Original Research Paper

Volume-8 | Issue-2 | February-2018 | PRINT ISSN No 2249-555X

Pathology

SPERMATOCYTIC SEMINOMA REVIEW.

Dr Anuradha R Bindu — Bindu Hospital, Aurangabad

Rajan S Bindu — Professor and Head, Department of Pathology, Government Medical College and Cancer Hospital, Aurangabad, Maharastra, India.

Santosh G Rathod* — Senior Resident, Department of Pathology, Government Medical College and Cancer Hospital, Aurangabad, Maharastra, India. *Corresponding Author

ABSTRACT Spermatocytic seminoma is a rare germ cell tumor distinct from classical seminoma, both clinically and pathologically. It affects older men, has not been associated with a history of cryptorchidism, and has no known counterpart in ovary or any other site. Pathologically, it is characterized by 3 distinct cell types, lack of cytoplasmic glycogen, and scant to absent lymphocytic infiltrate. Gain of chromosome 9 is the most consistent genetic abnormality. There have been few case reports of sarcomas arising in spermatocytic seminoma and only an occasional report of metastasis. It is important to differentiate this condition from its frequent mimics, such as classic seminoma and embryonal carcinoma, because patients with spermatocytic seminoma may not require further treatment after surgery.

KEYWORDS :

Spermatocytic seminoma is a rare testicular neoplasm which occurs only in adults (mean age 54years). It has no ovarian homologue.[1–4] clinically, and histologically, three entities of germ cell tumors can be distinguished in the human testis. The first group includes teratomas–yolk sac tumors, which become manifest usually within the first 4 years of life and almost always before puberty. The second group comprises seminomas and nonseminomatous germ cell tumors, which manifest after puberty. The third group includes spermatocytic seminomas, which usually affect older men.[5]groups differ in their presentation, treatment, and prognosis. Seminoma is the most common testicular tumor and the only one that is treated with radiation.[1]

Spermatocytic seminoma was first described by Masson et al [6]1946. It is a rare tumor, with a frequency varying from 1.3% to 2.3% for all patients with seminoma.[7,8] the age-standardized incidence rate of spermatocytic seminoma is 0.4 cases per million.[9]

HISTOGENESIS

The origin of seminomas and spermatocytic seminomas of the adult testis remains disputed. It is thought that spermatocytic seminomas originate from cells capable of maturing at least to the stage of spermatogonia-pachytene spermatocyte. This theory is supported by the presence of proteins encoded by genes*SCP1*(synaptonemal complex protein 1),*XPA*(Xeroderma pigmentosa type A), and *SSX* (synovial sarcoma on X chromosome) in spermatocytic seminomas. The absence of these proteins in conventional seminomas points to the embryonic germ cell as the cell of origin[10]expression profiling of 156 microRNAs with quantitative polymerase chain reaction demonstrated that the spermatocytic seminoma cluster is in the same branch as the more differentiated tissue like normal testis and teratomas.in seminomas and dysgerminomas also cluster with embryonic carcinomas.[11]

CLINICAL FEATURES

Most spermatocytic seminomas occur in older white men, in their sixth decade of life[7]usually manifest as a unilateral, painless swelling of variable duration without an associated history of cryptorchidism .

MACROSCOPY

Spermatocytic seminoma lacks an ovarian homologue and has been found only in the testis.

The tumor size ranges from 2 to 20 cm with an average of 7 cm.[8]Grossly, spermatocytic seminomas are often homogenous, solid, pale grey, soft, well-circumscribed, lobulated, cystic, hemorrhagic, edematous, and even necrotic with bulging mucoid cut surfaces(Figure 1)[7]

HISTOPATHOLOGY

The tumor is well-circumscribed and encapsulated with rare extension into the paratesticular soft tissue. The tumor cells are noncohesive with little or no intervening stroma. Collagen bands, when present, may enclose tumor compartments, but lymphocytic infiltration and granulomatous stromal reaction are not characteristic . Typically, there are 3 types of cells (Figure 2 and 3). The predominant cell type is of medium size, 15 to 20 µm, with variable amount of dense eosinophilic cytoplasm and a round nucleus, often with a fine granular chromatin. The second type of cell is small, 6 to 8 µm, with dark-staining nuclei and scant eosinophilic cytoplasm resembling lymphocyte. The third cell type is large, 80 to 100 µm, mononucleated or, rarely, a multinucleated giant cell with round, oval, or indented nuclei. These often have the typical spiremelike lacy chromatin distribution. Sometimes, the cells are relatively monotonous with prominent nucleoli, although wider sampling reveals characteristic areas. Mitoses including abnormal forms are frequent. [12]Glycogen is not demonstrable on periodic acid–Schiff stain.[7]

