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Editorial

Ebb and Lapse-Regressed Germ Cell Tumour-Testis

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Testicular germ cell tumour representing with spontaneous, comprehensive or partial neoplastic retrogression configures as regressed testicular germ cell tumour. Tumefaction manifests with distant metastasis, residual testicular germ cell tumour or a dual component comprising of residual neoplasm and distant metastasis. Additionally designated as regressed germ cell tumour, burned out germ cell tumour or burnt out germ cell tumour, neoplasm is commonly associated with pure seminoma delineating distant metastasis and residual testicular tumour. Morphological composition of testicular tumour regression is comprised of fibrous scar and germ cell neoplasia *in situ*.

Testicular regressed germ cell tumour enunciates ~3% of testicular tumours. Median age of disease discernment appears between 28 years to 32 years wherein the neoplasm is commonly encountered between 17 years to 67 years. Nearly 10% of retroperitoneal germ cell tumours demonstrate retrogression of a testicular primary neoplasm [1,2].

Testicular regressed germ cell tumour is frequently associated with distant metastasis to the retroperitoneum. Besides, metastasis into hepatic parenchyma, pulmonary parenchyma, bone and brain is documented [1,2].

Of obscure aetiology and pathogenesis, immune modulated or ischaemic retrogression of tumour tissue is posited to contribute to disease emergence [1,2].

Fluorescent *in situ* hybridization (FISH) may display isochromosome 12p, a feature which appears pathognomonic of testicular germ cell tumour with distant metastasis. Aforesaid chromosomal anomaly may commonly be discerned with cogent immunohistochemistry [2,3].

Testicular regressed germ cell tumour associated with symptoms of distant metastasis emerges as a frequently discerned initial disease representation. Alternatively, an extra-gonadal tumefaction, abdominal pain, dorso-lumbar pain, regional lymphadenopathy, loss of weight and pyrexia may ensue. Infrequently, lesion may be associated with testicular pain, testicular tumefaction, elevated serum levels of tumour markers as alpha fetoprotein (AFP), beta human chorionic gonadotrophin (β hCG) and lactate dehydrogenase (LDH) [2,3].

Testicular regressed germ cell tumour commonly represents as metastatic disease devoid of palpable testicular tumefaction [2,3].

Grossly, tumefaction represents as a fibrous scar which replaces normal testicular parenchyma. Neoplastic scar configures as a stellate lesion or well demarcated focus. Besides, nodular tissue with whitish to tan hue is enunciated [3,4].

Upon microscopy, intratesticular fibrous scar expounds acellular zones of collagenous tissue commingled with innumerable, miniature, disseminated vascular articulations. Pure seminoma is a commonly discerned neoplasm associated with retrogression [3,4].

A population of foamy or hemosiderin laden macrophages appear intermixed with the cellular component. Intra-tumour chronic inflammatory infiltrate of lymphoid cells and plasma cells is significant and discernible [3,4].

Foci of tumour necrosis or intra-tubular calcification configuring microlithiasis are exemplified. Incomplete tumour regression is associated with residual germ cell tumour [4,5].

An estimated 50% lesions delineate germ cell neoplasia *in situ* confined to testicular parenchyma. Besides, testicular parenchyma may be atrophic and expounds shrinking of seminiferous tubules in concurrence with decimated spermatogenesis. Testicular tubules may singularly be layered by Sertoli cells. Circumscribing peritubular basement membrane appears thick and dense [4,5].

Germ cell tumours derived from germ cell neoplasia *in situ*
Non invasive lesions as germ cell neoplasia *in situ*/gonadoblastoma
Germinoma
Seminoma, pure
Seminoma with syncitiotrophoblastic cells
Non seminomatous germ cell tumour, pure
Embryonal carcinoma
Yolk sac tumour, postpubertal type
Trophoblastic tumours, choriocarcinoma
Teratoma, postpubertal or teratoma with somatic type transformation
Non seminomatous mixed germ cell tumours
Regressed germ cell tumour
Germ cell tumours unrelated to germ cell neoplasia *in situ*
Spermatocytic tumour
Prepubertal (paediatric) tumours
Teratoma, prepubertal type
Dermoid cyst
Epidermoid cyst
Yolk sac tumour, prepubertal type
Prepubertal type testicular neuroendocrine tumour
Mixed prepubertal type tumours

Table: *World health organization of testicular germ cell tumours [4].*

Neoplasms as germ cell neoplasia *in situ* or seminoma appear immune reactive to SALL4, OCT3/4, CD117 (c-KIT), placental alkaline phosphatase (PLAP) or podoplanin (D2-40).

