

MRI of Small Renal Masses

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The serendipitous discovery of renal cell carcinoma (RCC) is increasing with the proliferation of cross-sectional imaging studies (1). This discovery can lead to management quandaries as the natural history and growth rates of incidentally detected small renal masses (SRM) are variable (2). For example, early detection and surgical resection has not translated into reduced mortality (3), implying that many small, potentially indolent renal masses are subjected to unnecessary escalation of care.

There is a growing body of evidence indicating indolent oncologic behavior of most incidentally detected SRMs. On average, these tumors are smaller than those detected in symptomatic patients, and although most are initially diagnosed as malignant RCC, up to 20-30% of them are benign (4-6). When confirmed malignant, SRMs have a lower histologic grade than that of larger renal tumors (4, 7-9). Similarly, malignant SRMs demonstrate low metastatic potential with only 1.8% of tumors <4cm presenting with metastatic disease (10). Furthermore, SRMs have a slow growth rate (mean 0.1- 0.86 cm per year), with as many as 15-53% demonstrating no significant growth over time (11-13). These findings suggest that many SRMs have an indolent course, and have stimulated debate as to the appropriate management of these patients.

Active surveillance approaches have been suggested to avoid unnecessary surgical and ablation procedures, especially in the elderly and infirm. However, the most important reason for resistance to active surveillance as a clinical paradigm in SRM management centers on the lack of reliable predictors of oncologic behavior. Furthermore, the different histopathologic subtypes in RCC differ in their prognosis and biologic behavior (14, 15), as well as in their response to available therapies (16, 17). Thus, accurate preoperative diagnosis may be important for the selection of the best treatment option in patients with SRMs.

Magnetic Resonance imaging (MRI) is a useful tool for the characterization of renal masses. In this case-based presentation, we will review an MRI imaging protocol for evaluation of renal masses using standard T1- and T2-weighted images (18). The advantages of 3D spoiled-gradient echo T1-weighted images over 2D approaches will be emphasized. The MR imaging findings that allow for accurate characterization of malignant renal neoplasms and their distinction from non-neoplastic lesions and benign neoplasms will be presented.

Non-neoplastic lesions (e.g. complex cysts, hemorrhagic cysts) and benign neoplasms (e.g. angiomyolipoma, oncocytoma) can mimic renal cancer and their preoperative distinction is often challenging, particularly in the evaluation of SRMs. We will discuss the MR imaging findings that are associated to these entities and that may facilitate their diagnosis, as well as review the use of MRI to select potential candidates for percutaneous biopsy to avoid an unnecessary surgical resection.

The correlation between the MRI appearance of renal masses, or MRI phenotype, and their histologic characterization will be discussed. The use of these MRI features may also be helpful to identify patients with masses more suitable for active surveillance. For example, masses with homogeneous low signal intensity on T2-weighted images are less likely to grow when followed over time (19). In some other cases, the MRI phenotype may predict an aggressive behavior. For example, the infiltrative MRI phenotype in pRCC is a prognostic feature associated with higher likelihood of developing metastatic disease, independent of tumor type, grade, and stage (20). Large size, intratumoral necrosis, retroperitoneal vascular collaterals, and renal vein thrombosis on MRI predict more aggressive histology (i.e. high Fuhrman grade) in ccRCC (21). The sensitivity and specificity of a classification system based on MR phenotypes for diagnosing ccRCC and pRCC is 92% and 83%, and 80% and 94%, respectively. Detection of intratumoral lipids in a heterogeneous renal mass by chemical shift MRI is moderately sensitive (50%-82%) although highly specific (90%-97%) for characterizing ccRCC versus other histologic subtypes of RCC (21-23). Although this feature can also be seen in AMLs containing minimal fat, these lesions tend to exhibit homogeneous low signal intensity on T2-weighted images (24).

The most common subtypes of kidney cancer (clear cell, papillary, and chromophobe) demonstrate different patterns of enhancement when assessed with dynamic contrast enhanced MRI. These subtypes can be distinguished based upon the corticomedullary phase percentage enhancement. The use of a threshold value of 84% enhancement in the corticomedullary phase allows differentiation of clear cell and papillary renal cell carcinoma with sensitivity of 93% and specificity of 96% (25).

During this talk, we will review examples of SRM that illustrate the role of MRI in the staging of these lesions as well as the development of a surgical or percutaneous ablation plan.

References:

1. Jayson M, Sanders H. Increased incidence of serendipitously discovered renal cell carcinoma. *Urology*. 1998;51(2):203-5.
2. Volpe A, Panzarella T, Rendon RA, Haider MA, Kondylis FI, Jewett MA. The natural history of incidentally detected small renal masses. *Cancer*. 2004;100(4):738-45.
3. Russo P, Jang TL, Pettus JA, et al. Survival rates after resection for localized kidney cancer: 1989 to 2004. *Cancer*. 2008;113(1):84-96.

