Print ISSN: 2321-6379
Online ISSN: 2321-595X
DOI: 10.17354/ijss/2017/385

Original Article

A Radiology-pathological Correlation of Renal Cell Carcinoma in a Tertiary Care Hospital - A Retrospective Study

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Abstract

Introduction: This study was conducted to understand the clinical algorithm of renal cell carcinoma (RCC). Correlation was done by a clinical presentation with radiological features and histopathology of RCC. The stress upon to understand the necessity for a team approach between clinician, radiologist, and pathologist and vice versa is emphasised.

Aim: The aim of this study was to correlate the histopathology of RCC with the radiological features.

Materials and Methods: The total number of renal tumors studied during the 8-year period was 45 cases, of which 25 cases were diagnosed by histopathology as various types of RCC conclusively. This is a retrospective study of renal tumors, diagnosed by histopathology as various types of RCC. All the relevant clinical data of the patients were searched from the ward records. The various radiological features were collected.

Results: The total number of renal tumors studied during the 8-year period was 45 cases, of which 25 cases were diagnosed by histopathology as various types of RCC conclusively. Magnetic resonance imaging provides molecular information with regard to RCC and potentially aid in biopsy planning. The total cases reported in the department are 25 cases out of which 16 cases are attending follow-up after 3 years.

Conclusion: The Fuhrman grading of RCC correlated grading of RCC. Pre-operative radiological classification can be used as a supplement to the histopathological grading. RCC needs correlation between radiologist, pathologist, and clinician.

Key words: Chromophobe renal cell carcinoma, Clear cell renal cell carcinoma, Magnetic resonance imaging, Multidetector computed tomography, Papillary renal cell carcinoma, Treatment protocols, Tumor staging (TNM)

INRODUCTION

Representing 2-3% of adult cancers, renal cell carcinoma (RCC) accounts for 90% of renal malignancies and is the most lethal neoplasm of the urologic system. RCC is a kidney cancer that originates in the lining of the proximal convoluted tubule, the 2004 World Health Organization Classification of adult renal tumors stratifies RCC into several distinct histologic subtypes of which clear cell, papillary, and chromophobe tumors account for 70%, 10-15%, and 5%, respectively. RCC accounts for 90% of adult renal malignancies and is the most lethal of all urologic cancers.[1-3] RCC is not a single entity but rather a heterogeneous group of neoplasms with varying histological findings, cytogenetic abnormalities, biologic behavior, prognosis, and response to therapy.[1,4-10] Chromosome 3p deletions are found in up to 96% of clear cell RCCs including somatic inactivating mutations of the von Hippel–Lindau (VHL) gene.[11,12] Cytogenetic abnormalities are associated with the papillary subtype include trisomies of chromosomes 3, 7, 12, 16, 17, and 20, c-MET mutations, and loss of the Y chromosome.[11,13,14] Cytogenetic abnormalities associated with chromophobe RCC include loss of multiple chromosomes such as 1, 2, 6, 10, 13, 17, and 21.[15] The clear cell subtype shows a less favourable outcome compared with papillary and chromophobe subtypes, and is more likely to be symptomatic, present at an advanced stage, and show a

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Month of Submission : 06-2017
Month of Peer Review : 07-2017
Month of Acceptance : 08-2017
Month of Publishing : 08-2017

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greater propensity to metastasize.[1,4-6,8,9,11,16] The 5-year survival rate is 44-69% in clear cell tumors, 82-92% in papillary tumors, and 78-92% in chromophobe tumors.[6-10,17] In advanced disease, a tailored management approach is recommended as the effectiveness of systemic therapy including the specific regime used may be influenced by the RCC subtype.[5,18-29] Studies have suggested that clear cell, papillary, and chromophobe subtypes can be differentiated non-invasively on imaging.[1] Studies have also found that RCCs that develop in patients with end-stage renal disease tend to be less aggressive than RCCs that occur in the general population.[30-32] Hereditary RCCs account for 4% and show a predilection toward early onset, bilaterality, and multicentricity.[32] Around 25-60% of VHL patients develop RCC with the risk of metastasis related to tumor size.[33-35] Birt–Hogg–Dube (BHD) syndrome, an autosomal dominant condition caused by mutations in the folliculin gene, predisposes to cutaneous tumors, oncocytomas and clear cell, papillary, and chromophobe RCCs.[33,36] Recently, it has been discovered that patients with hereditary succinate dehydrogenase mutations are at risk of developing aggressive early-onset RCCs in addition to pheochromocytomas and paraganglioma.[36] Most RCCs are asymptomatic and discovered as unexpected findings on imaging performed for unrelated clinical indications.[37-40] The classic triad of a palpable mass, flank pain, and hematuria is found in 6-10% and portends a more aggressive histology and advanced disease.[41,42] Clear cell RCC typically shows a heterogeneous consistency (secondary to necrosis, cystic change, or hemorrhage) has high signal intensity on T2-weighted magnetic resonance imaging (MRI), and is hypervascular on dynamic contrast-enhanced (DCE) computed tomography or MRI examinations. Most papillary RCCs (PRCC) are detected while at a low grade and small size, show low signal intensity on T2-weighted MRI, and are hypovascular following contrast administration. Chromophobe RCCs may have a homogeneous solid appearance even when large and may exhibit a central stellate scar and spoke-wheel enhancement. Clear cell RCC typically exhibits exophytic growth and has a tendency to be heterogeneous due to intratumoral necrosis, cystic change, or hemorrhage. Interruption of the tumor capsule has also been correlated with high tumor grade.[43] Cystic PRCCs may show hemorrhagic fluid content and internal mural nodules or papillary projections while cystic clear cell RCCs typically show clear fluid content and irregular walls and septations.[44] Chromophobe RCC tends to appear well-circumscribed and homogeneous (cystic change and necrosis are uncommon) even when large and perinephric infiltration and vascular involvement (<4%) are rare.[1,11] Most PRCCs demonstrate low T2 signal intensity.[45,46] In contrast, most clear cell RCCs show high T2 signal intensity.[11,47,48] Several preliminary studies have shown encouraging results in utilizing diffusion-weighted imaging (DWI) for characterizing RCCs into its main subtypes as well as into high-grade and low-grade tumors.[49-52] DWI has been used to differentiate various subgroups of renal masses. MRI is also useful for imaging renal vein and IVC tumor thrombus and the rostral extension (important in pre-operative planning). The presence of enhancement in the thrombus is able to distinguish between bland and tumor thrombus.

