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The Compact Bleb-Eosinophilic Solid and Cystic Renal Cell Carcinoma

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Abstract

The contemporary, indolent, Eosinophilic solid and cystic renal cell carcinoma configures as a predominantly solitary neoplasm delineating solid and cystic pattern of tumour evolution. Neoplastic cells are incorporated with abundant Eosinophilic cytoplasm and intracytoplasmic inclusions or basophilic stippling. Tumour cells display frequent somatic loss of function genetic mutations confined to TSC1 (9q34.13) gene or TSC2 (16p13) gene. Eosinophilic tumour cell clusters appear admixed with macroscopic cysts or microscopic tubules and cystic structures coated with singular layer of hobnail cells. Neoplastic cells are permeated with abundant, granular, eosinophilic cytoplasm. Tumour cells appear immune reactive to PAX8, CK20, AMACR, CD10, SDHB, fumarate hydratase, vimentin, cathepsin K and Melan A.

Keywords: Eosinophilic; Solid; Cystic; Carcinoma; Staging

Abbreviations: PAX8: Paired Box Protein 8; CK20: Cytokeratin 20; AMACR: Alpha Methyacyl CoA Racemase; mTOR: Mammalian Target of Rapamycin.

Introduction

Eosinophilic solid and cystic renal cell carcinoma configures as a contemporary variant of renal cell carcinoma demonstrating solid and cystic pattern of tumour evolution and an indolent clinical course. Neoplastic cells are incorporated with abundant eosinophilic cytoplasm. Clinical history or features indicative of associated tuberous sclerosis complex appear absent. However, tumefaction frequently exhibits somatic loss of function genomic mutations within TSC1 gene or TSC2 gene. Eosinophilic solid and cystic renal cell carcinoma is comprised of solid and cystic components. Tumour cells are imbued with copious eosinophilic cytoplasm and frequent, intracytoplasmic inclusions or basophilic stippling. Tumour cells appear immune reactive to CK20+ and PAX8+ and immune non-reactive to CK7- or c-Kit-. A mild female predilection is observed. Tumefaction may arise within subjects of 14 years to 75 years. Neoplasm is devoid of specific clinical features or family history. Eosinophilic solid and cystic renal cell carcinoma incriminates left and right kidneys with equivalent predisposition [1,2].

Sporadic neoplasms represent within a spectrum of renal neoplasms which harbour TSC1 or TSC 2 genetic alterations. The feature is exemplified within tumours as granular Eosinophilic macro-cystic subtype of tuberous sclerosis associated renal cell carcinoma or oncocytoid subtype of renal cell carcinoma post-neuroblastoma [1,2]. The TSC1 (hamartin) gene and TSC2 (tuberin) gene configures functional intracytoplasmic heterodimer implicating cell signalling and proliferation through activation of kinase mTOR. Additionally, negative regulators of AKT / mTOR signalling pathway appear contributory. Besides, loss

of function mutations within TSC1 gene or TSC2 gene engenders constitutive mTORC1 activation which appears disparate and uncoupled from upstream signalling inputs [1,2]. Tumour cells display frequent somatic loss of function genetic mutations confined to TSC1 (9q34.13) gene or TSC2 (16p13) gene. Also, 'second hit' genetic alterations as loss of heterozygosity or > one deleterious chromosomal mutation within a singular TSC1 gene or TSC2 gene may be occasionally encountered [2,3].

Copy number gains within chromosomal loci 16p13.3-q23.1, 7p21.2-q36.2 or TSC2 locus may ensue. Copy number loss within TSC1 gene may be expounded. Besides, loss of heterozygosity within chromosome 16p11.2-1 or Xq11.1-13.1 may be encountered. Although predisposing factors appear obscure, tumefaction is associated with somatic loss of tumour suppressor function initiated by TSC1 gene or TSC2 gene [2,3].

Grossly

Solitary lesions are frequently observed. Occasionally, multifocal neoplasms may be discerned. Bilateral tumours are exceptional. Tumefaction appears as well delineated, well circumscribed and exemplifies a 'pushing' border. Tumour manifests as yellow to tan, solid nodule characteristically interspersed with macro-cystic spaces. Occasionally, entirely solid neoplasms may be enunciated. Tumour magnitude varies from < 1.0 centimetres to 20.5 centimetres. Foci of haemorrhage and tumour necrosis are variable [2,3].

