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Twine and String-Sertoli Cell Tumour – Testis

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Abtract

Sertoli cell tumour represents as a pure sex cord stromal tumour preponderantly or focally composed of foetal, pre-pubertal, adult, or atrophic Sertoli cells. Sertoli cell tumour exemplifies CTNNB1 genetic mutation. Factors contributing to emergence of Sertoli cell syndrome appear as cryptorchidism or familial adenomatous polyposis. Cytological smears display tumour cells as uniform, miniature, ovoid or plasmacytoid which are pervaded with nuclei demonstrating granular chromatin and smooth cellular outline. Upon microscopy, Sertoli cell tumour exhibits specific architectural patterns as tubular structures impregnated with lumen, cellular cords devoid of lumen or foci of tubulo-papillary articulations. Additionally, neoplastic patterns as macro-cystic, micro-cystic, tumour cell nests, trabecular, whorled, solid areas, retiform or pseudo-papillary configurations may be encountered.

Keywords: Sex cord stromal tumour; foetal; pre-pubertal; adult or atrophic Sertoli cells.

Abbreviations: SF1: Steroidogenic Factor 1; WT1: Wilm's Tumour 1; PLAP: Placental Alkaline Phosphatase; AFP: Alpha Fetoprotein; HCG: Human Chorionic Gonadotrophin; CT: Computerized Tomography; βHCG: Beta Human Chorionic Gonadotropin; AFP: Alpha Fetoprotein; LDH: Lactate Dehydrogenase.

Introduction

Sertoli cell tumour represents as a pure sex cord stromal tumour incriminating testicular parenchyma. Tumefaction is preponderantly or focally composed of cells delineating features of foetal, pre-pubertal, adult, or atrophic Sertoli cells. Following Leydig cell tumour in frequency, the sex cord stromal Sertoli cell tumour configures as a miniature, unilateral neoplasm. Adults are commonly incriminated. Tumefaction is predominantly benign. Characteristically, neoplasm delineates a focal tubular or cord-like cellular pattern. Neoplastic cells appear immune reactive to steroidogenic factor 1 (SF1) and nuclear β catenin. Variable, inconsistent immune reactivity to calretinin, inhibin A, androgen receptor or CD56 is encountered. Tumour cells appear immune non-reactive to SALL4, OCT4 or diverse germ cell markers. Neoplasms associated with distant metastasis demonstrate inadequate response to therapy.

Previously designated as Andro blastoma, Sertoli cell tumour represents < 1% of testicular neoplasms. Tumefaction is exceptionally discerned < 20 years wherein mean age of disease emergence is 45 years [1,2].

Sertoli cell tumour exemplifies CTNNB1 genetic mutation. Tumour cells appear to lack isochromosome 12p. Nearly 40% instances manifest gain of X chromosome. Besides, loss of chromosomes 2 and 19 may be expounded [1,2]. Factors contributing to emergence of Sertoli cell syndrome appear as cryptorchidism or familial adenomatous polyposis. Specific environmental factors contributing to disease occurrence appear absent. Sertoli cell tumour emerges as a gradually progressive, unilateral testicular tumefaction. Clinical symptoms as testicular discomfort or pain may occur [1,2]. Notwithstanding, neoplasm may be discovered as an incidental finding. Hormonal manifestations are extremely exceptional and concur with malignant neoplasms. Infrequently, distant metastasis occurs as an initial disease manifestation [2,3]. Cytological smears obtained from malignant Sertoli cell tumour associated with distant metastasis enunciate branching tubules, micro-acinar structures or papillary groups intermingled with innumerable individual cells. Tumour cells appear uniform, miniature, ovoid or plasmacytoid and are pervaded with nuclei demonstrating granular chromatin and smooth cellular outline [2,3]. Foci of malignant metamorphosis exhibit discordant, enlarged nuclei with nuclear overlapping and significant mitotic activity. Cytological aspirate simulates the cellular content of neuroendocrine tumour or well differentiated adenocarcinoma [2,3].

Grossly, benign Sertoli cell tumour manifests as a well circumscribed neoplasm with magnitude varying from 2 centimetres to 5 centimetres. Malignant Sertoli cell tumour exceeds > 5centimetre diameter and appears inadequately circumscribed with foci of extra-testicular extension, necrosis, or haemorrhage. Nearly 30% neoplasms represent with a cystic component. Cut surface appears homogenous, whitish, or yellow [3,4]. Upon microscopy, Sertoli cell tumour exhibits specific architectural patterns as tubular structures impregnated with lumen, cellular cords devoid of lumen or foci of tubulo-papillary articulations. Additionally, neoplastic patterns as macro-cystic, micro-cystic, tumour cell nests, trabecular, whorled, solid areas, retiform or pseudo-papillary configurations may be encountered. Nevertheless, majority of neoplasms expound a mixed tumour pattern [3,4].

