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Ternate and Trine - Spermatocytic Tumour

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Abstract

Spermatocytic Tumour emerges as a polymorphous, tripartite germ cell neoplasm reminiscent of developing spermatogonia, non-concordant to germ cell neoplasia in situ and demonstrates favorable prognostic outcomes. Tumefaction demonstrates aneuploidy and tumour cells characteristically lack anomalies within chromosome 12p. Cytological examination exhibits a cellular neoplastic component of variable magnitude as miniature cells, predominant intermediate cells and enlarged cells. Upon microscopy, pathognomonic tripartite cellular component is constituted of cells of variable magnitude described as miniature cells, intermediate cells and giant cells. Tumour configures nodules, cord-like pattern or peripheral inter-tubular cells enmeshed within a fibrinous perimeter.

Keywords: Spermatocytic; Tripartite; Aneuploid

Abbreviations: PLAP: Placental Alkaline Phosphatase; FGRF3: Fibroblast Growth Factor Receptor 3; GCT: Germ Cell Tumour; GCNIS: Germ Cell Neoplasia in Situ.

Introduction

Spermatocytic tumour emerges as a polymorphous, tripartite germ cell neoplasm reminiscent of developing spermatogonia and associated with favorable prognostic outcomes. Neoplasm is non-concordant to germ cell neoplasia in situ. Tumefaction is commonly observed in elderly subjects, in contrast to diverse germ cell tumours. Tripartite tumour cells of variable magnitude classically configure as miniature cells, intermediate cells and giant cells. Tumour cells appear immune reactive to CD117 or SALL4 and immune non-reactive to placental alkaline phosphatase (PLAP), OCT3/4 or CD30.

Additionally designated as spermatocytic seminoma, spermatocytoma, spermatocytic variant of classic seminoma or type III germ cell tumour, spermatocytic tumour configures~1% of germ cell tumours. In contrast to germ cell neoplasia in situ derived tumours, tumefaction implicates elderly males or young subjects and demonstrates mean age of disease emergence at 52 years [1,2]. Spermatocytic tumour may occur within a comprehensive age of 19 years to 92 years. Nearly 30% individuals between 30 years to 39 years, ~35% individuals between 40 years to 59 years and ~35% subjects ≥ 60 years are implicated. A specific ethnic predisposition is absent [1,2].

Spermatocytic tumour preponderantly incriminates testis wherein 9% neoplasms are bilateral, synchronous and frequently metachronous. Primitive or extra-gonadal neoplasms appear undocumented. However, ovarian spermatocytic tumour-like lesion may concur with gonadal mixed germ cell tumour.

Neoplasm is derived from post-pubertal subtype of germ cells as spermatogonium or spermatocyte. Chromosomal anomalies are predominant and proportionate genetic mutations may amplify with increasing age.

Although cryptorchidism or germ cell neoplasia in situ may not contribute to neoplastic emergence, spermatocytic tumour arising within an undescended testis is documented [2,3].

Upon molecular assay, spermatocytic tumour demonstrates aneuploidy. Typically, tumour cells lack anomalies within chromosome 12p. Amplification of chromosome 12p appears concordant with neoplasms delineating cytological anaplasia or emergence of distant metastases. Genomic amplification of chromosome 9p is frequently discerned with consequent amplification of DMRT1 genetic locus. Gains within chromosome 1 and chromosome 20 with partial loss of chromosome 22 may ensue. Although genetic mutations are infrequent, tumours arising within elderly population may display activating mutations within HRAS gene and FGFR3 gene. Decimation of biparental genomic imprinting with restoration of paternal imprinting may be discerned.

Upon clinical examination, a painless testicular nodule is exemplified. Focal sarcomatous differentiation is associated with rapid tumour progression. Cytological smears are significantly cellular and preponderantly composed of singular, disseminated cells. Cellular neoplastic component exemplifies variable magnitude as miniature cells, intermediate cells and enlarged cells wherein intermediate cells are preponderantly observed. In contrast to seminoma, tigroid zone delineating foamy substances configured of fragile, glycogen rich cytoplasm is absent. Intervening lymphocytic infiltrate is absent. Upon frozen section examination, tumour is categorically ascertained by trimorphic cellular component of miniature, intermediate and giant cells. Focal intra-tubular spermatocytic tumour dispersed within circumscribing parenchyma may be misinterpreted as germ cell neoplasia in situ whereas specific subtypes of germ cell neoplasia appear absent [3,4].