Upon microscopy

The non-encapsulated, solid tumefaction delineates a compact, nested tumour configuration. Aggregates of Eosinophilic tumour cells appear admixed with macroscopic cysts or microscopic tubules and cystic structures. However, cystic articulations may be focal or absent. Focal papillary architecture may ensue [3,4]. Characteristically, cysts are coated with singular layer of hobnail cells. Besides, multinucleated tumour cells are frequently encountered. Neoplastic cells are permeated with abundant, granular Eosinophilic cytoplasm. Occasionally, cytoplasm may display centric condensation with well circumscribed perimeter and clear or flocculent cytoplasm. Intracytoplasmic inclusions are exemplified. Basophilic stippling appears as finely granular to coarse. Additionally, densely Eosinophilic or purple cytoplasmic globules appear encompassed within delicate, clear cytoplasm, reminiscent of cells of leishmaniasis [3,4]. Cytoplasmic vacuoles appear as macro-vesicular or micro-vesicular, thus imparting a 'clear cell' countenance to neoplastic cells. Tumour cells confined to solid areas may be pervaded with moderate cytoplasm. Tumour cell nuclei appear mildly pleomorphic, spherical to elliptical and are permeated with variably prominent nucleoli. Bi-nucleated or multi-nucleated cells appear disseminated within tumour cell aggregates. Clusters of foamy macrophages are commonly discerned. Focal calcification or osseous metaplasia is exceptional. Ultrastructural examination exhibits abundant rough endoplasmic reticulum in association with granular intracellular substance [4,5].

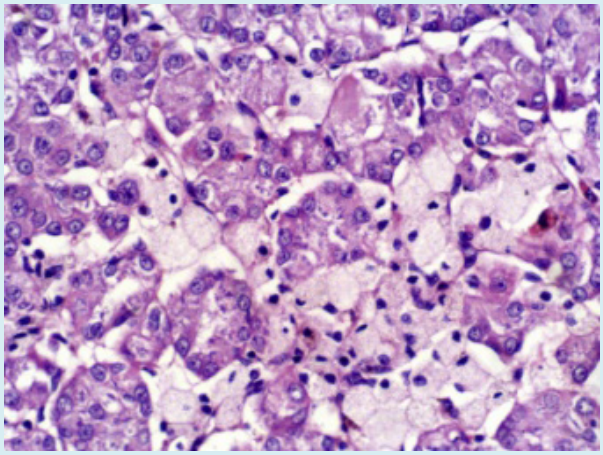

Figure 1: Eosinophilic solid and cystic renal cell carcinoma demonstrating cysts layered with epithelial cells imbued with abundant, Eosinophilic cytoplasm, intracytoplasmic vacuoles and uniform nuclei with prominent nucleoli. Few foamy macrophages are discerned [6].

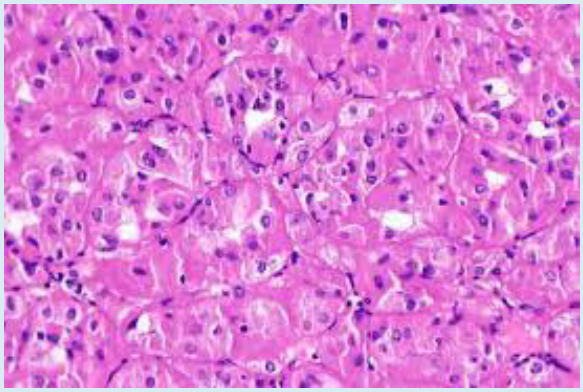

Figure 2: Eosinophilic solid and cystic renal cell carcinoma delineating cysts lined by cells pervaded with abundant, Eosinophilic cytoplasm, intracytoplasmic vacuoles and mildly pleomorphic nuclei with conspicuous nucleoli [7].

TNM Staging of Renal Cell Carcinoma

As per American joint committee on cancer 8th edition

Primary Tumour

TX: Primary tumour cannot be assessed
T0: No evidence of primary tumour

T1: is subdivided into

- T1a: tumour confined to renal parenchyma<4 centimetre diameter
- T1b: tumour confined to renal parenchyma>4 centimetre and<7 centimetre diameter

T2: Tumour confined to renal parenchyma ≥ 7 centimetre diameter

- T2a: tumour confined to renal parenchyma >7 centimetre and up to 10 centimetre diameter
- T2b: tumour confined to renal parenchyma >10 centimetre diameter

T3: Tumour of variable magnitude with extension into major veins as renal vein or inferior vena cava or perinephric tissues although ipsilateral adrenal gland is spared. Tumour extension beyond Gerota's fascia is absent.