Fuhrman *et al*. Grading of RCC

Specifically, grade I tumors consist of cells with small (approximately 10 mm), round, uniform nuclei with inconspicuous or absent nucleoli; grade II tumors have larger nuclei (approximately 15 mm) with irregular morphology and small nucleoli when examined under high power (400 magnification); grade III tumors have even larger nuclei (approximately 20 mm) with irregular outlines and large, prominent nucleoli that are evident even at low power (100 magnification); and grade IV tumors differ from grade III lesions in that they contain bizarre, multilobed nuclei, and heavy chromatin clumps.

The staging of RCC is the most important factor in predicting its prognosis. Staging can follow the TNM staging system, where the size and extent of the tumour (T), involvement of lymph nodes (N), and metastases (M) are classified separately. Furthermore, it can use overall stage grouping into stage I-IV, with the 1997 revision of AJCC described (Table 1).52

Surgical and Further Management

Stage IA

Partial nephrectomy is a widely accepted treatment for RCC tumors of <4 cm in diameter. Nephron-sparing partial nephrectomy - with the objective being the complete surgical extirpation of the tumor while retaining sufficient healthy tissue for adequate renal function - is the preferred treatment option for stage IA. Over the past decade, the clinical indications for partial nephrectomy have been expanded to include most patients with low-stage tumors as studies have demonstrated that partial nephrectomy is as effective a therapeutic option as radical nephrectomy with comparable rates of tumor-free survival and overall survival.

Stage IB

The NCCN recommends that either partial nephrectomy or radical nephrectomy be performed for stage IB tumors.[2] Both techniques show comparable oncologic control.

Stage II and III

The NCCN recommends that radical nephrectomy be performed for stage II and III tumors.[2] Routine adrenalectomy and lymphadenectomy is not advocated in

Table 1: Staging-based on TNM staging system

Stage I	Tumor of a diameter of 7 cm (approx. 2 3⁄4 inches) or smaller, and limited to the kidney. No lymph node involvement or metastases to distant organs
Stage II	Tumor larger than 7.0 cm but still limited to the kidney. No lymph node involvement or metastases to distant organs
Stage III any of the following	Tumor of any size with the involvement of a nearby lymph node but no metastases to distant organs. Tumor of this stage may be with or without spread to fatty tissue around the kidney, with or without spread into the large veins leading from the kidney to the heart
	Tumor with spread to fatty tissue around the kidney and/or spread into the large veins leading from the kidney to the heart, but without spread to any lymph nodes or other organs
Stage IV any of the following	Tumor that has spread directly through the fatty tissue and the fascia ligament-like tissue that surrounds the kidney
	Involvement of more than one lymph node near the kidney
	Involvement of any lymph node not near the kidney
	Distant metastases, such as in the lungs, bone, or brain

the absence of radiologic disease at these sites as it does not improve survival. A laparoscopic approach is favored for stage II tumors while stage III tumors are usually treated by an open approach.[45] (1) Baseline abdominal computed tomography (CT) or MRI within 3-6 months, then CT, MRI, or US every 3-6 months for at least 3 years and then annually up to 5 years, (2) baseline chest CT within 3-6 months after surgery with continued imaging (CT or chest X-ray) every 3-6 months for at least 3 years and then annually up to 5 years.

Stage IV

Renal cell cancers are typically treated with both local and systemic therapy. Local therapy consists of surgery to remove the entire affected kidney and any surrounding cancer. The surgery for Stage IV renal cell cancer is called a radical nephrectomy and involves removing the entire affected kidney, the attached adrenal gland, and any adjacent fat and involved lymph nodes or major blood vessels. Systemic therapy is directed at destroying cancer cells throughout the body and may include chemotherapy, targeted therapy, or immunotherapy. A randomized trial by Motzer *et al*. involving 750 patients with metastatic clear cell RCC showed that patients treated with sunitinib had longer progression free survival and overall survival compared with patients treated with interferon-Î±. Several studies have suggested that vascular endothelial growth factor (VEGF)-TKIs may be less effective in treating papillary and chromophobe RCCs compared with clear cell RCCs.[18,26,47-49] Potential agents include temsirolimus, sorafenib, sunitinib, pazopanib, axitinib, everolimus, bevacizumab, or erlotinib. Preliminary studies have suggested that temsirolimus has efficacy in treating PRCC. The NCCN guidelines for stage 4 patients are as follows:[2] (1) Cases that involve a potentially resectable solitary metastatic site should undergo nephrectomy and surgical metastasectomy, (2) cases that involve a potentially resectable RCC with multiple metastatic sites should undergo cytoreductive nephrectomy in appropriate patients before systemic therapy, and (3) cases with medically or surgically unresectable disease should undergo systemic therapy.

The NCCN suggests that stage IV patients should undergo baseline chest, abdominal, and pelvic imaging by CT or MRI pre-treatment or before observation, followed by repeat imaging every 6-16 weeks as per physician discretion and per patient clinical status.[22] The imaging frequency may be modified depending on the rate of disease change and the sites of active disease.[22]

Aim

The aim of this study was to correlate the histopathology of RCC with the radiological features.

MATERIALS AND METHODS

The total number of renal tumors studied during the 8-year period was 45 cases, of which 25 cases were diagnosed by histopathology as various types of RCC conclusively. This is a retrospective study of renal tumors, diagnosed by histopathology as various types of RCC. All the relevant clinical data of the patients were searched from the ward records. The various radiological features were collected. The clinical features examined included age, gender, smoking history, recent onset hypertension, performance status, and presenting symptoms. A comprehensive health checkup on general conditions were taken and stored in the computer server.

RESULTS

Clear cell carcinoma was the most reported case and 16 cases were reported, other tumors were papillary carcinoma and chromophobe carcinoma in the study covering 8-year period was 45 cases, of which 25 cases were diagnosed by histopathology as various types of RCC conclusively at Thoothukudi Medical College (Tables 2-5).