Grossly, a soft, oedematous or mucoid, grey/white, homogenous tumefaction or lobulated nodule of magnitude varying from 1.4 centimeters to 28 centimeters and mean tumour dimension of 7 centimeters is encountered. Cut surface exhibits foci of necrosis and hemorrhage. Tumefaction may extend beyond tunica albuginea. Additionally, fleshy and firm tumour zones along with or devoid of necrosis may represent foci of sarcomatous differentiation. Upon low power microscopic examination, the homogenous neoplasm is constituted of diffuse sheets of tumour cells lacking segregation by fibrous tissue septa or infiltration with lymphocytes. Multinodular lesion exhibits bands of loosely cohesive, fibro-vascular tissue demarcating tumour nodules. Intervening fibrous tissue septa may or may not be infiltrated by lymphocytes. Remnants of testicular parenchyma between tumour nodules appear absent. Tumour parenchyma exemplifies spaces of varying outlines imbued with clear or pinkish fluid. Fluid filled spaces represent as ~irregular pseudo-cystic areas of variable diameter ~pseudo-follicular arrangement comprised of spherical, oedematous areas encompassed by amalgamated neoplastic cells ~micro-cystic spaces which demarcate tumour parenchyma into anastomosing islands accompanied with peripheral palisading of cells. Neoplasm is expansive although cord-like pattern with inter-tubular tumour cells may appear upon periphery of lesion. Neoplastic nodules may be enmeshed within a fibrinous perimeter. Tumefaction is preponderantly confined to testicular parenchyma although rete testis, epididymis or tunica albuginea may be incriminated. Juxtaposed non-neoplastic testicular parenchyma may enunciate mild atrophy. High power examination expounds a pathognomonic tripartite cellular component constituted of cells of variable magnitude described as ~miniature cells simulating small lymphocytes pervaded with spherical, intensely staining nuclei, narrow perimeter of eosinophilic cytoplasm and magnitude between 6µ to 8µ. ~intermediate cells permeated with spherical nuclei, granular to filamentous nuclear 'spireme-like chromatin' simulating nuclear chromatin upon commencement of prophase, inconspicuous nucleoli, dense eosinophilic to amphophilic cytoplasm, absent intracytoplasmic glycogen and inadequately defined cytoplasmic membrane with dimension between 15µ to 20µ. Besides, clear cells with distinct cellular membranes may about the oedematous areas. ~giant cells incorporated with singular or multiple nuclei, eosinophilic cytoplasm and morphological features simulating intermediate cells and dimension between 50µ to 150µ.

Variable magnitude of tumour cells expounds a tumefaction composed of medium cells > miniature cells > giant cells in decreasing order of frequency [4,5]. Anabundance of mitotic figures, atypical mitosis and apoptotic bodies is observed. Intervening stroma is scanty and miniature strips of connective tissue may be discerned. Infiltration with lymphocytes within tumour parenchyma or intervening strips of connective tissue is minimal or absent. Epithelioid cell granulomas are exceptionally encountered. Intra-tubular expansion of spermatocytic tumour appears to circumscribe tumour cell aggregates. Nevertheless, a component of germ cell neoplasia in situ is absent. Foci of intravascular invasion may emerge. Tumefaction appears non concurrent with diverse categories of germ cell tumour . As per contemporary World Health Organization (WHO) classification, spermatocytic tumour with sarcomatous differentiation is a distinct, exceptional entity associated with inferior prognostic outcomes. The high grade, sarcomatous component articulates as spindle shaped cells, wherein segregation from rhabdomyosarcoma or chondrosarcoma may be challenging Of variable proportion, sarcoma cells

may intermingle with or appear adjacent to spermatocytic tumour cells. Notwithstanding, lesions with minimal residual component of spermatocytic tumour may be challenging to ascertain. As per contemporary World Health Organization (WHO) classification, anaplastic spermatocytic tumour may not represent a distinct subtype of spermatocytic tumour and neoplasm lacks aggressive biological behaviour. Characteristically, tumefaction is constituted of monomorphic, intermediate cells pervaded with prominent nucleoli. Distinctive foci of conventional spermatocytic tumour may be discerned. Upon ultrastructural examination, tumour cells appear adhered with intercellular bridges reminiscent of bridges between spermatocytes and spermatids. Foci of syncytial articulations may occur. Intra-cytoplasmic glycogen and lipids appear minimal or absent [5,6] (Figures 1 & 2).