- T3a: tumour demonstrates macroscopic extension into renal vein or muscular, segmental branches with tunica media or tumour invades peri-renal tissue or adipose tissue of renal sinus. Tumour extension beyond Gerota's fascia is absent.
- T3b: tumour dissemination into infra diaphragmatic inferior vena cava
- T3c tumour dissemination into supra diaphragmatic inferior vena cava or invasion into wall of inferior vena cava

T4: Tumour of variable magnitude incriminating ipsilateral adrenal gland or tumour extension beyond Gerota's fascia.

Regional Lymph Nodes

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

Distant Metastasis

- M0: Distant metastases absent
- M1: Distant metastases present

Staging of Renal Cell Carcinoma

- Stage I: T1, N0, M0
- Stage II: T2, N0, M0
- Stage III: T3, N0, M0 OR T1, T2, or T3, N1, M0
- Stage IV: T4, any N, M0 OR any T, any N, M1.

Contiguous incrimination of ipsilateral adrenal gland is contemplated as stage T4 whereas non-contiguous involvement is designated as M1.Commonly discerned sites of distant metastasis emerge as pulmonary parenchyma, bone, lymph node, hepatic parenchyma, adrenal gland and brain, in decreasing order of frequency. Tumour cells appear immune reactive to PAX8, CK20, alpha methyacyl CoA racemase (AMACR), CD10, succinate dehydrogenase subunit B(SDHB), fumarate hydratase, vimentin, cathepsin K and Melan A. Neoplastic cells appear immune non-reactive to CK7, CD117, carbonic anhydrase IX(CAIX), human melanoma black 45(HMB45) antigen and transcription factor E3(TFE3). Germline genetic evaluation indicative of tuberous sclerosis complex remains undocumented [4,5].

Eosinophilic solid and cystic renal cell carcinoma requires segregation from neoplasms such as tuberous sclerosis complex associated renal cell carcinoma, renal cell carcinoma in tuberous sclerosis complex, renal angiomyoadenomatosis tumour-like renal cell carcinoma with angioleiomyoma-like stroma, renal cell carcinoma postneuroblastoma, oncocytoid renal cell carcinoma, hybrid oncocytic chromophobe tumour(HOCT), clear cell renal cell carcinoma, papillary renal cell carcinoma, MiT family translocation renal cell carcinoma, TSC2 or MTOR mutated sporadic renal cell carcinoma, high grade oncocytic tumour, epithelioid angiomyolipoma, papillary renal cell carcinoma, solid variant oncocytic type, chromophobe renal cell carcinoma, Eosinophilic type, SDH deficient renal cell carcinoma or clear cell renal cell carcinoma, Eosinophilic morphology [4,5]. Upon radiography, a heterogeneous, well defined tumour nodule with disseminated, miniature cystic areas is encountered. Cystic spaces may display thickened, irregular walls and may be traversed by distinct, fibrous tissue septa [4,5]. Eosinophilic solid and cystic renal cell carcinoma may be appropriately alleviated by precise surgical eradication. Commonly, lesion is devoid of progression and represents as a solitary tumefaction confined to renal parenchyma [4,5].

Chemotherapeutic agents with mTOR inhibition may be beneficially adopted for treating neoplasms with distant metastasis. Comprehensive clinical response is documented within metastatic neoplasms delineating TSC2 genetic mutation with utilization of therapeutic agents as first generation rapamycin analogue, everolimus [4,5]. Characteristically, neoplasm is associated with indolent biological behaviour. Exceptionally, localized tumour aggression or distant metastasis may ensue, especially within sites such as lymph nodes, hepatic parenchyma, pulmonary parenchyma or bone [4,5].

Conclusion

Eosinophilic solid and cystic renal cell carcinoma requires segregation from neoplasms such as tuberous sclerosis complex associated renal cell carcinoma, renal cell carcinoma in tuberous sclerosis complex, renal angiomyoadenomatosis tumour-like renal cell carcinoma with angioleiomyoma-like stroma, renal cell carcinoma postneuroblastoma, oncocytoid renal cell carcinoma, hybrid oncocytic chromophobe tumour(HOCT), clear cell renal cell carcinoma, papillary renal cell carcinoma, MiT family translocation renal cell carcinoma,

TSC2 or MTOR mutated sporadic renal cell carcinoma, high grade oncocytic tumour, epithelioid angiomyolipoma, papillary renal cell carcinoma, solid variant oncocytic type, chromophobe renal cell carcinoma, Eosinophilic type, SDH deficient renal cell carcinoma or clear cell renal cell carcinoma, Eosinophilic morphology. Radiographic imaging exhibits a heterogeneous, well defined tumour nodule with disseminated, miniature cystic areas. Surgical extermination of the neoplasm is a recommended mode of therapy.

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6. Image 1 Courtesy: Science direct

7. Image 2 Courtesy: Wikimedia commons