IMMUNOPROFILE

Many of the markers useful in other types of germ cell tumor are generally negative in spermatocytic seminoma, including OCT3/4, AE1/AE3, and CD30 -Kit staining has been shown to be positive in around 40% of the spermatocytic seminomas.[13] [14]Placental alkaline phosphatase has been observed in isolated or small groups of tumor cells.[15]The cancer-specific antigen NY-ESO-1 is found in 50% of spermatocytic seminomas but not in other germ cell tumors.[16]stains like α-fetoprotein, human chorionic gonadotropin, carcinoembryonic antigen, S100 protein, vimentin, epithelial membrane antigen, leukocyte common antigen, neuron-specific enolase, and human placental lactogen are not demonstrable in spermatocytic seminoma.[17]

ANAPLASTIC AND SARCOMATOUS CHANGE

Anaplastic change is heralded by presence of prominent nucleoli in the 3 cell types seen in spermatocytic seminoma. Cells with vesicular nucleus resembling embryonal carcinoma, bizarre giant cells, areas of necrosis, and frequent mitosis, including abnormal forms and blood vessel invasion, are also seen. These features are not absolute and can be rarely seen in conventional spermatocytic seminomas. Sarcomatous change in spermatocytic seminoma is characterized by the presence of 2 components. One component is typical spermatocytic seminoma. The second component usually comprises undifferentiated sarcomatous elements and, occasionally, more differentiated sarcoma such as rhabdomyosarcoma. Such change confers a worse prognosis and is often lethal.[18–19]

ULTRASTRUCTURE

The most important ultrastructural features are prominent nucleolus with dispersed nucleolonema and specialized cell junctions of the zonula adherens type, true intercellular bridges, identical to those normally found between spermatocytes and between spermatids. Comparison of these findings with ultrastructure of classic seminomas

suggests that both originate from the same cell type but also indicates that spermatocytic seminoma is a tumor distinct from the former by virtue of its greater differentiation.[20]

DIFFERENTIAL DIAGNOSIS

Spermatocytic seminoma is most commonly misinterpreted as typical seminoma, embryonal carcinoma, or lymphoma. The distinction of spermatocytic seminoma from other forms of testicular germ cell tumor is important because spermatocytic seminomas, unless complicated by sarcomatous transformation, lack the capacity to metastasize and therefore are adequately treated by orchiectomy alone without any form of adjuvant therapy.[7]Spermatocytic seminoma lacks the features of classic seminoma including a fibrous stroma, lymphocytic and/or granulomatous stromal reaction, cells with abundant glycogen, positivity for placental alkaline phosphatase, and intratubular germ cell neoplasia component. Unlike many usual seminomas, the cytoplasm in spermatocytic seminoma is typically dense and amphophilic rather than clear.

Lymphoma has a predominant interstitial growth pattern with a relatively monotonous cell population that lacks the lacy chromatin distribution. Relatively monomorphic examples of spermatocytic seminoma may particularly be misdiagnosed because they have a sheetlike arrangement of large tumor cells with round nuclei, prominent nucleoli, and frequent mitotic figures but they are negative for leukocyte commen antigen.[17]

Embryonal carcinoma lacks the 3 different types of cells described in spermatocytic seminoma..

GENETICS

The gain of chromosome 9 appears to be a consistent finding in all spermatocytic seminomas, which is not found in classic seminomas.[20] genetic abnormalities include gain of X chromosome.

TREATMENT AND PROGNOSIS

Spermatocytic seminomas rarely metastasize and hence orchidectomy alone is indicated for treatment.7

SUMMARY AND CONCLUSION

Spermatocytic seminoma is a rare testicular tumor that poses a diagnostic challenge. It should be considered, especially while evaluating germ cell tumor in older men. It is distinct in its histologic appearance with 3 different cell types, lack of cytoplasmic glycogen, and sparse or absent lymphocytic infiltrate. Since it rarely metastasizes or undergoes sarcomatous differentiation, correct histologic diagnosis can have great impact on treatment and prognosis.

Grossly, spermatocytic seminomas are often homogenous, solid, pale grey (FIG.1)

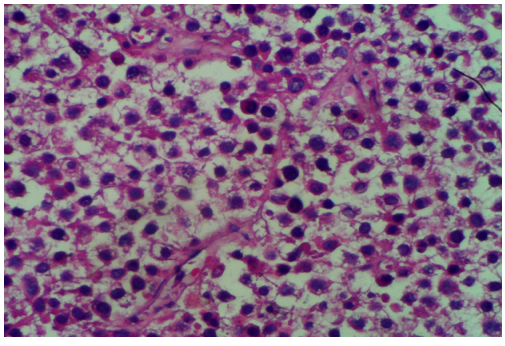

FIGURE 2. Spermatocytic seminoma consisting of sheets of round cells.