Embryonal carcinoma appears immune reactive to SALL4, cytokeratin AE1/AE3, CD30, OCT3/4 and variably immune reactive to placental alkaline phosphatase (PLAP). Yolk sac tumour appears immune reactive to SALL4, cytokeratin AE1/AE3, glypican3 and alpha fetoprotein (AFP).

Choriocarcinoma appears immune reactive to SALL4, cytokeratin AE1/AE3, CK7, human chorionic gonadotropin (HCG) and glypican 3 [6,7].

Germ cell neoplasia *in situ* or seminoma appears immune non reactive to cytokeratin AE1/AE3, CK7, glypican 3, human chorionic gonadotropin (HCG) and alpha fetoprotein (AFP) [6,7].

Regressed germ cell tumours of testis require segregation from conditions as testicular atrophy, non-neoplastic scar or therapeutic effect contingent to preceding history of chemotherapy employed for alleviating germ cell tumour [6,7].

Upon ultrasonography a distinct testicular abnormality is observed. Definitive extra-gonadal germ cell tumour is identified.

Ultrasonography may depict manifestations of tumour retrogression. The lesion is associated with macrolithiasis wherein testicular stones > 0.2 centimetre magnitude or microlithiasis wherein stones < 0.2 centimetre magnitude may be exemplified. Lesion appears devoid of testicular homogeneity.

Alternatively, nonspecific hyperechoic or hypoechoic lesions may be detected. Testicular atrophy may be observed [6,7].

Upon computerized tomography (CT), features of distant metastasis may be enunciated. Regional lymphadenopathy or an extra-gonadal tumefaction may be observed.

Histopathological features of tumour retrogression may be detected within the testis [6,7].

Metastasis from testicular regressed germ cell tumour may be suitably managed with preponderant administration of combination systemic chemotherapy. Besides, seminoma may be treated with chemotherapy combined with radiotherapy. Upon ultrasonography, tumours associated with anomalous morphological features may be subjected to radical orchiectomy [8,9].

Five year proportionate disease free survival following therapy appears at ~29% whereas 10 years survival emerges at ~12%.

Prognostic outcomes of complete retrogression or partial retrogression of the neoplasm appear identical [8,9].

Contingent to regressed germ cell tumour testis associated with distant metastasis, pure seminoma demonstrates a superior prognosis. However, prognostic outcomes of regressed and non regressed testicular germ cell tumours remain undocumented [8,9].

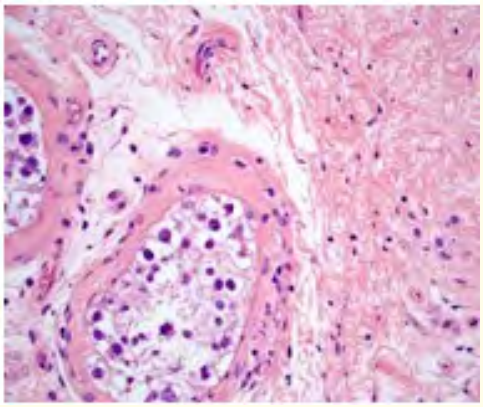

Figure 1: *Regressed germ cell tumour demonstrating a fibrous scar invaded by chronic inflammatory infiltrate composed of lymphocytes and plasma cells. Surrounding seminiferous tubules depict germ cell neoplasia in situ [10].*

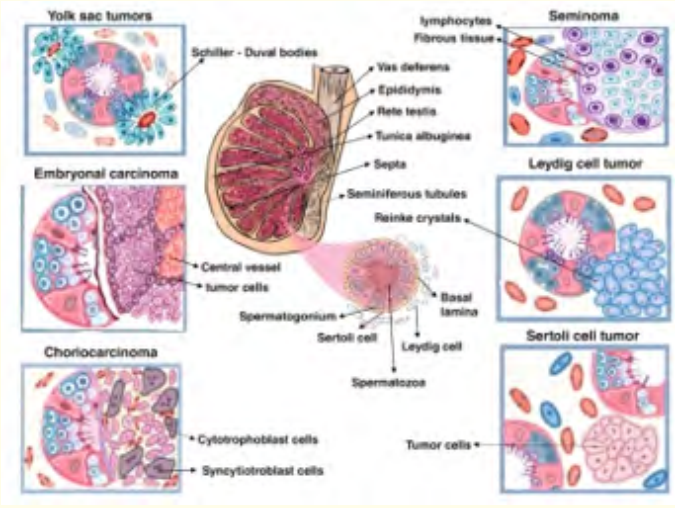

Figure 2: *Regressed germ cell tumour testis delineating varieties of testicular neoplasms with subsequent retrogression [11].*

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10. Image 1 Courtesy: Pathology outlines.

11. Image 2 Courtesy: Springer link.com.

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