by way of the retroperitoneal lymph nodes. Epididymal invasion of the tumor increases the risk of iliac lymph node involvement. Tunica vaginalis invasion or in scrotal surgery inguinal lymph node metastases may occur [3].

Visceral metastases are generally observed in lungs, liver, brain, kidneys, gastrointestinal tract, bones, adrenal glands, peritoneum, tonsil and spleen, which were rare (5%). In autopsy studies (12%) thyroid and prostate glands metastases were reported [4, 5]. One theory is that retrograde passage of metastatic germ cell tumors may occur in pelvic lymphatic vessels obstructed by neoplastic involvement, which becomes responsible for prostatic seeding.

Burned-out testicular tumor describes a spontaneously and completely regressed testicular tumor. It presents by its metastases to the retroperitoneum, mediastinum, lymph nodes, lungs, liver, and prostate [2]. Prym [6] had reported testicular scarring in a patient with an extragonadal tumor at autopsy in 1927.

Comiter et al. [7] reported diffuse atrophy a hematoxyphilic bodies and psammoma bodies in some patients. They believed that these bodies represent echogenic foci on sonography. Ultrasonographic findings in burned-out tumors are increased echogenicity in a focal area. These findings are caused by calcium and fibrosis. Pathologically, there is no tumor in the testis [8].

Despite normal ultrasonographic findings, if there is any risk factor for *in situ* malignancies, testicular biopsy must be performed. Azzopardi et al. [9] showed cell-poor, collagen-rich fibrous scar in which some metastatic nonseminomatous germ cell tumors in some palpably normal tests of the patients. They detected hematoxyphilic deposits in seminiferous tubuli which were called hematoxyphilic bodies.

The diagnosis of seminoma was based on the characteristic histological type of tumor. This was supported by the immunohistochemical studies; in particular the negative stains for epithelial markers and the positive stain for placental alkaline phosphatase. Staging and therapy of these tumors must be similar to that of primitive testis tumors.

Primary seminoma of the prostate is very rare and only four cases have been reported previously [10]. The source of this tumor is not known and contoversial. The prostate develops between the 4^{th} and 7^{th} months of fetal life and is very closely related to seminal vesicle, ductus deferens, and urethra development. It has been suggested that there could be a sequestration of germ cells during migration from the *de-novo* metaplastic-dysplastic stem cells or the primitive yolk sac to the gonadal region and that a tumor in the prostate could arise from such a developmental defect. But remember, that is a risk of metastases to the secret primary intratubular scar (Burned-out).

Only four previous cases of primary testicular seminoma, metastatic to the prostate, have been published [3, 4, 10]. In all of these cases, seminoma was shown in the testis and prostate at same time. Although in our case seminoma was shown only in prostate specimens. Testicular specimen did not show any tumoral differen-

tiation and burned-out testicular carcinoma was detected.

Treatment alternatives are centered on the combination of chemotherapy and radiation therapy. For extragonadal germ cell tumors, radiation is associated with survival rates of 50-60%. In addition, the combination of surgery and radiation controls the local and regional disease in most patients. Distant metastasis is responsible for 40-50% of mortality [11, 12].

Our patient was treated with combined therapy of etoposide, cisplatin, and bleomycin. This regimen is highly effective in the treatment of seminomatous and nonseminomatous germ cell tumors. The patient had a complete response after cisplatin-based chemotherapy and continues to be tumor-free 7 years after diagnosis.

REFERENCES

1. Ray B, Hajdu SI, Whitmore WF Jr: *Distribution of retroperitoneal lymph node metastases in testicular germinal tumors*. Cancer 1974; 33: 340-348.
2. Johnson DE, Chalbaud R, Ayala AG: *Secondary tumors of the prostate*. J Urol 1974; 112: 507-508.
3. Sagalowsky AI, McConnell JD, Admire R: *Uncommon sites of recurrent seminoma and implications for therapy*. Cancer 1986; 57: 1060-1065.
4. Motley RC, Utz DC, Farrow GM, Earle JD: *Testicular Seminoma Metastatic to the prostate*. J Urol 1986; 135: 801-802.
5. Johnson DE, Appelt G, Samuels ML, Luna M: *Metastases from Testicular Carcinoma. Study of 78 autopsied cases*. Urology 1976; 8: 234-239.
6. Prym P: *Spontanheilung eines bosartigen, wahrscheinlich chorionephiteliomatosen gewachses im hoden*. Virchows Arch Pathol Anat 1927; 265: 239-258.
7. Comiter CV, Renshaw AA, Benson CB, Loughlin KR: *Burned-out primary testicular cancer: sonographic and pathological characteristics*. J Urol 1996; 156: 85-88.
8. Shawker TH, Javadpour N, O'Leary T et al: *Ultrasonographic detection of "burned-out" primary testicular germ cell tumors in clinically normal testes*. J Ultrasound Med 1983; 2: 477-479.
9. Azzopardi JG, Mostofi FK, Theiss EA: *Lesions of testes observed in certain patients with widespread choriocarcinoma and related tumors. The significance and genesis of hematoxylin-staining bodies in the human testis*. Am J Pathol 1961; 38: 207-225.
10. Farnham SB, Mason SE, Smith JA Jr: *Metastatic testicular seminoma to the prostate*. Urology 2005; 66: 195.
11. Einhorn LH, Williams SD, Loehrer PJ et al: *Evaluation of optimal duration of chemotherapy in favorable-prognosis disseminated germ cell tumors; a South-eastern Cancer Study Group protocol*. J Clin Oncol 1989; 7: 387-391.
12. Motzer RJ, Bosl GJ, Geller NL et al: *Advanced seminoma; the role of chemotherapy and adjunctive surgery*. Ann Intern Med 1988; 108: 513-518.

Correspondence

Mehmet Yucel
Dumlupinar University
Department of Urology
43270 Kutahya, Turkey
phone: +90 274 2652031
myucel75@gmail.com