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Editorial

Substantial and Jutting-Well Differentiated Papillary Mesothelial Tumour Testis

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Well differentiated papillary mesothelial tumour of testis emerges as a neoplasm which is engendered from mesothelium of the pleura, peritoneum or tunica vaginalis. The tumefaction is pre-eminently comprised of papillary configurations and tubules layered by singular layer of bland cuboidal epithelial cells. Neoplasm lacks distinct invasion into subjacent stroma or circumscribing testicular parenchyma.

Tumour cells appear immune reactive to mesothelial markers as calretinin, Wilm's tumour antigen 1 (WT1), BAP1 or MTAP.

Additionally designated as well differentiated papillary mesothelioma, the mesothelial neoplasm is devoid of significant cellular and nuclear atypia and demonstrates an uncertain malignant potential.

Well differentiated papillary mesothelial tumour arising from tunica vaginalis is exceptionally encountered and configures < 1% of mesotheliomas. Generally, the neoplasm is discerned between 18 years to 70 years [1,2].

Of obscure aetiology and pathogenesis, testicular well differentiated papillary mesothelial tumour appears confined to the peritoneum, tunica vaginalis, pleura or pericardium [1,2].

Well differentiated papillary mesothelial tumour of testis may depict CDC42 missense genetic mutations or TRAF7 missense genomic mutations situated upon C terminus of the protein. Besides, repetitive missense mutations within EHD1, ATM, FBXO10, SH2D2A, CDH5, MAGED1 and TP73 genes may be exemplified [2,3].

Clinically, tumours confined to pleura are frequently symptomatic and accompanied by pleural effusion. Lesions confined to peritoneal surfaces are characteristically and incidentally detected during surgical intervention. Neoplasms confined to tunica vaginalis demonstrate symptoms as testicular pain and appear concurrent with hydrocele [2,3].

Cytological examination depicts clusters of uniform mesothelial cells lacking cellular and nuclear atypia [2,3].

Upon frozen section, tumefaction may be incidentally discovered during certain investigative procedures. Frozen section expounds expansive papillary articulations demonstrating oedema or myxoid change. The papillary configurations are coated by singular layer of bland mesothelial cells. Morphological exclusion of neoplastic invasion is necessitated [3,4].

Grossly, peritoneal surface demonstrates a sprinkling of miniature nodules or singular to innumerable papillary excrescences [3,4].

Upon microscopy, papillary articulations appear imbued with an expansible centric core which may appear as myxoid. The papillary configurations are layered by singular coat of uniform, flattened to cuboidal epithelial cells. Adjacent fibro-vascular core appears devoid of inflammatory exudate or psammoma bodies. Occasionally, gland-like or tubulocystic zones may be encountered [4,5].

The neoplasm depicts a pathognomonic lack of stromal invasion or significant cellular and nuclear atypia. Mitotic figures are absent [4,5].

TNM staging of carcinoma testis [3,4]

Primary tumour

- TX primary tumour cannot be assessed.
- Tis germ cell neoplasia *in situ* (GCNIS).
- T0 no evidence of primary tumour within the testis.
- T1 primary tumour confined to testis and rete testis. Vascular or lymphatic infiltration is absent. Tunica albuginea is invaded. Tumour invasion into tunica vaginalis is absent. Pure seminoma is subdivided as:
- T1a tumour magnitude < 3 centimetres.
- T1b tumour magnitude ≥ 3 centimetres.
- T2 tumour confined to testis, rete testis and extends into ≥ one components of testis as blood vessels, lymphatics, epididymis, adipose tissue confined to hilar soft tissue adjacent to epididymis or tunica vaginalis.
- T3 tumour extends into spermatic cord.
- T4 tumour extends into scrotum.

Regional lymph nodes

Clinical staging of regional lymph nodes is assessed with imaging techniques as computerized tomography (cN).

Pathological staging of regional lymph nodes is assessed with dissection of regional, retroperitoneal, para-aortic, peri-aortic, inter-aortocaval, paracaval, pre-aortic, precaval, retro-aortic and retrocaval lymph nodes (pN).

- NX regional lymph nodes cannot be assessed.
- N0 regional lymph node metastasis absent.
- N1 regional lymph node metastasis confined to one to five retroperitoneal lymph nodes with magnitude < 2 centimetres.
- N2 regional lymph node metastasis into minimally a singular enlarged lymph node or lymph node mass > 2 centimetre and < 5 centimetre diameter OR metastasis into > 5 regional lymph nodes < 5 centimetre diameter OR metastasis into minimally a singular lymph node between 2 centimetre and 5 centimetre diameter.
- N3 regional lymph node metastasis into minimally a singular enlarged retroperitoneal lymph node or lymph node mass > 5 centimetre magnitude OR metastasis into minimally a singular enlarged lymph node or lymph node mass > 5 centimetre diameter.

