Computed Tomography of Renal Masses in Adults

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Renal masses are a common incidental finding in abdominal imaging. They typically are benign cysts but sometimes are kidney cancers—most frequently renal cell carcinoma (RCC) in adults. Alongside noncontrast computed tomography images, multiphase contrast-enhanced computed tomography (CECT) supports rapid, accurate differentiation of renal cysts from potential kidney cancers and can help with lesion characterization, staging, and surgical planning. CECT also can help physicians assess RCC histology or tumor subtype. This article discusses the biology, genetics, diagnostic imaging, classification, and staging of renal masses, with an emphasis on CECT kidney imaging, renal cysts, and RCC.

Renal masses are surprisingly common, with as many as 50% of adults older than 50 years harboring a benign or malignant lesion in one or both kidneys. Most renal masses are discovered incidentally during the course of other abdominal diagnostic imaging examinations. These incidental findings are typically benign cysts, but renal malignancies require diagnosis, characterization, staging, and either active surveillance or surgical planning.\(^1\)

Computed tomography (CT) plays a central role in differentiating potentially malignant renal masses from benign masses and in classifying, characterizing, and staging these masses. CT also is used widely in guided biopsy and aspiration procedures, active surveillance of suspicious masses over time, surgical planning, and post-treatment monitoring. Techniques have been developed and refined for rapid, high-quality diagnostic imaging of renal masses with the use of succeeding generations of CT equipment and other imaging modalities.\(^2\)\(^3\) Multidetector CT (MDCT) scanners are standard equipment for performing contrast-enhanced CT (CECT) of the kidneys. Multiphase CECT, which captures image data as contrast material passes through various renal tissues, is the standard approach to evaluating renal masses.

Anatomy and Physiology

The kidneys are paired, fist-sized organs positioned on either side of the spinal column in the retroperitoneum against the posterior wall of the abdomen, immediately below the rib cage and behind the abdominal cavity.\(^4\)\(^5\) The left kidney is slightly larger and is situated somewhat higher than the right kidney; it is surrounded by the spleen, stomach, pancreas, and small bowel.\(^6\)\(^7\)

The adrenal glands are situated immediately superior to the kidneys, and each kidney is supplied by microvasculature furnished by the renal
Functionally, each of the kidneys is composed of approximately 1 million filtering units called nephrons (see Figure 2). Each nephron contains...
a corpuscle and a tubule. Each corpuscle contains a dense network of capillaries known as the glomerulus, supplied by the renal artery; the glomerulus supplies blood to the Bowman capsule (sometimes called the nephron capsular space), in which blood cells, proteins, and other biomolecules are filtered from the blood. Filtered blood exits the Bowman capsule through efferent arterioles (see Figure 3). Each tubule is composed largely of epithelial and cuboidal cells and consists of 3 regions:

- The proximal nephron tubule, which exits the Bowman capsule and serves reabsorption functions.
- The loop of Henle.
- The distal nephron tubule, a coiled region where the tubule drains into collecting tubules or ducts, which filters water from the Bowman capsule to maintain fluid homeostasis and passes waste to the renal pelvis and ureters for excretion in the urine. The distal nephron tubule responds to the antidiuretic hormone by adjusting its wall permeability to resorb more water (rendering urine more concentrated) or less water (diluting urine).

As part of the endocrine system, renal endocrine tissues secrete hormones involved in blood cell production and regulation of arterial diameter and blood pressure, vitamin D activation and bone metabolism, and salt and fluid balance (see Box 7). These crucial life functions are disrupted in kidney failure, which affects 1 in 2000 adults and can result in hypertension, fluid imbalances and tissue edema, altered urinary output, cognitive changes or seizures, and bone disease.

When plasma osmolality is too high (a state of dehydration), an antidiuretic hormone causes the kidneys to reabsorb more water to increase the volume of circulating plasma in the body. Two renal systems help maintain body plasma volume: urea recycling and single-effect (nephron loop) systems. When blood plasma volume is low, urea is recycled into the renal nephron instead of being excreted to the ureters.

### Renal Cysts

Cysts are the most common renal mass found during abdominal imaging. A kidney with 3 or more cysts is referred to as a cystic kidney. Cysts are fluid-filled masses arising from a nephron or collecting tubule. They usually are asymptomatic and benign but sometimes represent disease processes ranging from infection or lithium-induced nephrotoxicity to inherited kidney diseases and life-threatening kidney cancers. The presence of renal cysts might be associated with hypertension and impaired renal function. Cysts are therefore an indication for kidney disease screening.

Renal cysts might be developmental, heritable, or acquired and can be simple or complex. The cysts are categorized as malignant or benign under the Bosniak classification system, which is based on CT scan findings. Uncomplicated, simple renal cysts are the most common renal masses, occurring in up to 27% of adults older than 50 years; simple cysts are found alone in 70% to 80% of cases, and in 20% to 30% of cases they occur as multiple cysts. Simple cysts can be unilateral or bilateral. Because simple cysts are benign and asymptomatic, they often are found incidentally during ultrasonographic or CT examinations of the abdomen. Simple cysts are small (< 3 cm diameter) and tend to be oval or round, with thin walls composed of a single layer of epithelial cells and no calcification. Extraparenchymal (ie, peripelvic) simple cysts develop in the renal sinus, adjacent to the renal pelvis (see Figure 4). These renal sinus cysts tend to be found incidentally, as they are benign and asymptomatic. In contrast to benign simple
cysts, complex renal cysts are more likely to involve complications, such as infection or hemorrhage, or to involve an elevated risk of malignancy. Surveillance or surgical excision could be indicated depending on the degree of cyst complexity and corresponding risk of malignancy.\textsuperscript{13,15}