MRIT1 shows often heterogeneous due to necrosis, hemorrhage, and solid components T2 shows appearances depend on histology clear cell RCC with hyperintense signal and PRCC with hypointense signal. Histopathology section shows clear cell carcinoma with increased vascularity and clear cytoplasm with four grades of Fuhrman nuclei, PRCC showing papillary architecture and chromophobe renal carcinoma showing the neoplastic cells have a fine reticular cytoplasm and bland nuclei some of which have "raisinoid" appearance.

Table 2: Histopathological subtypes of RCC and corresponding grades

Grades	I	II	III	IV	Total
Types					
Clear cell	10	4	2	0	16
Papillary	3	2	1	0	6
Chromophobe	1	2	0	0	3
Total	14	8	3	0	25

RCC: Renal cell carcinoma

Clear Cell RCC

Clear cell renal carcinoma is derived from the proximal convoluted tubule - frequency 60-70%. Most commonly affects male patients in their sixties and seventies. Microscopically, the tumor cells are large, the appearance of the cytoplasm ranging from optically clear, with sharply outlined boundaries. Clear cell renal carcinoma (conventional) arises from proximal convoluted tubules large uniform cells with clear cytoplasm highly vascular.

Table 3: Histopathological age, sex, distribution, and signs and symptoms in the subtypes of RCC

Tumor type	Age group	M: F ratio	Signs and symptoms
Clear cell carcinoma	Females range from 66 to 74 years of age and the age involved in male was 69-74 years	7:9	Ten patients had complaints of fever, hematuria, and flank pain. Two patients had hematuria. Four patients had incidental findings
Papillary carcinoma	Females range from 60 to 74 years of age and the age involved in male was 64-75 years	3:3	Three patients had hematuria. Three patients had incidental findings
Chromophobe carcinoma	Females range from 62 to 70 years of age and the age involved in male was 75 years	2:1	Two patients had hematuria. One patient had incidental findings

RCC: Renal cell carcinoma

Table 4: Correlation study of RCC

Tumor	Radiographic findings	Histopathological Findings
Clear cell renal carcinoma		
Grade I	MRI scan shows heterogeneously enhancing mass at the upper pole of the right kidney with low T1 signal intensity after administration of contrast	Section studied shows clear cell carcinoma with increased vascularity and clear cytoplasm with features of Fuhrman grade 1 nuclei which are uniform, lack, or have inconspicuous nucleoli
Grade II	Renal cell cancer (RCC) lower pole of left kidney. Arterial (40 s) contrast-enhanced CT shows areas of tumor with low intake of contrast surrounded by areas of increased contrast uptake	Section studied shows clear cell carcinoma with features of tumor cells have mild nuclear pleomorphism with occasional small nucleoli and stippled chromatin typical of Fuhrman grade 2 nuclei
Grade III	An arterial phase CT will show enhancement of a tumor thrombus due to neovascularization of the tumour, as in this case	Section studied shows clear cell carcinoma with features of Fuhrman grade 3 nuclei, patchy, and show moderate pleomorphism and large nucleoli
Grade IV	Left heterogenous enhancing mass in the mid-pole of the left kidney. This is well illustrated on this 4 phase renal angiogram CT study	The marked nuclear enlargement of grade 4 nuclei is obvious when compared to the scattered inflammatory cells. Prominent cherry-red nucleoli, with some nuclei harboring two or three nucleoli
PRCC		
Grade I	A well-circumscribed, 31 mm diameter, lesion is identified along the superoposterior cortical pole of the right kidney. It extends deep toward the calyceal system	Section studied shows papillary carcinoma with features of numerous papillae. Note the nuclei are small, have open chromatin, and indistinct nucleoli typical of type 1 PRCC
Grade II	Left heterogenous enhancing mass in the mid-pole of the left kidney. This is well illustrated on this 4 phase renal angiogram CT study	Section studied shows papillary carcinoma a tumor composed of several tubulopapillary structures. There is nuclear enlargement and hyperchromasia, prominent nucleoli, and more abundant basophilic cytoplasm
Chromophobe renal carcinoma	RCC in the lower pole of left kidney. MRI scan shows low signal intensity in T1-weighted image in the tumor area	Section studied shows the neoplastic cells have a fine reticular cytoplasm and bland nuclei some of which have "raisinoid" appearance. The cells have distinct, thick nuclear membranes, and perinuclear halos

PRCC: Papillary renal cell carcinoma

Table 5: Final outcome of the study

Tumor	Surgery done	Cure rate measured after 3 years (%)	Follow-up
Clear cell renal carcinoma	Ten cases were stage I. Four cases were stage II. Two cases were stage III. Partial nephrectomy was done treatment for RCC tumors of<4 cm in diameter. Partial nephrectomy or radical nephrectomy was done for stage IB tumors. Radical nephrectomy be performed for stage II and III tumors	75	Eleven cases attend regular follow-up. One case died of multiple organ failure
PRCC	Three cases were stage I. Two cases were stage II. One case was in stage III. Partial nephrectomy was done treatment for RCC tumors of<4 cm in diameter. Partial nephrectomy or radical nephrectomy was done for stage IB tumors. Radical nephrectomy be performed for stage II and III tumors	50	Three cases are attending the follow-up
Chromophobe RCC	One case were stage I. Two cases were stage II. Partial nephrectomy was done treatment for RCC tumors of less than 4 cm in diameter. Radical nephrectomy was done for stage IB tumors. Radical nephrectomy be performed for stage II	66	Two cases are attending the follow-up

Based on nuclear features, these tumors are graded into four grades. Fuhrman grade 1 nuclei which are uniform, lack, or have inconspicuous nucleoli. The tumor cells have mild nuclear pleomorphism with occasional small nucleoli and stippled chromatin typical of Fuhrman grade 2 nuclei. Fuhrman grade 3 nuclei may be patchy, show moderate pleomorphism, and large nucleoli. The marked nuclear enlargement of grade 4 nuclei is obvious when compared to the scattered inflammatory cells. Prominent cherry-red nucleoli, with some nuclei harboring two or three nucleoli. Sixteen cases clear cell renal carcinoma of were reported. Nine cases involving females and seven case involving males. The age group involved in females range from 66 to 74 years of age and the age involved in male was 69-74 years.

Clear Cell RCC

The histopathology and radiology correlation was perfect in all the cases (Figures 1-8).