Tumour cells are permeated with clear cytoplasm varying from minimal, foamy, eosinophilic, abundant, or markedly lipid laden. Hyaline globules are commonly discerned. Benign neoplasms depict bland, uniform, spherical to ovoid tumour cell nuclei. In contrast, malignant neoplasms enunciate miniature, hyperchromatic nuclei or enlarged nuclei pervaded with prominent nucleoli [3,4]. Intervening stroma is quantifiably variable and expounds basement membrane-like material circumscribing neoplastic tubules. Additionally, sclerotic, myxoid, oedematous or angiomatous stroma permeated with innumerable vascular articulations may be expounded [3,4]. Neoplasm depicting stromal sclerosis > 50% is designated as sclerotic variant and is currently contemplated as a subtype of Sertoli cell tumour, NOS. Inflammatory cell exudate confined to the stroma is absent. Exceptionally, a prominent stromal inflammatory cell exudate is exemplified.

Ultrastructural examination exhibits prominent Golgi complex, variable smooth endoplasmic reticulum, lipid droplets and intracellular glycogen. Tumour cells are interconnected with desmosomes and peripheral basement membrane [3,4]. Figure 1,2, Table 1.

Table 1: World Health Organization of Testicular Tumours (4).

Sex cord/Stromal Tumours
Leydig cell tumour
Malignant Leydig cell tumour
Sertoli cell tumour
Malignant Sertoli cell tumour
Large cell calcifying Sertoli cell tumour
Intra-tubular large cell hyalinising Sertoli cell neoplasia
Granulosa cell tumour
Adult type
Juvenile type
Thecoma/fibroma group of tumours
Other sex cord gonadal/stromal tumours
Mixed
Unclassified
Tumours containing germ cell & sex cord/gonadal stromal component
Gonadoblastoma
Miscellaneous nonspecific stromal cell tumours
Ovarian epithelial tumours
Tumours of collecting ducts and rete testis
Adenoma
Carcinoma
Tumours of paratesticular structures
Adenomatoid tumour
Mesothelioma(epithelioid/biphasic)
Epididymal tumours
Cystadenoma of epididymus
Papillary cystadenoma
Adenocarcinoma of the epididymis
Mesenchymal tumours of spermatic cord and testicular adnexa

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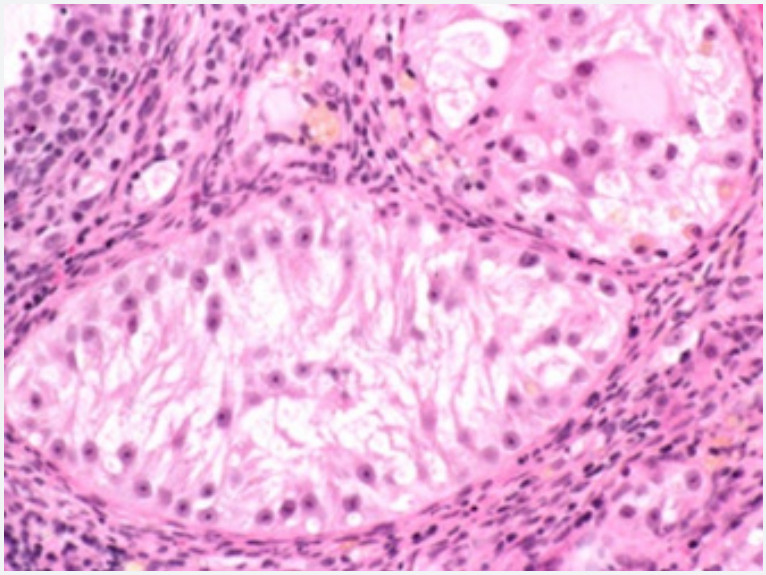

Figure 1: Sertoli cell tumour delineating tubules layered by Sertoli cells imbued with clear to eosinophilic, abundant cytoplasm, bland, uniform nuclei, and a well-defined basement membrane. Intervening stroma is fibrotic and variably infiltrated by chronic inflammatory cells [8].

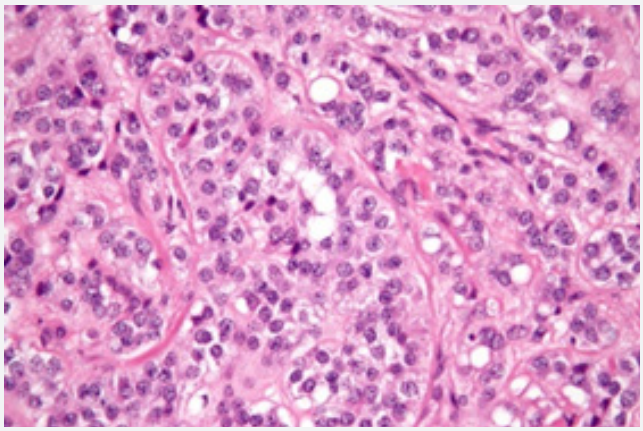

Figure 2: Sertoli cell tumour exhibiting tubules and cords of Sertoli cells permeated with abundant eosinophilic cytoplasm and bland, uniform nuclei situated upon a well demarcated basement membrane. Intervening stroma is fibrotic and variably infiltrated by chronic inflammatory cells [9].