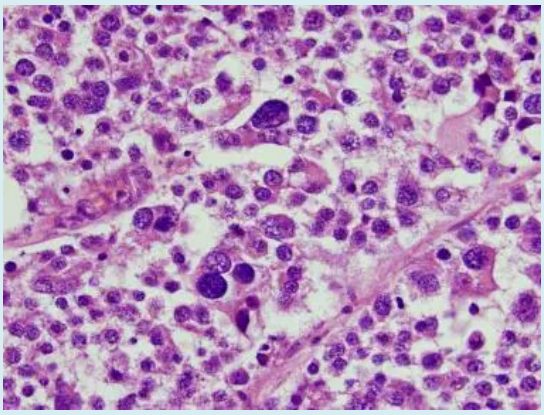

Figure 1: Spermatocytic tumour composed of tripartite cells as small, intermediate and giant cells traversed by fibrous tissue septa [7].

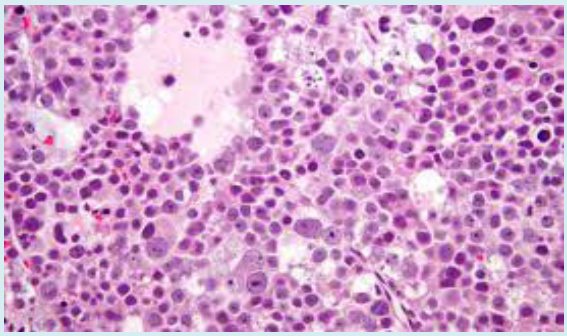

Figure 2: Spermatocytic tumour constituted of triphasic cellular component as small, intermediate and giant cells intermingled with fluid filled spaces and traversed by fibrous tissue septa [8].

	Spermatocytic Tumour	Seminoma
CD117/cKit	+	+
D2-40/ podoplanin	-	+
OCT3/4	-	+
PLAP	-	+
FGRF3	+	-
HRAS	+	-
DMRT1	+	-

Table 1: Immunohistochemistry of Spermatocytic Tumour and Seminoma [2].
PLAP: Placental Alkaline Phosphatase, FGRF3: Fibroblast Growth Factor Receptor 3.

	Spermatocytic Tumour	Seminoma
Median Age	54 years	35 years
Frequency of GCT	<1%	60%
Presence of GCNIS	Absent	Present
Extra-gonadal site(%)	Absent	2% to 5%
Isochromosome 12p expression	Absent	Present
Sarcomatous component	5% to 8%	Absent
Bilateral lesions (synchronous/ metachronous)	8% to 10%	5%
Stromal inflammatory reaction	Absent	Present
Associated crypto-orchidism	Absent	Present
Overall 5 year survival clinical stage I(%)	95	99
Overall 5 year survival advanced disease (%)	Poor	72-86

Table 2: Clinical and Pathological Features of Spermatocytic Tumour and Seminoma [2].
GCT: Germ Cell Tumour, GCNIS: Germ Cell Neoplasia in Situ.