PRCC

Majority of tumors occur sporadically, but some may develop in members of families with hereditary. PRCC arises from distal convoluted tubules can be multifocal and bilateral most common form in dialysis-associated RCC type I is sporadic, generally good prognosis type II is inherited, bilateral, and multifocal. Microscopically, type I have papillae covered by a single layer of cuboidal or low columnar cells with scanty cytoplasm and low-grade nuclei and carry a better prognosis than type II tumors. Microscopically, type II are of a higher nuclear grade and contain more than one layer of cells with abundant eosinophilic cytoplasm. Six cases papillary cell renal carcinoma of were reported. Three cases involving females and three case involving males. The age group involved in females range from 60 to 74 years of age and the age involved in male was 64-75 years.

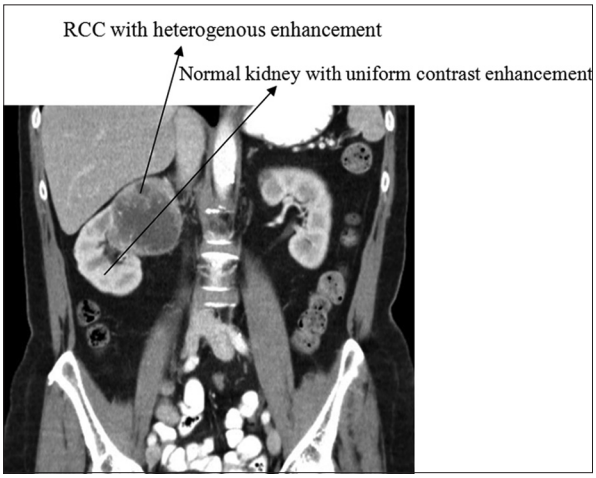

Figure 1: CT scan shows heterogeneously enhancing mass at the upper pole of the right kidney after administration of contrast

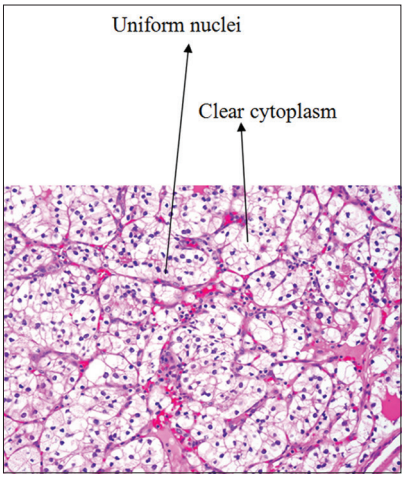

Figure 2: Section studied shows small uniform nuclei with evenly distributed chromatin and the absence of nucleoli, all of which are features of Fuhrman grade 1 nuclei

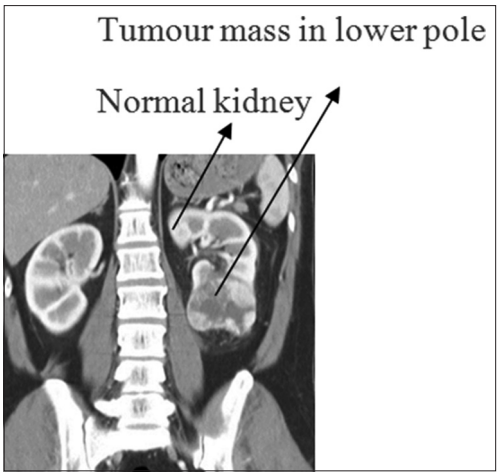

Figure 3: Renal cell cancer (RCC) lower pole of left kidney. Arterial (40sec) contrast enhanced phase

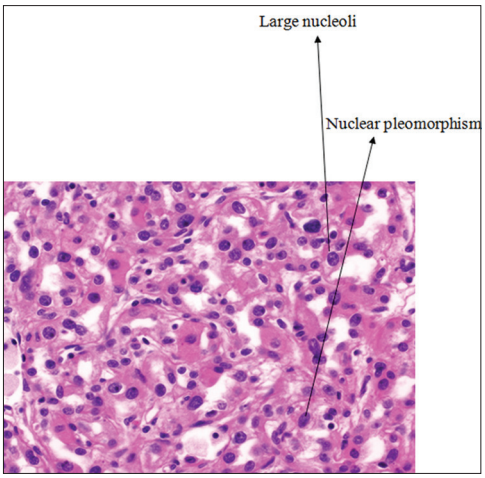

Figure 6: Section studied shows clear cell carcinoma with features of Fuhrman grade 3 nuclei, patchy, and show moderate pleomorphism and large nucleoli.

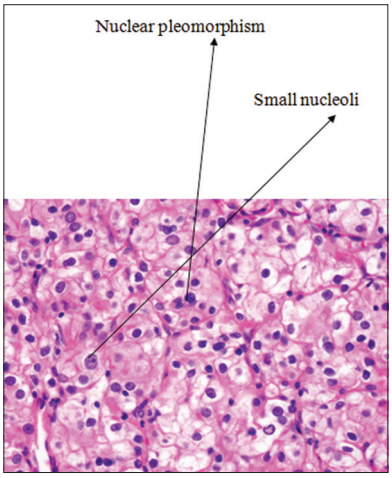

Figure 4: Section studied shows clear cell carcinoma with features of tumor cells have mild nuclear pleomorphism with occasional small nucleoli and stippled chromatin typical of Fuhrman grade 2 nuclei

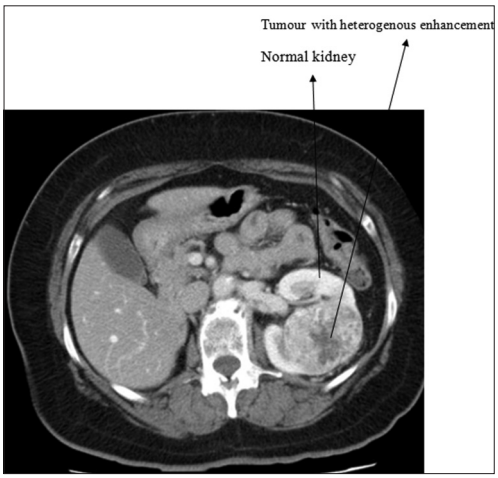

Figure 7: Left heterogenous enhancing mass in the mid-pole of the left kidney. This is well illustrated on this 4 phase renal angiogram CT study

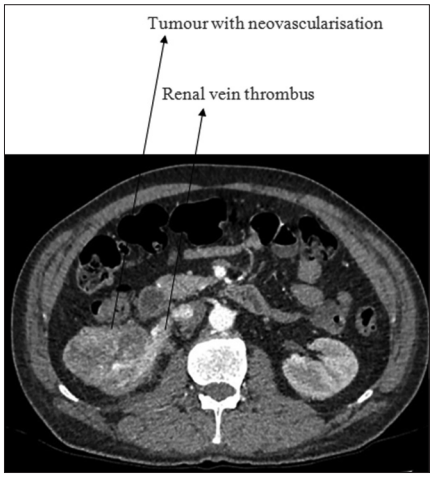

Figure 5: An arterial phase CT will show enhancement of a tumour thrombus due to neovascularisation of the tumour, as in this case.