Distant metastasis

- MX distant metastasis cannot be assessed.
- M0 distant metastasis into distant lymph nodes or various organs absent.
- M1 distant metastasis into:

- M1a metastasis into pulmonary parenchyma or distant lymph nodes as pelvic, thoracic, supraclavicular or visceral lymph nodes apart from retroperitoneal lymph nodes.
- M1b distant metastasis into viscera as hepatic parenchyma, skeletal system or brain. Pulmonary parenchyma may or may not be incriminated.

Serum tumour markers

- SX serum tumour marker levels unavailable.
- S0 serum tumour marker levels appear normal.
- S1 minimally a singular tumour marker level exceeds normal range as:
- Lactic dehydrogenase (LDH) < 1.5 times upper normal limit (ULN).
- βHCG < 5,000 mIu/mL.
- Slpha fetoprotein (AFP) < 1,000 ng/mL.
- S2 minimally a singular tumour marker appears substantially above normal range as:
- Lactic dehydrogenase (LDH) between 1.5 times to 10 times upper normal limit (ULN).
- βHCG between 5,000 to 50,000 mIu/mL.
- Alpha fetoprotein (AFP) between 1,000 to 10,000 ng/mL.
- S3 minimally ≥ one or more tumour markers are significantly elevated:
- Lactic dehydrogenase (LDH) > 10 times upper normal limit (ULN).
- βHCG > 50,000 mIu/mL.
- Alpha fetoprotein (AFP)> 10,000 ng/mL.

Tumour cells appear immune reactive to MTAP, BAP1, PAX8, Wilms's tumour factor 1 (WT1), podoplanin (D2-40), cytokeratin CK5/6, and calretinin.

Neoplastic cells appear immune non reactive to BerEP4 and MOC31 [6,7].

Well differentiated papillary mesothelial tumour of the testis requires segregation from neoplasms as mesothelioma, mesothelioma *in situ*, serous cystadenoma, serous borderline tumours, low grade serous cystadenocarcinoma or adenomatoid tumour [6,7].

Upon radiography, specific imaging features or cogent imaging anomalies appear absent. Occasionally, miniature peritoneal nodules, singular nodule or disseminated zones of peritoneal thickening may be discerned. Tumour calcification may occur.

Neoplasm may be appropriately ascertained by precise morphological evaluation [7,8].

Comprehensive surgical extermination of the neoplasm appears to be an optimal mode of therapy. Pre-eminently benign, tumour cells appear to morphologically simulate cells constituting mesothelioma *in situ* wherein tumour reappearance as mesothelioma may be discerned [7,8].

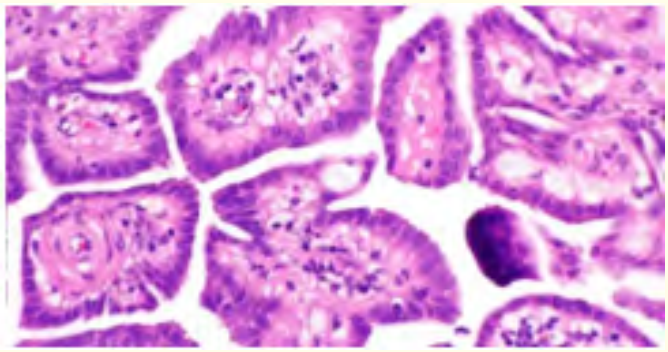

Figure 1: *Well differentiated papillary mesothelial tumour delineating papillary excrescences lined by single layer of cuboidal epithelial cells impregnated with a distinct fibro-vascular core [9].*

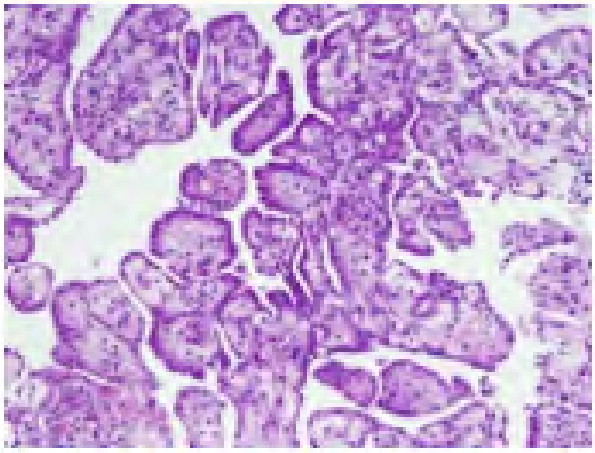

Figure 2: *Well differentiated papillary mesothelial tumour expounding papillary excrescences lined by single layer of cuboidal epithelial cells imbued with a distinct fibro-vascular core [10].*

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9. Image 1 Courtesy: Mesothelioma.com.

10. Image 2 Courtesy: Science direct.

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