

Spermatocytic seminoma: Review of the Literature and Description of a new case Managed by Surveillance

Anouar El Ghazoui, Hayani Mounir, Hamza Lamchahab, Tarik Karmouni Khalid Elkhader, Abdelatif Koutani and Ahmed Ibn Attaya
Urology B, Ibn Sina University, Hospital, Rabat, Morocco.

ARTICLE INFO

Article history:

Received: 2 February 2016;

Received in revised form:

1 March 2016;

Accepted: 4 March 2016;

Keywords

Spermatocytic,
Seminoma,
Surveillance.

ABSTRACT

Spermatocytic seminoma (SS) is a distinct testicular germ cell tumor, representing less than 1% of testicular cancers. The clinical features that distinguish (SS) from classical seminoma (CS) are an older age at presentation and a reduced propensity to metastasize. Currently, the management (SS) has changed to the increased use of surveillance, provided that there are no risk factors which may predict recurrence. Here, we report an additional case of SS and present a comprehensive relevant literature review concerning current clinical, histopathological, and therapeutic features.

© 2016 Elixir all rights reserved.

Introduction

Spermatocytic seminoma is an uncommon neoplasm first described by Masson in 1946 and rarely occurs before the fifth decade. It represents 1 to 2% of germ cell tumors and 4 to 7% of all seminoma patients [1-2]. Unlike classical seminoma (CS) originated from undifferentiated germ cells, (SS) may derive from spermatogonia and represented a more differentiated type of germ cell neoplasm.

It is a solid tumor found solely in the testis with long duration of symptoms, presentation evident at an early stage, absence of metastasis, and bears an excellent prognosis [3].

Immunohistochemical staining can be extremely helpful to assess the diagnosis based on the negativity of all tested classic markers [4,5]. (SS) rarely metastasizes and there is no documented benefit of radiotherapy or preventive chemotherapy [3-6].

Observation

A 42-year-old man presented complaining of gradually increasing right testicular painless swelling for one year. There was no history of cryptorchidism, bilateral scrotal pain, voiding complaints, local trauma, weight loss, or here dietary disease. A comprehensive physical examination revealed right testis enlargement and displayed firm consistencies to palpation. Inguinal lymph nodes were not palpable.

Scrotal ultrasonography revealed a well-defined 65 × 30 × 25 mm right testicular solid tumor with heterogeneous echogenicity associated with a small hydrocele (Figure 1).

The tumor markers alpha-fetoprotein, human chorionic gonadotropin, and serum lactate dehydrogenase were within normal limits.

The patient underwent outside our department of a right orchietomy via scrotal approach. On gross examination, the testicle measured 12 × 6 × 3 cm and weighed 174 g. The masse had fleshy, pale-grey cut surfaces within vasion of the tunica (Figure 2). There were some skin changes, whence the

realization of further scrotal excision and complete removal of the rest of the spermatic cord.



Figure 1. Intra-testicular solid tumor with hydrocele



Figure 2. Right orchietomy Piece

A histological examination confirmed the S(S) (Figures 3 and 4) showing anarchic cell proliferation, making tablecloths and pseudo glandular areas located within a very small and edematous stroma. We can distinguish within this germ component three types of tumor cells; small cells with dense and hyperchromatic nuclei surrounded by a thin cytoplasmic halo. Intermediate-sized round cells with granular chromatin and eosinophilic cytoplasm. Finally large cells with enlarged nuclei and filamentous chromatin.