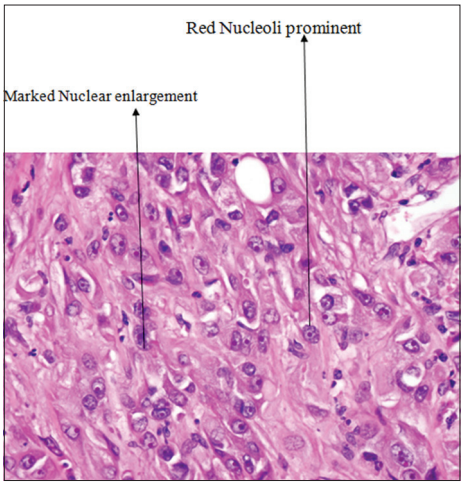

Figure 8: The marked nuclear enlargement of grade 4 nuclei is obviously seen. Prominent cherry-red nucleoli, with some nuclei harbouring two or three nucleoli

PRCC, Type 1

The histopathology and radiology correlation was perfect in all the cases (Figures 9 and 10).

RCC (Type II Papillary)

The histopathology and radiology correlation was perfect in all the cases (Figures 11 and 12).

Chromophobe RCC: The frequency of incidence among overall RCC is chromophobe is derived from the cortical collecting duct. Chromophobe RCC has a much better prognosis than clear cell and PRCC, with 5-year survival rate of >90%. Most cases arise sporadically, whereas some familial cases are associated with BHD syndrome. Microscopically, the neoplastic cells have a fine reticular cytoplasm and bland nuclei some of which have "raisinoid" appearance. The cells have distinct, thick nuclear membranes, and perinuclear halos. Some cells have no nuclei in the plane of section due to the voluminous cytoplasm. This RCC arises from intercalated cells of collecting ducts, similar histologically to renal oncocytomas best prognosis. Three cases were reported. The age group involved in two females range from 62 to 70 years and the age involved in male was 75 years.

The histopathology and radiology correlation was perfect in all the cases (Figures 13 and 14).

Metastatic RCC

The most common sites for metastasis are the lymph nodes, lung, bones, liver, and brain. Average survival time in 2008 for the metastatic form of the disease was under a year and by 2013, this improved to an average of 22 months. From 2007 to 2013, seven

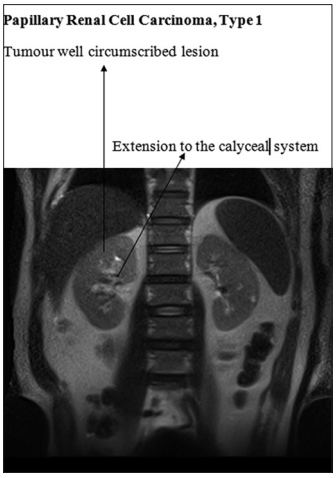

Figure 9: MRI scan shows a well circumscribed, 31mm diameter, lesion is identified along the supero-posterior cortical pole of the right kidney. It extends deep towards the calyceal system

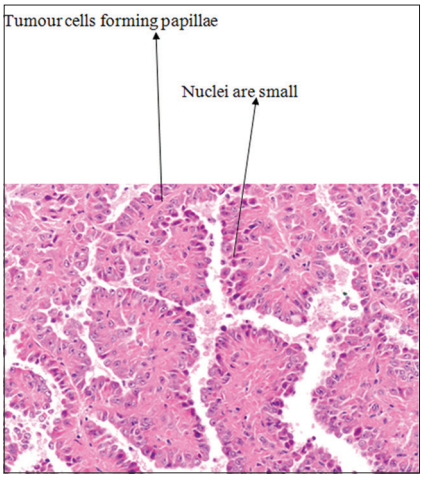

Figure 10: Section studied shows Renal Cell Carcinoma sub type papillary carcinoma with features of numerous papillae, the nuclei are small, and have open chromatin and indistinct nucleoli typical of type 1 papillary renal cell carcinoma

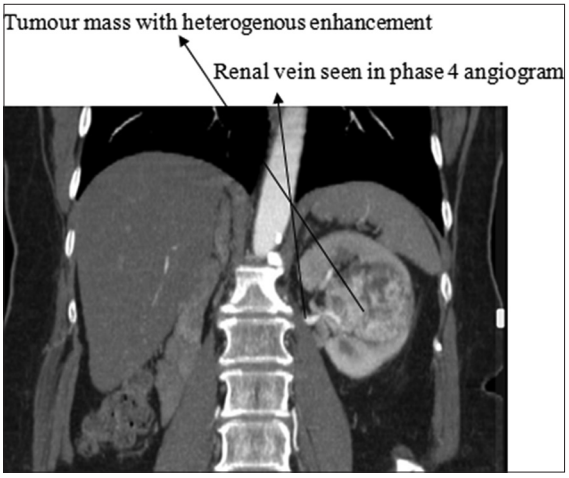

Figure 11: Left heterogenous enhancing mass in the mid-pole of the left kidney. This is well illustrated on this 4 phase renal angiogram CT study

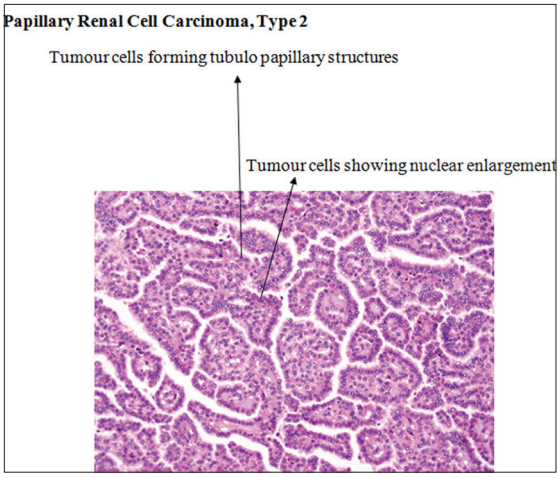

Figure 12: Section studied shows Renal Cell Carcinoma sub type papillary carcinoma a tumour composed of several tubulo-papillary structures. There is nuclear enlargement and hyperchromasia, prominent nucleoli and more abundant eosinophilic cytoplasm

