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The Metamorphosed Girdle-Carcinoma Prostate

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Introduction

Carcinoma prostate is a frequently discerned malignancy of prostate gland emerging from prostatic secretory epithelium. Tumefaction is devoid of specific or sensitive clinical and radiological features which may assist appropriate disease discernment. Neoplasm is frequently detected by non-targeted fine or core needle tissue samples obtained secondary to evaluation of elevated serum prostate specific antigen (PSA) levels [1, 2]. Additionally designated as prostate cancer or prostatic adenocarcinoma, carcinoma prostate exhibits distinctive subtypes as acinar adenocarcinoma, ductal adenocarcinoma, atrophic adenocarcinoma, pseudo-hyperplastic adenocarcinoma, micro-cystic adenocarcinoma, foamy gland adenocarcinoma, mucinous adenocarcinoma, signet ring variant of adenocarcinoma, pleomorphic giant cell adenocarcinoma or sarcomatoid adenocarcinoma [1, 2].

Majority of carcinoma prostate are multifocal and predominantly (~80%) confined to posterior or posterolateral peripheral zone. Few neoplasms (~20%) emerge within transition or periurethral zone [1, 2]. Clinically significant neoplasms pre-eminently emerge within peripheral zone and are amenable to cogent discernment by fine or core needle tissue samples. Carcinoma prostate emerging within transition zone is associated with favourable morphology and superior reoccurrence free survival [1, 2].

Lesions confined to anterior prostate are infrequently discerned possibly due to inadequate, standardized surgical tissue sampling [1, 2].

Factors contributing to emergence of carcinoma prostate are obesity or non-modifiable risk factors as age, racial predilection and family history. Carcinoma prostate can be inherited. Genetic susceptibility with first degree relative of subjects with carcinoma prostate enhances possible disease emergence [1, 2].

BRCA2 genetic mutation elevates proportionate disease occurrence wherein BRCA2 associated neoplasms incriminate young subjects and demonstrate inferior survival outcomes. Additionally, germline variants of HOXB13 appear associated with carcinoma prostate. Certain germline variants may augment emergence of adenocarcinoma prostate. Lynch syndrome depicts enhanced incidence of carcinoma prostate.

Familial disease occurs due to genetic factors which may induce certain exceptional disease variants with significant penetration or commonly observed variants with minimal to moderate disease occurrence. Variants with enhanced disease penetration are associated with BRCA2 and HOXB13 genetic mutations [1, 2].

Several single nucleotide pleomorphisms (SNPs) are associated with occurrence of carcinoma prostate and may display minimal to moderate influence upon disease progression. Majority of SNPs delineate an obscure molecular mechanism of carcinogenesis as noncoding regions of genome are commonly afflicted. Elevated levels of IGF1 enhances possible occurrence of carcinoma prostate [1, 2].

Carcinoma prostate exhibits distinctive subtypes contingent to specific genetic fusions within ETS transcription family members as ERG, ETV1, ETV4 and FLI1 or chromosomal mutations within SPOP, FOXA1, IDH1, PTEN, TP53, MYC or CDH1 genes [1, 2]. Frequently discerned somatic genomic rearrangement is fusion of androgen regulated gene TMPRSS2 with a member of ETS transcription family. Somatic genetic mutation profiles of carcinoma prostate appear associated with definitive clinical and pathological outcomes [1, 2].

Diverse subtype's exhibit varying molecular profiles as ~SPOP mutant subset exemplify distinctive profiles of somatic copy number alterations as deletions of CHD1 and chromosomal region 6q or 2q. ~an ETS subset appears enriched with PTEN genomic mutations. Carcinoma prostate is generally asymptomatic. Locally advanced or metastatic neoplasms may depict specific clinical symptoms. Neoplasm can be discerned following evaluation of nonspecific lower urinary tract symptoms [1, 2].