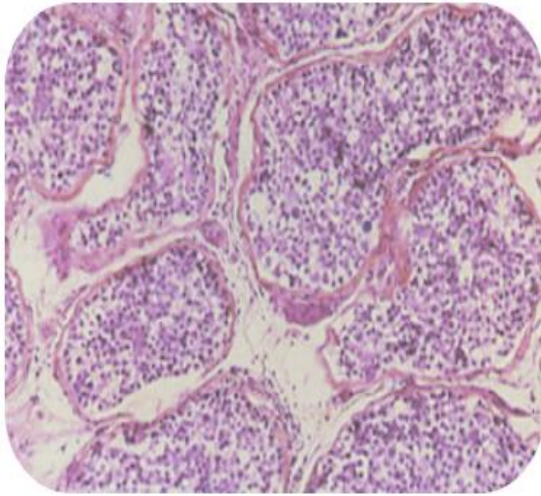


Figure 3. Polymorphic intra-tubular cell proliferation

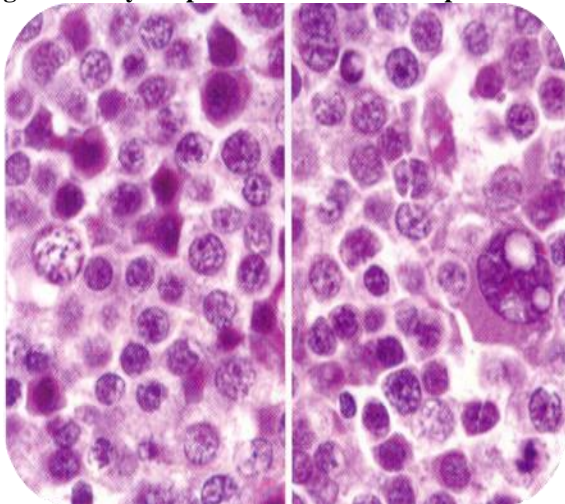


Figure 4. Germinal proliferation with variable-sized cells

Computed tomography of the thorax, abdomen and pelvis was negative for lymphadenopathy or other metastases.

Following the operation, the patient was followed closely without any adjuvant therapy and was in good condition with no evidence of metastasis 24 months after the operation.

Discussion

The (SS) has been regarded as a malignancy along the lines of CS, but it exhibits different pathology and natural history, albeit the same clinical behavior. It is an uncommon tumor and, at our institution, represents less than 1% of all (CS) patients and 4.4% of stage I. (SS) is found exclusively in the testis and is not associated with any known risk factors for germ cell tumors including cryptorchidism, subfertility, or gonadal dysgenesis[6]. These tumors originate from a post-natal germ cell [2]. The detection of proteins SCP1 and XPA, which are normally expressed in the primary and pachytene spermatocyte stages, provide a clue that the origin of SS is in a more differentiated cell than in classical seminoma [7].

Clinically, the main difference between spermatocytic and classical seminoma is the age of occurrence. (SS) tends to

occur more commonly, in men aged over 50, while in (CS), the age at diagnosis is between 25 and 40 years. The duration of symptoms was on the whole longer compared with (CS), indicating a slower evolution and less malignant biological behavior.

The size of the tumor was ranged from 10 to 16 cm with an average of 6.6cm [8], usually replacing the whole testis.

The spermatocytic variant is distinct from (CS) in its morphological characteristics with three different cell types (small, medium, large), spherical nuclei, eosinophilic to amphophilic cytoplasm, lack of cytoplasmic glycogen, and sparse to absent lymphocytic infiltrate[9]. Others studies reporting different histogenesis of SS in comparison with (CS) and based on analysis of DNA ploidy and immunohistochemical profiles. While SS contains diploid to polyploid cells as the principal finding, (CS) is predominantly aneuploid [10].

Differential features between Spermatocytic and Classical Seminoma are presented in table 1.

Table 1. Clinical and Pathological Comparison of Spermatocytic Seminoma with Classic Seminoma

	Spermatocytic seminoma	Classic seminoma
Clinical		
1. Site of origin	Testis only	Testis, ovary, retroperitoneum, central nervous system (midline structures)
2. Arise in cryptorchid testes	No	10%
3. Age (years): mean (range)	54 (25-87)	41 (childhood to 85+)
4. Fraction of testis involved by tumor	2%	40%
5. Associated other germ cell tumor types	None	Common
6. Association with sarcoma of testis	5%	None
MICROSCOPIC PATHOLOGY		
1. Cell size	Small, medium, large	Medium
2. Nuclei	Spherical	Irregular
3. Cytoplasmic glycogen	Absent	Abundant
4. Lymphocyte-rich fibrovascular septae	Absent	Present
5. Associates in intratubular germ cell tumor	None	Common
MOLECULAR BIOLOGY/IMMUNOHISTOCHEMISTRY		
1. Placental alkaline phosphatase staining	Rarely	Strong, diffuse
2. CD-117 staining	Absent	Present
3. Cytokeratin 18	Absent	Present
4. Gene over expression chromosome 9	Positive	Negative

The presence of an anaplastic component does not seem to impact the excellent prognosis of (SS). The malignant potential of (SS) is very low. Only proven three cases of metastatic (SS) have been described [11-12]. The sarcomatous component is usually rhabdomyosarcoma or undifferentiated, high-grade sarcoma and it appears that the metastatic disease develops usually from the sarcomatous elements [13]. The sarcomatous differentiation was associated in the most

reported cases with aggressive behavior and poor outcome [13,14].

The choice of therapy for an individual patient requires a consideration of the patient's ability to comply with a surveillance regimen as well as acute and delayed complications of adjuvant chemotherapy or adjuvant radiotherapy. We generally suggest active surveillance for patients able to comply with an intensive follow-up schedule, because of the decreased risk of late complications and because of the ability to achieve the same overall cure rate when patients who relapse are treated appropriately.