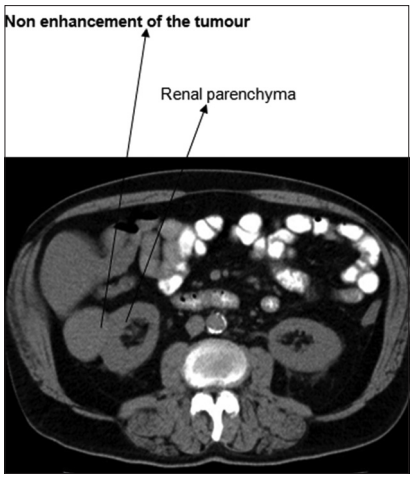

Figure 13: CT shows a 5 cm right renal mass lesion isodense to renal parenchyma. Weak contrast enhancement from 40 to 50 HU on the average

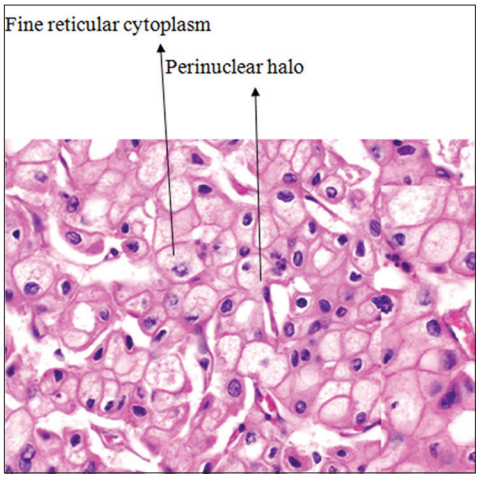

Figure 14: Section studied shows the neoplastic cells have a fine reticular cytoplasm and bland nuclei some of which have 'raisinoid' appearance. The cells have distinct, thick nuclear membranes and perinuclear halos

new treatments have been approved specifically for metastatic RCC (sunitinib, temsirolimus, bevacizumab, sorafenib, everolimus, pazopanib, and axitinib). These new treatments are based on the fact that RCCs are very vascular tumors - they contain a large number of blood vessels. The drugs aim to inhibit the growth of new blood vessels in the tumors, hence slowing growth and in some cases reducing the size of the tumors. Paraneoplastic syndromes are seen in about 25% of RCC patients will develop a paraneoplastic syndrome. They are hypercalcemia (20%), hypertension (20%), and polycythemia: From erythropoietin secretion (~5%), Stauffer Syndrome: Hepatic dysfunction not related to metastases, feminization, and limbic encephalitis.

The prognosis is influenced by several factors, including tumor size, degree of invasion and metastasis, histologic type, and nuclear grade. The PRCC and chromophobe RCC responded moderately to the treatment. The clear cell renal carcinoma did not respond to treatment to the expected mark.

Baseline abdominal CT or MRI within 3-6 months, then CT, MRI, or US every 3-6 months for at least 3 years and then annually up to 5 years; baseline chest CT within 3-6 months after surgery with continued imaging (CT or chest X-ray) every 3-6 months for at least 3 years and then annually up to 5 years.

DISCUSSION

RCC, in our study, the common subtypes were clear cell RCC, PRCC, and chromophobe RCC were the common subtypes observed. The PRCC and chromophobe RCC responded moderately to the treatment. The total cases reported in the department are 25 cases out of which 16 cases are attending follow-up after 3 years. The clear cell renal carcinoma did not respond to treatment to the expected mark. RCC is not a single uniform entity but a group of related neoplasms in which the histologic findings, cytogenetic abnormalities, biologic behavior, and imaging appearances of the tumors are subtype dependent. The 3 main subtypes - clear cell, papillary, and chromophobe - can often be differentiated non-invasively based on characteristic radiologic appearances. Based on the hypothesis that the diffusion of water to and from the cells is highly dependent on the ratio of intracellular and extracellular space, DWI MRI scan is used to differentiate the tumor grades. Organ-sparing treatment can be entertained in selected cases. This ranges from adrenal sparing nephrectomy to partial nephrectomy, performed both open or laparoscopically. In addition, percutaneous radiofrequency or cryoablation (typically under CT guidance), which can be carried out with only local anesthetic and sedation, has been introduced in selected cases. Avastin is a targeted therapy that blocks a protein known as VEGF. Votrient is a targeted oral medication known as an angiogenesis inhibitor. Sutent is an oral multitargeted tyrosine kinase inhibitor that targets proteins responsible for stimulating cancer cell growth. 5-fluorouracil appears to be the most effective chemotherapeutic agent currently available for kidney cancer.

CONCLUSION

Imaging remains the primary tool for the detection and screening of RCC. Perfusion MRI and diffusion MRI play important roles in tumor characterization, prediction, and early detection of therapeutic response and used to differentiate the histology of renal masses in some

preliminary studies. DCE and perfusion MRI can also be used to estimate the morphologic grading of RCC. Histopathology provided the final diagnosis. The prognostic significance of tumor necrosis in clear cell RCC has been confirmed by other groups. More aggressive RCC tumors, which are likely to exhibit necrosis, also harbor increased numbers of tumor-infiltrating T-cells. Tumor necrosis has garnered increasing attention over the past few years, in part because a number of studies have now shown that tumor necrotic tissues can be successfully targeted to facilitate both external tumor imaging and to foster a therapeutic antitumoral response by the host. Coagulative tumor necrosis represents a significant prognostic marker for clear cell and chromophobe RCC. The landscape for RCC treatment has changed dramatically in recent years, with the addition of three new FDA-approved agents this year. This brings our arsenal to seven drugs: Interleukin-2, the VEGF receptor TKI's sunitinib, sorafenib, and pazopanib, the VEGF neutralizing antibody bevacizumab in combination with interferon, and the mTOR inhibitors temsirolimus and everolimus. Pre-operative radiological classification can be used as a supplement to the histopathological grading. This study provides the importance of other medical faculty the surgeon, radiologist, and oncologist to work as a team for a successful outcome. We correlated the histopathological findings with radiological findings. This resulted in perfect correlation between the histopathology study and radiology study.