Digital rectal examination (DRE) of prostate may be unremarkable or the organ may appear enlarged, asymmetrical, and hard or manifest a palpable nodule [1, 2]. Aspiration of carcinoma prostate metastatic into regional lymph nodes may demonstrate micro-acinar complex, cellular clusters or singular cells with fragile cytoplasm and prominent nucleoli [1, 2].

Upon gross examination, the neoplasm appears imperceptible. Cut surface may exhibit an off white neoplastic zone [1, 2]. Upon microscopy, carcinoma prostate exhibits pathognomonic histological features as absence of basal layer, perineural invasion confined to circumferential nerves, glomerulations and collagenous micro-nodules denominated as mucinous fibroplasia [1, 2].

Additionally, an infiltrative tumour architecture, amphophilic cellular cytoplasm, nucleolar prominence and intraluminal contents as crystalloids, blue mucin or pink amorphous material can be observed. The infiltrative neoplasm exhibits glandular crowding. Tumour cells exhibit amphophilic cytoplasm and spherical, monomorphic or enlarged nuclei with prominent nucleoli. Mitotic figures or apoptotic bodies may be enunciated. Surrounding stroma is desmoplastic. Collagenous micro-nodules denominating mucinous fibroplasia are demonstrated [1, 2].

Intraluminal crystalloids, pink amorphous secretions or bluish mucin can be delineated. Foci of glomerulations may ensue. Glandular configurations are devoid of basal cell layer which can be confirmed upon pertinent immunohistochemistry [1, 2] and (Figures 1 and 2).

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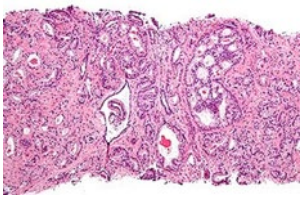

Figure 1: Carcinoma prostate depicting neoplastic glands layered by transformed columnar epithelium admixed with glands imbued with intraluminal eosinophilic aggregates and circumscribing fibrotic stroma [3].

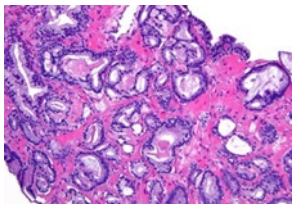

Figure 2: Carcinoma prostate delineating glands lined with neoplastic and desquamated epithelium surrounded by a dense, fibrotic stroma invaded by neoplastic glands [4].

TNM classification of carcinoma prostate is designated as **Primary tumour.**

- T1: Tumour discerned upon TURP, fine or core needle biopsy.
- T2: Tumour confined to organ and categorized as ~T2a: Tumour discerned with Digital Rectal Examination (DRE) or trans-rectal ultrasound and appears unilateral, ~T2b: Tumour discerned with Digital Rectal Examination (DRE) or trans-rectal ultrasound and extends to contralateral segment, ~T2c: Tumour discerned with Digital Rectal Examination (DRE) or trans-rectal ultrasound and appears bilateral
- T3 designated as ~T3a: Tumour with focal or multifocal extra-prostatic extension or microscopic invasion of dense musculature of urinary bladder neck with absence of adjacent non-neoplastic glands, ~T3b: Tumour invasion into musculature of seminal vesicle.
- T4: Tumour invasion into external urethral sphincter, rectum, bladder, levator muscles or pelvic wall.

Regional Lymph Nodes

- NX: Regional lymph nodes cannot be assessed
- N0: Regional lymph node metastasis absent
- N1: Regional lymph node metastasis present

Regional lymph nodes are exemplified by peri-prostatic, pelvic, hypogastric, obturator, internal iliac, external iliac or sacral lymph nodes.

Distant Metastasis

- M0: Distant metastasis absent.
- M1 is designated as ~M1a: Distant metastasis into non-regional lymph nodes as aortic, common iliac, superficial inguinal, deep inguinal or retroperitoneal lymph nodes, ~M1b: Distant metastasis into bone, ~M1c: Distant metastasis into diverse viscera [2, 5].