4. Frank I, Blute ML, Cheville JC, Lohse CM, Weaver AL, Zincke H. Solid renal tumors: an analysis of pathological features related to tumor size. *The Journal of urology*. 2003;170(6 Pt 1):2217-20.
5. Gill IS, Matin SF, Desai MM, et al. Comparative analysis of laparoscopic versus open partial nephrectomy for renal tumors in 200 patients. *The Journal of urology*. 2003;170(1):64-8.
6. Remzi M, Katzenbeisser D, Waldert M, et al. Renal tumour size measured radiologically before surgery is an unreliable variable for predicting histopathological features: benign tumours are not necessarily small. *BJU international*. 2007;99(5):1002-6.
7. Konnak JW, Grossman HB. Renal cell carcinoma as an incidental finding. *The Journal of urology*. 1985;134(6):1094-6.
8. Thompson IM, Peek M. Improvement in survival of patients with renal cell carcinoma--the role of the serendipitously detected tumor. *The Journal of urology*. 1988;140(3):487-90.
9. Sweeney JP, Thornhill JA, Graiger R, McDermott TE, Butler MR. Incidentally detected renal cell carcinoma: pathological features, survival trends and implications for treatment. *British journal of urology*. 1996;78(3):351-3.
10. Vander Heiden MG, Cantley LC, Thompson CB. Understanding the Warburg effect: the metabolic requirements of cell proliferation. *Science*. 2009;324(5930):1029-33.
11. Lane BR, Tobert CM, Riedinger CB. Growth kinetics and active surveillance for small renal masses. *Curr Opin Urol*. 2012;22(5):353-9.
12. Mason RJ, Abdolell M, Trottier G, et al. Growth kinetics of renal masses: analysis of a prospective cohort of patients undergoing active surveillance. *European urology*. 2011;59(5):863-7.
13. Patel N, Cranston D, Akhtar MZ, et al. Active surveillance of small renal masses offers short-term oncological efficacy equivalent to radical and partial nephrectomy. *BJU international*. 2012;110(9):1270-5.
14. Amin MB, Tamboli P, Javidan J, et al. Prognostic impact of histologic subtyping of adult renal epithelial neoplasms: an experience of 405 cases. *The American journal of surgical pathology*. 2002;26(3):281-91.
15. Bostwick DG, Murphy GP. Diagnosis and prognosis of renal cell carcinoma: highlights from an international consensus workshop. *Semin Urol Oncol*. 1998;16(1):46-52.
16. Lam JS, Shvarts O, Leppert JT, Figlin RA, Belldegrun AS. Renal cell carcinoma 2005: new frontiers in staging, prognostication and targeted molecular therapy. *The Journal of urology*. 2005;173(6):1853-62.
17. Schrader AJ, Olbert PJ, Hegele A, Varga Z, Hofmann R. Metastatic non-clear cell renal cell carcinoma: current therapeutic options. *BJU Int*. 2008;101(11):1343-5.
18. Zhang JB, Pedrosa I, Rofsky NM. MR techniques for renal imaging. *Radiologic Clinics of North America*. 2003;41(5):877-+.
19. Dodelzon K, Mussi TC, Babb JS, Taneja SS, Rosenkrantz AB. Prediction of growth rate of solid renal masses: utility of MR imaging features--preliminary experience. *Radiology*. 2012;262(3):884-93.

20. Rosenkrantz AB, Sekhar A, Genega EM, et al. Prognostic implications of the magnetic resonance imaging appearance in papillary renal cell carcinoma. *European Radiology*. 2013;23(2):579-87.
21. Pedrosa I, Chou MT, Ngo L, et al. MR classification of renal masses with pathologic correlation. *European Radiology*. 2008;18(2):365-75.
22. Outwater EK, Bhatia M, Siegelman ES, Burke MA, Mitchell DG. Lipid in renal clear cell carcinoma: detection on opposed-phase gradient-echo MR images. *Radiology*. 1997;205(1):103-7.
23. Yoshimitsu K, Honda H, Kuroiwa T, et al. MR detection of cytoplasmic fat in clear cell renal cell carcinoma utilizing chemical shift gradient-echo imaging. *J Magn Reson Imaging*. 1999;9(4):579-85.
24. Hindman N, Ngo L, Genega EM, et al. Angiomyolipoma with minimal fat: can it be differentiated from clear cell renal cell carcinoma by using standard MR techniques? *Radiology*. 2012;265(2):468-77.
25. Sun MRM, Ngo L, Genega EM, et al. Renal Cell Carcinoma: Dynamic Contrast-enhanced MR Imaging for Differentiation of Tumor Subtypes-Correlation with Pathologic Findings. *Radiology*. 2009;250(3):793-802.