REFERENCES

1. Eble JN, Sauter G, Epstein JI, Sesterhenn IA. World Health Organization classification of tumours. Pathology and Genetics of Tumours of the Urinary System and Male Genital Organs. Lyon: IARC Press; 2004.
2. Motzer RJ, Agarwal N, Beard C, Bhayani S, Bolger GB, Carducci MA, *et al.* Kidney cancer. J Natl Compr Canc Netw 2011;9:960-77.
3. Siegel RL, Miller KD, Jemal A. Cancer statistics, 2015. CA Cancer J Clin. 2015;65:5-29.
4. Lopez-Beltran A, Scarpelli M, Montironi R, Kirkali Z. 2004 WHO classification of the renal tumors of the adults. Eur Urol 2006;49:798-805.
5. Lopez-Beltran A, Carrasco JC, Cheng L, Scarpelli M, Kirkali Z, Montironi R. 2009 update on the classification of renal epithelial tumors in adults. Int J Urol 2009;16:432-43.
6. Cheville JC, Lohse CM, Zincke H, Weaver AL, Blute ML. Comparisons of outcome and prognostic features among histologic subtypes of renal cell carcinoma. Am J Surg Pathol 2003;27:612-24.
7. Moch H, Gasser T, Amin MB, Torhorst J, Sauter G, Mihatsch MJ. Prognostic utility of the recently recommended histologic classification and revised TNM staging system of renal cell carcinoma: A Swiss experience with 588 tumors. Cancer 2000;89:604-14.
8. Hoffmann NE, Gillett MD, Cheville JC, Lohse CM, Leibovich BC, Blute ML. Differences in organ system of distant metastasis by renal cell carcinoma subtype. J Urol 2008;179:474-7.
9. Beck SD, Patel MI, Snyder ME, Kattan MW, Motzer RJ, Reuter VE, *et al.* Effect of papillary and chromophobe cell type on disease-free survival after nephrectomy for renal cell carcinoma. Ann Surg Oncol 2004;11:71-7.
10. Amin MB, Corless CL, Renshaw AA, Tickoo SK, Kubus J, Schultz DS. Papillary (chromophil) renal cell carcinoma: Histomorphologic characteristics and evaluation of conventional pathologic prognostic parameters in 62 cases. Am J Surg Pathol 1997;21:621-35.
11. Gurel S, Narra V, Elsayes KM, Siegel CL, Chen ZE, Brown JJ. Subtypes of renal cell carcinoma: MRI and pathological features. Diagn Interv Radiol 2013;19:304-11.
12. Sükösd F, Kuroda N, Beothe T, Kaur AP, Kovacs G. Deletion of chromosome 3p14.2-p25 involving the VHL and FHIT genes in conventional renal cell carcinoma. Cancer Res 2003;63:455-7.
13. Lubensky IA, Schmidt L, Zhuang Z, Weirich G, Pack S, Zambrano N, *et al.* Hereditary and sporadic papillary renal carcinomas with c-met mutations share a distinct morphological phenotype. Am J Pathol 1999;155:517-26.
14. Jones TD, Eble JN, Cheng L. Application of molecular diagnostic techniques to renal epithelial neoplasms. Clin Lab Med 2005;25:279-303.
15. Prasad SR, Humphrey PA, Catena JR, Narra VR, Srigley JR, Cortez AD, *et al.* Common and uncommon histologic subtypes of renal cell carcinoma: Imaging spectrum with pathologic correlation. Radiographics 2006;26:1795-806.
16. Amin MB, Amin MB, Tamboli P, Javidan J, Stricker H, de-Peralta Venturina M, *et al.* Prognostic impact of histologic subtyping of adult renal epithelial neoplasms: An experience of 405 cases. Am J Surg Pathol 2002;26:281-91.
17. Young JR, Margolis D, Sauk S, Pantuck AJ, Sayre J, Raman SS. Clear cell renal cell carcinoma: Discrimination from other renal cell carcinoma subtypes and oncocytoma at multiphasic multidetector CT. Radiology 2013;267:444-53.
18. Choueiri TK, Plantade A, Elson P, Negrier S, Ravaud A, Oudard S, *et al.* Efficacy of sunitinib and sorafenib in metastatic papillary and chromophobe renal cell carcinoma. J Clin Oncol 2008;26:127-31.
19. Tannir NM, Thall PF, Ng CS, Wang X, Wooten L, Siefker-Radtke A, *et al.* A phase II trial of gemcitabine plus capecitabine for metastatic renal cell cancer previously treated with immunotherapy and targeted agents. J Urol 2008;180:867-72.
20. Vikram R, Ng CS, Tamboli P, Tannir NM, Jonasch E, Matin SF, *et al.* Papillary renal cell carcinoma: Radiologic-pathologic correlation and spectrum of disease. Radiographics 2009;29:741-54.
21. Upton MP, Parker RA, Youmans A, McDermott DF, Atkins MB. Histologic predictors of renal cell carcinoma response to interleukin-2-based therapy. J Immunother 2005;28:488-95.
22. Motzer RJ, Jonasch E, Agarwal N, Beard C, Bhayani S, Bolger GB, *et al.* Kidney cancer, version 3.2015. J Natl Compr Canc Netw 2015;13:151-9.
23. Zhang J, Lefkowitz RA, Bach A. Imaging of kidney cancer. Radiol Clin North Am 2007;45:119-47.
24. Prando A, Prando D, Prando P. Renal cell carcinoma: Unusual imaging manifestations. Radiographics 2006;26:233-44.
25. Park SB, Cho KS, Lee JH, Jeong YK, Choi SH, Kang BS, *et al.* Unusual manifestations of renal cell carcinoma. Acta Radiol 2008;49:839-47.
26. Sun MR, Ngo L, Genega EM, Atkins MB, Finn ME, Rofsky NM, *et al.* Renal cell carcinoma: Dynamic contrast-enhanced MR imaging for differentiation of tumor subtypes - correlation with pathologic findings. Radiology 2009;250:793-802.
27. Campbell N, Rosenkrantz AB, Pedrosa I. MRI phenotype in renal cancer: Is it clinically relevant? Top Magn Reson Imaging 2014;23:95-115.
28. Ramamurthy NK, Moosavi B, McInnes MD, Flood TA, Schieda N. Multiparametric MRI of solid renal masses: Pearls and pitfalls. Clin Radiol 2015;70:304-16.
29. Lee-Felker SA, Felker ER, Tan N, Margolis DJ, Young JR, Sayre J, *et al.* Qualitative and quantitative MDCT features for differentiating clear cell renal cell carcinoma from other solid renal cortical masses. AJR Am J Roentgenol 2014;203:W516-24.
30. Breda A, Lucarelli G, Rodriguez-Faba O, Guirado L, Facundo C, Bettocchi C, *et al*. Clinical and pathological outcomes of renal cell carcinoma (RCC) in native kidneys of patients with end-stage renal disease: A long-term comparative retrospective study with RCC diagnosed in the general population. World J Urol 2015;33:1-7.
31. Shrewsberry AB, Osunkoya AO, Jiang K, Westby R, Canter D, Pattaras J, *et al.* Renal cell carcinoma in patients with end-stage renal disease has favorable overall prognosis. Clin Transplant 2014;28:211-6.
32. Neuzillet Y, Tillou X, Mathieu R, Long JA, Gigante M, Paparel P, *et al.* Renal cell carcinoma (RCC) in patients with end-stage renal disease exhibits many favourable clinical, pathologic, and outcome features compared with RCC in the general population. Eur Urol 2011;60:366-73.
33. Byler TK, Bratslavsky G. Hereditary renal cell carcinoma: Genetics,

