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Opinion

Internecine and Melded-Adenocarcinoma Rete Testis

Anubha Bajaj*

University Panjab University, India

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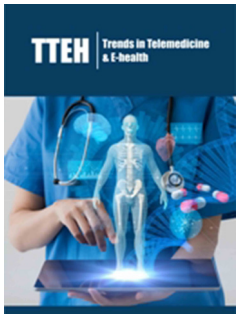

***Corresponding author:** Anubha Bajaj, University Panjab University, India

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Opinion

Adenocarcinoma rete testis emerges as an exceptionally discerned, malignant neoplasm engendered from epithelium of rete testis. Tumefaction manifests with diverse morphological representations whereas immune reactivity to varied biomarkers remains nonspecific. However, preceding terminology of papillary adenocarcinoma of rete testis or carcinoma of rete testis remains obsolete [1]. Cogent morphological features manifest with admixture of varying architectural configurations as complex tubulopapillary articulations and glandular, solid or cribriform pattern. Immune reactivity to cytokeratin 7, cytokeratin AE1/AE3 and Epithelial Membrane Antigen (EMA) is observed whereas markers as CD30, inhibin, OCT 3/4 and SALL4 appear immune non-reactive. Primary adenocarcinoma of collecting ducts and rete testis necessitates segregation from lesions as mesothelioma, metastatic adenocarcinoma or germ cell tumours. Thus, primary adenocarcinoma of rete testis or collecting ducts configures as a diagnosis of exclusion and expounds an aggressive clinical course. Adenocarcinoma rete testis is an extremely exceptional tumefaction, commonly arising within elderly Caucasian subjects. Mean age of disease emergence is 54 years. Nevertheless, young adults may display the neoplasm. A male predominance and preferential involvement of right rete testis may be encountered [2,3].

Genomic alterations may suitably be discerned within the neoplasm with massive, parallel DNA sequencing where in pathogenic variants may be encountered. Tumour cells may delineate chromosomal variants of CDKN2A, AKT1, RB1, NF2, SETD2 or TP53 genes [2,3]. Adenocarcinoma rete testis is principally confined to the testicular hilum. Subsequently, secondary involvement of adjacent anatomic structures may occur by direct tumour extension. Few subjects may depict satellite nodules situated upon the spermatic cord. Adenocarcinoma rete testis is posited to arise due to specific factors as preceding trauma, chronic epididymitis, cryptorchidism or inguinal hernia. Roughly 33% neoplasms concur with hydrocele where in individuals may be subjected to surgical procedures as ipsilateral hydrocelectomy [3,4]. Neoplasm may concur with adenomatous hyperplasia of rete testis with subsequent adenocarcinoma or concordant adenomatous hyperplasia and adenocarcinoma. Not with standing, a gradual progression from normal rete testis into adenomatous hyperplasia and subsequent primary adenocarcinoma requires appropriate as certainment [3,4]. Adenocarcinoma rete testis expounds nonspecific clinical symptoms. Predominantly, clinical representation as a scrotal mass is observed. Nearly 20% lesions are associated with scrotal pain. Occasionally, neoplasm may be discovered incidentally or following trauma to scrotal region. Additionally, initial clinical manifestation due to tumour metastases as an accompanying groin mass or lumbar pain may ensue [4,5].

Grossly, tumefaction is centred upon testicular hilum where in variable extension into testicular parenchyma, spermatic cord or superimposed cutis may occur due to direct neoplastic extension. Neoplasm is inadequately defined and firm where in tumour magnitude varies from 2 centimetres to 13 centimetres. Cut surface appears solid to cystic, tan, yellow or white and may depict focal haemorrhage [4,5]. Upon microscopy, neoplasm expounds an infiltrative pattern of tumour invasion with extension beyond rete testis and significant proliferation of intra-rete. Neoplasm exemplifies variable architecture as complex tubulopapillary, glandular, solid, cribriform, glomeruloid, retiform, sertoliform, kaposiform, micro-papillary or nested configuration. An amalgamation of aforesaid patterns may be enunciated [5,6]. Tumour cells appear as epithelioid, cuboidal or columnar epithelial cells pervaded with pale to eosinophilic cytoplasm and prominent nucleoli. Significant nuclear atypia and cellular or nuclear pleomorphism may ensure. Besides, a biphasic population of epithelioid and sarcomatoid cells may be exemplified. Circum scribing stroma is desmoplastic and may display psammoma bodies or focal calcification. Tumour necrosis may be enunciated. Mitotic figures are prominent with discernible atypical mitoses. Focal gradual transformation from normal epithelial cell layer of rete testis to dysplastic and malignant epithelial cell layer may be expounded, there by indicating a primary neoplastic origin from rete testis epithelium [5,6]. Ultrastructural examination depicts glandular spaces imbued with amorphous, electron dense material (Figure 1). Luminal spaces appear layered with epithelial cells pervaded with ovoid nuclei delineating nuclear indentations, smooth outline, dispersed nuclear heterochromatin and prominent nucleoli (Figure 2). Few cells may demonstrate lipid droplets and bundles of intermediate cytoplasmic filaments [5,6] (Table 1).