Sertoli cell tumour appears immune reactive to steroidogenic factor 1(SF1) or nuclear β catenin. Inconsistent immune reactivity to calretinin, inhibin A, cytokeratin, MelanA, Wilm's tumour 1(WT1), CD99, SOX9, vimentin, CD56, chromogranin, synaptophysin, nesting, PAX2, PAX8 or androgen receptor is observed. Tumour cells appear immune non-reactive to SALL4, OCT4, placental alkaline phosphatase (PLAP), alpha fetoprotein (AFP), human chorionic gonadotrophin (HCG) or CD30 [4,5]. Testicular Sertoli cell tumour requires segregation from neoplasms as rete testis adenoma, adenomatoid tumour, Leydig cell tumour, yolk sac tumour, seminoma, juvenile granulosa cell tumour, large cell calcifying Sertoli cell tumour or Sertoli cell nodule (Pick adenoma) [4,5]. Ultrasonography of the scrotum or testicular tumefaction is optimally recommended. Subsequently, referral to a urologist for additional assessment and cogent therapeutic management is appropriate and beneficial [6,7].

Male subjects between 15 years to 44 years delineating a retroperitoneal neoplasm or foci of distant metastases necessitate physical examination of testis and scrotal ultrasonography. Ultrasonography exhibits a solitary, hypoechoic lesion, an imaging feature which is recapitulated by several germ cell tumours. Testicular surgical sampling requires circumvention to inhibit seeding of tumour cells [6,7]. Cogent tumour staging is achieved with~imaging procedures as computerized tomography (CT) of abdomen or pelvis along with intravenous contrast infused radiography of chest which may be performed prior or after

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orchiectomy. Anomalies discerned within chest radiographs may be further evaluated with non-contrast computerized tomography of chest [6,7].

Evaluation of pertinent serum hormonal markers is recommended. Neoplasm expounds normal serum levels of tumour markers as beta human chorionic gonadotropin (βHCG) or alpha fetoprotein (AFP), values which exclude the emergence of nonvenomous germ cell tumours. Serum lactate dehydrogenase (LDH) levels appear optimal in assessing global tumour burden [6,7]. Surgical manoeuvres as total or partial orchiectomy are a recommended mode of therapy for managing Sertoli cell tumour. Additionally, retroperitoneal lymph node dissection may be suitably adopted for treating malignant Sertoli cell tumour.

Adjuvant chemotherapy or radiotherapy appears nonspecific and is associated with inferior response [6,7]. Sertoli cell tumour is preponderantly benign and ~ 5% neoplasms appear as malignant. Tumefaction depicting stromal sclerosis > 50% are pre-eminently benign [6,7]. Morphological features associated with malignant metamorphosis emerge as extra-testicular tumour dissemination, tumour magnitude > 5 centimetres, lymphatic and vascular invasion, significant cytological atypia, tumour necrosis or mitotic index exceeding > 5 mitosis per10 high power fields. Distant metastasis may exceptionally ensue within neoplasms devoid of morphological features concurrent with malignant transformation [6,7].

Conclusion

Sertoli cell tumour appears immune reactive to steroidogenic factor 1(SF1) or nuclear β catenin. Inconsistent immune reactivity to calretinin, inhibin A, cytokeratin, MelanA, Wilm's tumour 1(WT1), CD99, SOX9, vimentin, CD56, chromogranin, synaptophysin, nestin, PAX2, PAX8 or androgen receptor is observed. Tumour cells appear immune nonreactive to SALL4, OCT4, placental alkaline phosphatase (PLAP), alpha fetoprotein (AFP), human chorionic gonadotrophin (HCG) or CD30. Testicular Sertoli cell tumour requires segregation from neoplasms as rete testis adenoma, adenomatoid tumour, Leydig cell tumour, yolk sac tumour, seminoma, juvenile granulosa cell tumour, large cell calcifying Sertoli cell tumour or Sertoli cell nodule (Pick adenoma). Serum lactate dehydrogenase (LDH) levels appear optimal in assessing global tumour burden. Surgical manoeuvers as total or partial orchiectomy are a recommended mode of therapy for managing Sertoli cell tumour.

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