Discussion

Tumour cells appear immune reactive to CD117, SALL4, DMRT1, NUT, OCT2 and SSX2. Neoplastic cells appear immune non-reactive to epithelial membrane antigen(EMA), placental alkaline phosphatase(PLAP), OCT3/4, beta human chorionic gonadotrophin(βHCG), alfa fetoprotein(AFP), carcinoembryonic antigen (CEA), CD30, leucocyte common antigen (LCA) or CD45 and cytokeratin AE1/AE3. Tumour cells may not be highlighted with staining by Periodic acid Schiff's stain [5,6]. Spermatocytic tumour requires segregation from neoplasms such as testicular diffuse large B cell lymphoma(DLBCL), seminoma, germ cell neoplasia in situ, embryonal carcinoma or yolk sac tumour. Cogent histological examination appears appropriate for ascertaining spermatocytic tumour. Tumefaction is devoid of elevated levels of serum lactate dehydrogenase (LDH), alpha fetoprotein (AFP) or beta human chorionic gonadotropin (βHCG) [5,6]. Upon imaging, tumefaction appears well circumscribed and predominantly solid with multiple cystic areas. The heterogeneous signal intensity is comprised of commingled hyperechoic and hypoechoic components [5,6]. Spermatocytic tumour may be appropriately eradicated by surgical manoeuvers as orchiectomy [5,6]. Additionally, chemotherapeutic agents as bleomycin, etoposide and cisplatin (BEP) may be minimally efficacious in treating neoplasms devoid of sarcomatous differentiation or demonstrating distant metastasis [5,6]. Generally, spermatocyctic tumour is associated with superior prognostic outcomes. Conversely, spermatocytic tumour with sarcomatous differentiation delineates inferior prognostic outcomes, significant proportionate distant metastasis of sarcomatous component into pulmonary parenchyma and considerable tumour associated mortality. Spermatocytic tumour devoid of sarcomatous differentiation may exceptionally disseminate into sites as retroperitoneal lymph nodes, pulmonary parenchyma or brain [5,6]. Infiltration of intravascular spaces or tumour dissemination beyond testicular parenchyma of neoplasms devoid of sarcomatous differentiation or accompanying distant metastasis is documented. spermatocytic tumour appears non concordant with inferior prognostic outcomes [5,6].

Conclusion

Spermatocytic tumour is immune reactive to CD117, SALL4, DMRT1, NUT, OCT2 and SSX2. Neoplastic cells appear immune non-reactive to epithelial membrane antigen(EMA), placental alkaline phosphatase(PLAP), OCT3/4, beta human chorionic gonadotrophin(βHCG), alfa fetoprotein(AFP), carcinoembryonic antigen (CEA), CD30, leucocyte common antigen (LCA) or CD45 and cytokeratin AE1/AE3. Spermatocytic tumour requires segregation from neoplasms such as testicular diffuse large B cell lymphoma (DLBCL), seminoma, germ cell neoplasia in situ, embryonal carcinoma or yolk sac tumour. Tumefaction appears well circumscribed, predominantly solid with multiple cystic areas wherein heterogeneous signal intensity is comprised of commingled hyperechoic and hypoechoic components. Spermatocytic tumour is appropriately eradicated by surgical manoeuvers as orchiectomy. Neoplasm is associated with superior prognostic outcomes.

References

1. Dias AF, Dvindenko E, Santos F, Cabrera R (2023) Sarcomatoid Spermatocytic Tumour: Report of a Rare Case and Literature Review. Int J Surg Pathol 31(5): 728-733.

2. Secondino S, Viglio A, Neri G, Galli G, Faverio C, et al. (2023) Spermatocytic Tumor: A Review. Int J Mol Sci 24(11): 9529.

3. Petersen CL, Otto PO, Kjaer-Frifeldt S, Pederson MRV (2023) Spermatocytic tumors in 2 patients aged 50 and 77 years: 2 case reports and brief review of the literature. Radiol Case Rep 18(10): 3572-3576.

4. Bapir R, Aghaways I, Ali RM, Fakhralddin SS, Ali RM, et al. (2023) Spermatocytic tumor of the testis: A case report and mini review of the literature. Med Int (Lond) 3(5): 51.

5. Rabade K, Panjwani PK, Menon S, Praksh G, Pal M, et al. (2022) Spermatocytic tumor of testis: A case series of 26 cases elucidating unusual patterns with diagnostic and treatment dilemmas. Journal of Cancer Research and Therapeutics 18(2): S449-S454.

6. Kim JH, Page RD, Moses GA, Columbres RC (2023) Exceedingly Rare Bilateral Synchronous Germ Cell Testicular Tumors with Different Histopathological Features. Cureus 15(7): e42374.