Gleason grading of carcinoma prostate is contingent to tumour architecture and represents a morphological continuum of well-formed glandular pattern to complex glandular proliferation or lack of glandular differentiation. Gleason grades are designated as:

- Grade X: Gleason score cannot be determined.
- Grade 1(Gleason score ≤ 6): Individual, discrete, well configured glands and serum PSA <10 ng/ml or between 10 ng/ml and 20 ng/ml.
- Grade 2(Gleason score 3+4=7): Predominant well configured glands with a minimal component of inadequately defined, fused or cribriform glands and serum PSA <20ng/ml.
- Grade 3 (Gleason score 4+3=7 or 8): Predominant inadequately configured, fused or cribriform glands with miniscule component of well configured glands and serum PSA <20ng/ml.
- Grade 4 (Gleason score 8): Singularly composed of inadequately configured, fused or cribriform glands or predominantly well configured glands with minor component devoid of glands or a tumefaction predominantly devoid of glands with miniature component of well configured glands and serum PSA ≥ 20 ng/ml.
- Grade 5 (Gleason score 9 or 10): Absence of glandular formation or glands associated with necrosis along with or devoid of inadequately configured, fused or cribriform glands and an elevated serum PSA level [2, 5].

Clinically, grade 1 denominates a morphological 'low grade', grade 2 exemplifies an 'intermediate grade' and grade 3 or elevated grade enunciates 'high grade' neoplasms [2, 5].

Staging of carcinoma prostate is denominated as:

- Stage I: Tumour is preliminary, non-palpable, and gradually progressive and incriminates unilateral aspect of prostate. Serum PSA levels are minimal. Tumour cells simulate uninvolved prostatic epithelium.
- Stage II: Tumour confined to prostate. Serum PSA levels are minimal to moderate. Progression and dissemination of miniature tumefaction is exemplified.
- Stage IIA: Tumour is non-palpable and unilateral. Serum PSA levels are moderately elevated. Neoplastic cells are well differentiated. Enlarged neoplasms composed of well differentiated prostatic epithelial cells confined to prostate are categorized as stage IIA neoplasms.
- Stage IIB: Tumour is confined to prostate and appears palpable upon direct rectal examination (DRE). Serum PSA levels are moderately elevated. Tumour cells are moderately differentiated.
- Stage IIC: Tumefaction is confined to prostate and appears palpable upon DRE. Serum PSA levels are moderately elevated. Neoplastic cells are moderately or poorly differentiated.
- Stage III: Serum PSA levels are significantly elevated. Tumour is high grade and progressive. The stage is constituted of locally advanced prostatic carcinoma with significant tumour progression and metastasis.
- Stage IIIA: Tumefaction extends beyond extrinsic layer of prostate into circumscribing soft tissues or seminal vesicles. Serum PSA levels are significantly elevated.
- Stage IIIB: Tumefaction extends beyond prostate gland with extension into adjacent viscera as urinary bladder or rectum.
- Stage IIIC: Tumour cells appear poorly differentiated.

- Stage IV: Tumefaction extends beyond prostate ~ stage IVA: Tumefaction depicts regional lymph node metastasis, ~stage IVB: Tumefaction disseminates into distant lymph nodes, diverse viscera or bones [2, 5].

Recurrent carcinoma prostate is denominated by neoplastic reappearance following therapy. Tumour may re-emerge within prostatic zone or diverse areas. Extent of tumour reoccurrence necessitates additional evaluation [2, 5]. Carcinoma prostate is immune reactive to PSA, NKX3.1, AMACR, prostein or PSMA. Neoplasms may aberrantly express p63. Carcinoma prostate is immune non-reactive to CK7, CK20, high molecular weight cytokeratin as 34βE1212, CK5 or CK5/6, p63, CDX2,GATA3 or TTF1[5, 6].