Primary tumor size greater than 4 cm and invasion of the rete testis have been identified as independent factors associated with an increased risk of relapse in multivariate analysis [15]. However, surveillance is not contraindicated in men with these features, provided the patient understands that the risk of relapse may exceed 30 percent and that they must adhere rigorously to the surveillance protocol. For patients with clinical stage I seminoma for which active surveillance is not appropriate and for those who want to minimize any risk of relapse, adjuvant chemotherapy with single agent carboplatin is suggested rather than RT.

In all cases, there is no unanimity in the therapeutic procedure of (SS). It was stated that (SS) is a radio sensitive tumor [16], but no direct evidence for this sensitivity was presented and the usefulness of postoperative radio therapy was doubted. However, the majority of reported patients in the literature with (SS) have received postorchidectomy radio therapy to the draining lymph node area.

The main benefit of surveillance is that it avoids unnecessary treatment and the associated treatment related adverse effects.

Conclusion

Spermatocytic seminoma is a distinct neoplasm both clinically and pathologically from (CS) and it differs from the latter specially by its behavior, characterized by an almost complete inability to metastasize with only very few examples described with metastatic behavior.

The fact that radiotherapy is not necessary is important in view of the fact that many patients with (SS) are elderly and may be adversely affected by treatment.

Conflict of Interests

The authors declare no conflict of interest.

Authors contribution

All authors mentioned have contributed to the development of this manuscript. All authors also declare to have read and approved the final manuscript

Reference

1. Dundra Pavel, Pešl Michael, Povýšil Ctibor, Prokopová Petra, Pavlík Ivan, Soukup Viktor, Dvor Jan, Čekb : Anaplastic

variant of spermatocytic seminoma. *Pathology - Research and Practice* 2007, 203(8):621-62.

2. Gorecki T, Kaszuba B, Ostrowska M, Mazurczyk K, Spliter L: Giant spermatocytic seminoma with massive hemorrhage into accompanying hydrocele: case report. *Int Urol Nephrol* 2005, 37(3):529-531.

3. Chelly I, Mekni A, Gargouri MM, Bellil K, Zitouna M, Horchani A, Kchir N: Spermatocytic seminoma with rhabdomyosarcomatous contingent. *Prog Urol* 2006, 16(2):218-220.

4. Baldet P: Tumeurs germinales du testicule, conceptions actuelles. *Ann Pathol* 2001, 21(5):399-410.

5. Ulbright TM: Germ cell neoplasms of the testis. *Am J Surg Pathol* 1993, 17(11):1075-91.

6. Hel Fellah, Tijami F, Jalil A: Séminome spermatocytaire (à propos de deux cas). *J Maroc Urol* 2008, 11:25-28.

7. Rosai J, Silber I, Khodadoust K: Spermatocytic seminoma. Clinicopathologic study of six cases and review of the literature. *Cancer* 1969, 24:92-102

8. Oosterhuis J Wolter, Looijenga HJ Leendert: Testicular germ-cell tumours: a broader perspective. *Nat Rev Cancer* 2005, 5(3):210-222.

9. Burke AP, Mostofi FK: Spermatocytic seminoma: a clinicopathologic study of 79 cases. *J Urol Pathol* 1993, 1(6):21-32.

10. Eble JN: Spermatocytic seminoma. *Hum Pathol* 1994;25(10):1035-42.

11. Verdorfer I, Rogatsch H, Tzankov A, Steiner H, Mikuz G: Molecular cytogenetic analysis of human spermatocytic seminomas. *J Pathol* 2004, 204(3):277-281.

12. Horn T, Schulz S, Maurer T, Gschwend JE, Kübler HR: Poor efficacy of BEP polychemotherapy in metastatic spermatocytic seminoma. *Med Oncol* 2011 Dec;28 Suppl 1:S423-5. doi: 10.1007/s12032-010-9739-1. Epub 2010 Nov 18.

13. True LD, Otis CN, Rosai J, Scully RE, Delprado W: Spermatocytic seminoma of testis with sarcomatous transformation. *Am J Surg Pathol* 1988, 12(10):806.

14. Trivedi P, Pasricha S, Gupta A: Spermatocytic seminoma associated with undifferentiated sarcoma: a rare case report. *Indian J Pathol Microbiol* 2011, 54(1):138-140.

15. Warde P, Specht L, Horwich A, et al: Prognostic factors for relapse in stage I seminoma managed by surveillance: a pooled analysis. *J Clin Oncol* 2002, 20(22):4448.

16. Talerma A: Spermatocytic seminoma. Clinicopathological study of 22 cases. *Cancer* 1980, 45(8):2169-2176.