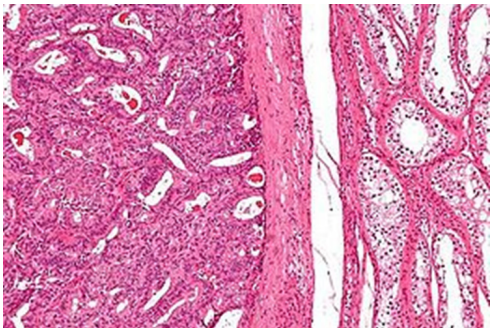

Figure 1: Adenocarcinoma demonstrating multiple configurations of tubules, papillae, glands, nests, solid aggregates and retiform structures lined by cuboidal to columnar epithelium imbued with eosinophilic cytoplasm and prominent nucleoli. Mitotic figures are seen. Surrounding stroma is fibrotic [11].

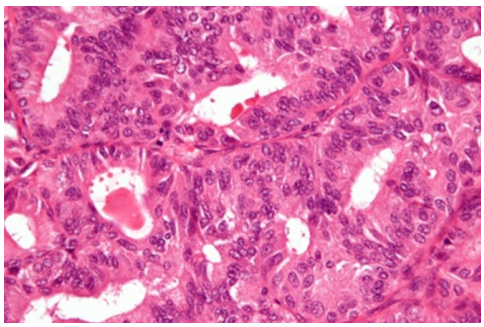

Figure 2: Adenocarcinoma delineating cords, nests, glands and papillae lined by columnar to cuboidal epithelium impregnated with eosinophilic cytoplasm and prominent nucleoli. Several mitotic figures are seen. Intervening stroma is minimal [12].

Table 1: Prognostic groups of testicular cancer as per Union for International Cancer Control (UICC) [5].

Stage	T	N	M	S
Stage 0	Tis	N0	M0	S0
Stage I	T1-T4	N0	M0	SX
Stage IA	T1	N0	M0	S0
Stage IB	T2-T4	N0	M0	S0
Stage IS	Any T/TX	N0	M0	S1-S3
Stage II	Any T/TX	N1-N3	M0	SX
Stage IIA	Any T/TX	N1	M0	S0
	Any T/TX	N1	M0	S1
Stage IIB	Any T/TX	N2	M0	S0
	Any T/TX	N2	M0	S1
Stage IIC	Any T/TX	N3	M0	S0
	Any T/TX	N3	M0	S1
Stage III	Any T/TX	Any N	M1a	SX
Stage IIIA	Any T/TX	N1-N3	M0	S0
	Any T/TX	Any N	M1a	S1
Stage IIIB	Any T/TX	N1-N3	M0	S2
	Any T/TX	Any N	M1a	S2

Stage IIIC	Any T/TX	N1-N3	M0	S3
	Any T/TX	Any N	M1a	S3
	Any T/TX	Any N	M1b	Any S

Adenocarcinoma rete testis appears immune reactive to cytokeratin AE1/AE3, CK7, CK5/6, Epithelial Membrane Antigen (EMA), vimentin, CD10, BerEP4, MOC1 or PAX8. Variable immune reactivity to calretinin and Wilm's Tumour 1(WT1) antigen is observed [6,7]. Tumour cells appear immune non-reactive to CD30, CK20, inhibin, NKX3.1, OCT3/4, Prostate Specific Antigen (PSA), Prostatic Acidic Phosphatase (PSAP) and SALL4[6,7]. Adenocarcinoma of rete testis requires segregation from neoplasms as mesothelioma, distant metastasis from adenocarcinoma primaries, germ cell tumours, epididymal carcinoma, malignant sertoli cell tumour or ovarian subtype of epithelial tumour arising within para-testicular region. As primary adenocarcinoma of collecting ducts or rete testis is a diagnosis of exclusion, various testicular, para-testicular and metastatic neoplasms require appropriate discernment and categorization in order to obtain a diagnosis of primary rete testis adenocarcinoma [7,8].