Carcinoma prostate requires segregation from conditions such as benign prostatic hyperplasia, prostatic atrophy, adenosis, atypical small acinar proliferation (ASAP), high grade prostatic intraepithelial neoplasia, post-atrophic hyperplasia, partial atrophy, radiation atypia, urothelial carcinoma, acute bacterial prostatitis, prostatic abscess, chronic bacterial prostatitis, nonbacterial prostatitis or tuberculosis of genitourinary system [5, 6]. Carcinoma prostate can be appropriately discerned with systematic trans-rectal ultrasonography and ultrasound guided surgical tissue samples. Alternatively, transperineal fine or core needle samples can be obtained which appear associated with minimal prostatic infection. Magnetic resonance imaging (MRI) can be adopted prior to and supplemented with systematic, targeted surgical tissue sampling obtained from anomalous zones as discerned with radiography. Aforesaid manoeuver is superior to singular systematic tissue sampling adopted for identification of clinically significant carcinoma prostate. Incidental carcinoma prostate may occasionally be diagnosed with transurethral resection [5, 6].

Pertinent immunohistochemistry with basal cell markers as HMWCK, p63 or AMACR can be employed to adequately diagnose debatable prostatic adenocarcinomas [5, 6]. Serum PSA levels are elevated with malignant metamorphosis of prostate. Thus, periodic evaluation of serum PSA for screening of carcinoma prostate is contemplated to be an individual preference. PCA3 is a contemporary urine biomarker which can be employed for detecting carcinoma prostate [5, 6].

Examination of urine samples for discerning the neoplasm appears to be minimally sensitive and is not recommended [5, 6]. Ultrasonography (US) is beneficially employed to obtain prostatic tissue samples. Upon sonography, carcinoma prostate emerges as a hypoechoic zone. However, ultrasonography lacks sensitivity or specificity for appropriately detecting carcinoma prostate [5, 6].

Computerized tomography (CT) is beneficial in assessing instances of carcinoma prostate metastatic to regional lymph nodes. Bone scan is optimal in detecting bony metastases [5, 6]. Magnetic resonance imaging (MRI) is commonly adopted to ascertain localized tumour staging. The procedure can be utilized to identify parenchymal anomalies for obtaining targeted surgical tissue samples. Anomalies detected upon MRI are categorized with Prostate Imaging - Reporting and Data System (PI-RADS) or Likert score [5, 6].

Positron emission tomography (PET) scan is advantageous in discerning micro-metastatic disease in specific subjects as individuals with elevated serum PSA levels following therapy [5, 6]. Preoperative risk stratification of carcinoma prostate is contingent to serum PSA, clinical stage, morphological features as extent and grade of tumefaction, cribriform morphology, intra-ductal carcinoma and perineural invasion [5, 6].

Cogent therapeutic options pertaining to preoperative risk stratification are denominated as active surveillance, focal therapy as Cryotherapy or high intensity ultrasonography, radical prostatectomy, brachytherapy, external beam radiotherapy, hormonal therapy with luteinizing hormone releasing hormone (LHRH) analogues or antiandrogens and surgical manoeuvers as orchidectomy [5, 6]. Chemotherapy is suitable for treating carcinoma prostate exhibiting regional lymph node and distant metastasis [5, 6].

Following prostatectomy, monitoring with serum PSA levels is recommended. Preliminary salvage therapy can be adopted for instances depicting increasing serum PSA [5, 6]. Adjuvant therapy can be infrequently employed for advanced stage disease or tumour deposits within surgical perimeter [5, 6]. Prognostic outcomes are contingent to extent of tumefaction denominated as tumour depth in millimetres or percentage core involvement, tumour grade with Gleason score, perineural invasion and extra-prostatic tumour extension [5, 6]. Radical prostatectomy can be employed contingent to tumour magnitude, tumour grade with Gleason score, and tumour stage and tumour metastasis into surgical perimeter [5, 6].

Morphological features as a cribriform morphology and intra-ductal carcinoma associated with invasive carcinoma prostate emerge as adverse prognostic indicators [5, 6]. Small cell variant of carcinoma prostate is associated with aggressive biological behaviour and mandates diverse therapeutic strategies. Intra-ductal component of carcinoma prostate may be incorporated into Gleason score with tumour grading or may be indicated with pertinent comment [5, 6].

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