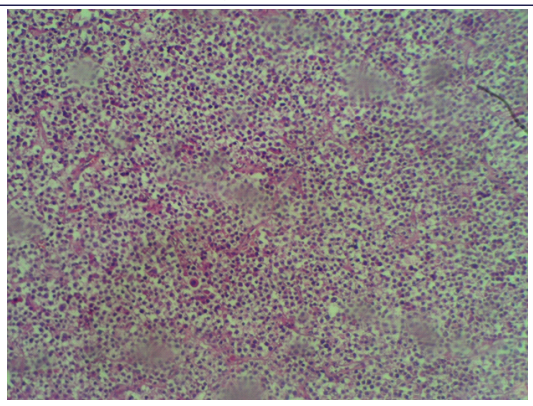

FIGURE 3. Spermatocytic seminoma is composed of 3 sizes of cells: small, medium, and large. Most nuclei are spherical

References

1. Ulbright, T. M. The most common, clinically significant misdiagnoses in testicular tumor pathology, and how to avoid them. Adv Anat Pathol 2008. 15 1:18–27.
2. Spitz, M. R. , J. G. Sider , E. S. Pollack , H. K. Lynch , and G. R. Newell . Incidence and descriptive features of testicular cancer among United States whites, blacks, and Hispanics, 1973–1982. Cancer 1986. 58 8:1785–1790.
3. Wu, X. , F. D. Groves , C. C. McLaughlin , A. Jemal , J. Martin , and V. W. Chen . Cancer incidence patterns among adolescents and young adults in the United States. Cancer Causes Control 2005. 16 3:309–320.
4. McGlynn, K. A. , S. S. Devesa , A. J. Sigurdson , L. M. Brown , L. Tsao , and R. E. Tarone . Trends in the incidence of testicular germ cell tumors in the United States. Cancer 2003. 97 1:63–70.
5. Looijenga, L. H. and J. W. Oosterhuis . Pathogenesis of testicular germ cell tumours. Rev Reprod 1999. 4 2:90–100.
6. Masson, P. Etude sur le siminome. Rev Canad Biol 1946. 5:361–387.
7. Talerman, A. Spermatocytic seminoma: clinicopathological study of 22 cases. Cancer 1980. 45 8:2169–2176.
8. Burke, A. P. and F. K. Mostofi . Spermatocytic seminoma: a clinicopathologic study of 79 cases. J Urol Pathol 1993. 1:21–32.
9. Carrière, P. , P. Baade , and L. Fritschi . Population based incidence and age distribution of spermatocytic seminoma. J Urol 2007. 178:125–128.
10. Stoop, H., R. van Gurp , and R. de Krijger . et al. Reactivity of germ cell maturation stage-specific markers in spermatocytic seminoma: diagnostic and etiological implications. Lab Invest 2001. 81 7:919–928.
11. Gillis, A. J., H. J. Stoop , and R. Hersmus . et al. High-throughput microRNAome analysis in human germ cell tumours. J Pathol 2007. 213 3:319–328.
12. Muller, J. , N. E. Skakkebaek, and M. C. Parkinson . The spermatocytic seminoma: views on pathogenesis. Int J Androl 1987. 10 1:147–156.
13. Kraggerud, S. M., A. Berner, M. Bryne , E. O. Pettersen , and S. D. Fossa . Spermatocytic seminoma as compared to classical seminoma: an immunohistochemical and DNA flow cytometric study. APMIS 1999. 107 3:297–302.
14. Zeeman, A. M. , H. Stoop , and M. Boter . et al. VASA is a specific marker for both normal and malignant human germ cells. Lab Invest 2002. 82 2:159–166.
15. Dekker, I. , T. Rozeboom , J. Delemarre , A. Dam , and J. W. Oosterhuis . Placental-like alkaline phosphatase and DNA flow cytometry in spermatocytic seminoma. Cancer 1992. 69:993–996.
16. Satie, A. P. , E. Rajpert-De Meyts , and G. C. Spagnoli . et al. The cancer-testis gene, NY-ESO-1, is expressed in normal fetal and adult testes and in spermatocytic seminomas and testicular carcinoma in situ. Lab Invest 2002. 82 6:775–780.
17. Cummings, O. W. , T. M. Ulbright , J. N. Eble , and L. M. Roth . Spermatocytic seminoma: an immunohistochemical study. Hum Pathol 1994. 25 1:54–59.
18. Albores-Saavedra, J. , H. Huffman , I. Alvarado-Cabrero , and A. G. Ayala . Anaplastic variant of spermatocytic seminoma. Hum Pathol 1996. 27 7:650–655.
19. Floyd, C. , A. G. Ayala , C. J. Logothetis , and E. G. Silva . Spermatocytic seminoma with associated sarcoma of the testis.Cancer 1988. 61 2:409–414.
20. True, L. D., C. N. Otis , W. Delprado , R. E. Scully , and J. Rosai . Spermatocytic seminoma of testis with sarcomatous transformation: a report of five cases. Am J Surg Pathol 1988. 12 2:75–82.
21. Verdorfer, I., H. Rogatsch, A. Tzankov, H. Steiner, and G. Mikuz . Molecular cytogenetic analysis of human spermatocytic seminomas. J Pathol 2004. 204 3:277–281.