clinical features, and surgical considerations. World J Urol 2014;32:623-30.
34. Rosner I, Bratslavsky G, Pinto PA, Linehan WM. The clinical implications of the genetics of renal cell carcinoma. Urol Oncol 2009;27:131-6.
35. Lonser RR, Glenn GM, Walther M, Chew EY, Libutti SK, Linehan WM, *et al.* von Hippel-Lindau disease. Lancet 2003;361:2059-67.
36. Ricketts CJ, Shuch B, Vocke CD, Metwalli AR, Bratslavsky G, Middelton L, *et al.* Succinate dehydrogenase kidney cancer: An aggressive example of the Warburg effect in cancer. J Urol 2012;188:2063-71.
37. Escudier B, Eisen T, Porta C, Patard JJ, Khoo V, Algaba F, *et al.* Renal cell carcinoma: ESMO Clinical Practice Guidelines for diagnosis, treatment and follow-up. Ann Oncol 2012;23 Suppl 7:vii65-71.
38. Bellmunt J, Puente J, Garcia de Muro J, Lainez N, Rodríguez C, Duran I; Spanish Society for Medical Oncology. SEOM clinical guidelines for the treatment of renal cell carcinoma. Clin Transl Oncol 2014;16:1043-50.
39. Krabbe LM, Bagrodia A, Margulis V, Wood CG. Surgical management of renal cell carcinoma. Semin Intervent Radiol 2014;31:27-32.
40. Volpe A, Panzarella T, Rendon RA, Haider MA, Kondylis FI, Jewett MA. The natural history of incidentally detected small renal masses. Cancer 2004;100:738-45.
41. Pastore AL, Palleschi G, Silvestri L, Moschese D, Ricci S, Petrozza V, *et al.* Serum and urine biomarkers for human renal cell carcinoma. Dis Markers 2015;2015:251403.
42. Lee CT, Katz J, Fearn PA, Russo P. Mode of presentation of renal cell carcinoma provides prognostic information. Urol Oncol 2002;7:135-40.
43. Pedrosa I, Chou MT, Ngo L, H Baroni R, Genega EM, Galaburda L, *et al.* MR classification of renal masses with pathologic correlation. Eur Radiol 2008;18:365-75.
44. Muglia VF, Prando A. Renal cell carcinoma: Histological classification and correlation with imaging findings. Radiol Bras 2015;48:166-74.
45. Kim JK, Kim TK, Ahn HJ, Kim CS, Kim KR, Cho KS. Differentiation of subtypes of renal cell carcinoma on helical CT scans. AJR Am J Roentgenol 2002;178:1499-506.
46. Pedrosa I, Alsop DC, Rofsky NM. Magnetic resonance imaging as a biomarker in renal cell carcinoma. Cancer 2009;115 10 Suppl:2334-45.
47. Yoshimitsu K, Irie H, Tajima T, Nishie A, Asayama Y, Hirakawa M, *et al.* MR imaging of renal cell carcinoma: Its role in determining cell type. Radiat Med 2004;22:371-6.
48. Oliva MR, Glickman JN, Zou KH, Teo SY, Mortelé KJ, Rocha MS, *et al.* Renal cell carcinoma: t1 and t2 signal intensity characteristics of papillary and clear cell types correlated with pathology. AJR Am J Roentgenol 2009;192:1524-30.
49. Goyal A, Sharma R, Bhalla AS, Gamanagatti S, Seth A, Iyer VK, *et al.* Diffusion-weighted MRI in renal cell carcinoma: A surrogate marker for predicting nuclear grade and histological subtype. Acta Radiol 2012;53:349-58.
50. Choi YA, Kim CK, Park SY, Cho SW, Park BK. Subtype differentiation of renal cell carcinoma using diffusion-weighted and blood oxygenation level-dependent MRI. AJR Am J Roentgenol 2014;203:W78-84.
51. Wang H, Cheng L, Zhang X, Wang D, Guo A, Gao Y, *et al.* Renal cell carcinoma: Diffusion-weighted MR imaging for subtype differentiation at 3.0 T. Radiology 2010;257:135-43.
52. Lassel EA, Rao R, Schwenke C, Schoenberg SO, Michaely HJ. Diffusion-weighted imaging of focal renal lesions: A meta-analysis. Eur Radiol 2014;24:241-9.

How to cite this article: Kannan P, Revathi R. A Radiology-pathological Correlation of Renal Cell Carcinoma in a Tertiary Care Hospital - A Retrospective Study. Int J Sci Stud 2017;5(5):14-23.

Source of Support: Nil, **Conflict of Interest:** None declared.