Cogent and current diagnostic criterion for ascertaining primary adenocarcinoma of rete testis, as denominated by Nochomovitz and Orenstein are represented as

a) Tumour localization within testicular hilum.

b) Absence of morphologically identical extra-scrotal neoplasm configuring as a probable site of primary tumour.

c) Tumour morphology inconsistent with diverse subtypes of testicular or paratesticular neoplasms.

d) Precise immuno histochemistry and exclusion of diverse neoplastic variants, particularly mesothelioma or papillary serous carcinoma, upon immuno histochemistry.

e) Discernible transition of normal epithelium into neoplastic epithelium layering rete testis. However, aforesaid morphology frequently remains unidentifiable as normal anatomical configuration is obliterated by neoplastic transformation [7,8].

Neoplasm depicts normal serum values of Alkaline Phosphatase (ALP), Alpha Fetoprotein (AFP), CA-125, CA 19-9, Carcinoembryonic Antigen (CEA) or β Human Chorionic Gonadotropin (βHCG). Alternatively, elevated levels of serum CA 19-9 and Carcinoembryonic Antigen (CEA) may be discerned [8,9]. Cogent radiographic imaging may be adopted for exclusion of possible primary extra-scrotal tumour, visualization of scrotal tumour and precise tumour staging. Foci of intra-scrotal calcification may be discerned. Ultrasonography delineates a poorly defined, hypoechoic, heterogeneous tumour mass with enhanced vascularization [8,9]. Positron Emission Tomography (PET/CT) may be beneficially adopted, in contrast to conventional computerized tomography, for apply detecting tumour associated distant metastases [8,9]. Precise therapy of primary rete testis adenocarcinoma remains non standardized. Not with standing, neoplasm may suitably be subjected to surgical manoeuvers as radical orchiectomy along with or in the absence of retroperitoneal lymph node dissection. Concordant, adjuvant chemotherapy or radiotherapy may be employed. Nevertheless, therapeutic resistance to conventional chemotherapy regimens is frequently encountered [9-12].

Prognostic factors associated with superior therapeutic outcomes are denominated as

A. Tumour magnitude < 5 centimetres.

B. Tumour confined to the testis [9,10].

References

1. Feek JD, Hunter WC (1945) Papillary carcinoma arising from rete testis. Arch Pathol (Chic) 40: 399-402.
2. Chen Y, Chen Y, Sun Q, Hou X, Fu S, et al. (2024) Adenocarcinoma of the rete testis: Clinicopathological study of 18 cases with emphasis on MET amplification and a review of the literature. Histopathology 86(5): 762-771.
3. Li K, Chen D, He M, Yu J, Mi H (2024) Sunitinib for adenocarcinoma of the rete testis: A case report. Front Oncol 14: 1358133.
4. Mitchell CF, Pearce S (2022) Adenocarcinoma of the rete testis: A case report. Cureus 14(2): e22210.
5. Meyts ER, Aksglaede L, Bandak M, Toppari J, Jørgensen N, et al. (2023) Testicular cancer: Pathogenesis, diagnosis and management with focus on endocrine aspects. In: Feingold KR, Anawalt B, Blackman MR et al. (Eds.), Endotext. South Dartmouth (MA): MDText.com.
6. Al-Obaidy KI, Idrees MT, Grignon DJ, Thomas MU (2019) Adenocarcinoma of the rete testis: Clinicopathologic and immunohistochemical characterization of 6 cases and review of the literature. Am J Surg Pathol 43(5): 670-681.
7. Owa S, Sasaki T, Uchida K, Susumu W, Kato M, et al. (2021) A case of adenocarcinoma of the rete testis with durable response to cisplatin-based chemotherapy. IJU Case Rep 4(5): 267-271.
8. Zhang Y, He X, Gao H (2022) KMT_2C mutation in a Chinese man with primary multidrug-resistant metastatic adenocarcinoma of rete testis: A case report. BMC Urol 22(1): 123.
9. Jin J, Xie Y, Zhang JS, Wang JQ, Dai SJ, et al. (2023) Sunitinib resistance in renal cell carcinoma: From molecular mechanisms to predictive biomarkers. Drug Resist Updat 67: 100929.
10. Kitano H, Sentani K, Goto K, Sekino Y, Yamanaka R, et al. (2020) Primary adenocarcinoma of the rete testis with elevated serum CA19-9 antigen levels. Int Cancer Conf J 9(4): 240-243.
11. Image 1 Courtesy: Libre Pathology.
12. Image 2 Courtesy: Wikimedia commons.