**Developmental Renal Cysts**

Developmental cysts, which usually are benign, might have genetic risk factors or might result from errors in the development of the renal tissues. Details remain unclear, but in some cases, including multicystic dysplastic kidney disease, developmental cystic lesions appear to have their roots in early embryogenesis.\textsuperscript{17} The cause of the abnormal development could be genetic or result from a defect in either an inducer or responder tissue. Along with multicystic dysplasia, examples of developmental cysts include medullary sponge kidney and pyelocaliceal cysts.\textsuperscript{12,13}

**Genetic Cystic Renal Diseases**

Individuals with a family history of certain renal cystic diseases might require genetic screening and counseling.\textsuperscript{12,13} Examples of genetic cystic renal diseases include von Hippel-Lindau (VHL) syndrome, polycystic kidney disease, and tuberous sclerosis.\textsuperscript{12,13}

VHL syndrome is a genetic disorder that leads to the formation of fluid-filled cysts throughout the body, including the kidneys, and increases the risk of renal cell carcinoma (RCC). Polycystic kidney disease (PKD) is a common and life-threatening heritable genetic syndrome associated with mutations in the polycystic kidney disease 1 (PKD1) gene on chromosome 16. Patients with PKD develop multiple fluid-filled cysts throughout both kidneys. The disease is caused by 2 genetic variants: autosomal dominant PKD, representing 80\% of cases and occurring in up to 1 per 500 live births, and a rarer autosomal recessive PKD.\textsuperscript{14,18} PKD leads to enlarged kidneys and liver damage. Tuberous sclerosis is associated with both benign angiomylipomas and cysts in the kidneys and other organs; renal masses usually are present by late childhood.\textsuperscript{14}

Although cysts rarely are malignant, heritable cystic renal diseases can be life threatening. As patients age and more functional nephron tissue is displaced by cysts and associated benign neoplasms, end-stage renal disease can ensue.\textsuperscript{14}

**Acquired Cystic Kidney Disease**

Acquired cystic kidney disease is a common, bilateral, and progressive multicystic condition affecting patients undergoing dialysis with end-stage renal disease.\textsuperscript{13} The longer a patient has been undergoing dialysis, the more likely he or she is to develop the disease, and the more likely the disease is to be severe. Twenty percent of patients beginning dialysis already have been diagnosed with acquired cystic kidney disease, and by the time they have been undergoing dialysis for 8 years, that percentage increases to 90\%.\textsuperscript{13,19} Patients with acquired cystic kidney disease also face a 7\% chance of developing RCC, usually involving multiple and bilateral tumors.\textsuperscript{13} Therefore, regular RCC screening with CT imaging is recommended after several years of dialysis, depending on a patient’s condition.

**Renal Cell Carcinoma**

RCC involves tumorigenesis in the renal tubules.\textsuperscript{20} It can result from sporadic genetic changes in renal cells or hereditary genetic syndromes.\textsuperscript{11} The primary

![Figure 4. Peripelvic cysts. Coronal excretory-phase maximum intensity projection computed tomography (CT) image shows the contrast-filled collecting system surrounded by multiple cysts in the renal pelvis. Used with permission from Wood CG, Stromberg LJ, Harmath CB, et al. CT and MR imaging for evaluation of cystic renal lesions and diseases. Radiographics. 2015;35(1):136.](308CT)
inherited RCC syndromes involve autosomal-dominant mutations; these include VHL, hereditary leiomyomatosis and RCC, hereditary papillary RCC, and Birt-Hogg-Dubé syndrome.\textsuperscript{21}

There are 3 main subtypes of RCC. Conventional or clear cell RCC, so named because of cells’ microscopic appearance, is the most common subtype, representing 70% of cases.\textsuperscript{22,23} Papillary RCC is the second-most common form, representing 10% of RCC diagnoses,\textsuperscript{21,22} and chromophobe RCC represents 5% of cases.\textsuperscript{22,24}

Rarer forms of RCC include multilocular cystic renal cell carcinoma, sometimes called \textit{multilocular clear cell RCC}, a cystic tumor type (3% of cases); collecting duct RCC (<1% of cases); medullary RCC; granular cell RCC; and tubulocystic carcinoma.\textsuperscript{22,23,25} Sarcomatoid (or spindle) RCC tumors can arise from any of these subtypes.\textsuperscript{22}

Some researchers classify oncocytes as benign rather than as a subtype of RCC because they rarely metastasize.\textsuperscript{23,26} However, these tumors can become so large that surgery is required, and some benign renal masses can occur alongside RCC and might transform into RCC.\textsuperscript{23,27}

In some cases, RCCs are deemed unclassified because more than one RCC subtype cell is apparent in a tumor.\textsuperscript{24}

**Epidemiology and Etiology**

Among the benign renal neoplasms, the fibromas, lipomas, and metanephrin adenomas are relatively rare.\textsuperscript{27} Angiomyolipomas are far more common and occur either in a sporadic or isolated unilateral form or a bilateral form associated with tuberous sclerosis and tuberous sclerosis 1 (\textit{TSC1}) or tuberous sclerosis 2 (\textit{TSC2}) mutations.\textsuperscript{27} These genes occur in close genomic proximity to the \textit{PKD1} gene and cause tuberous sclerosis.

Relatively little research has been published about benign renal neoplasms’ epidemiology and risk factors. Women are more likely than men to develop angiomyolipomas, fibromas, lipomas, and metanephrin adenomas.\textsuperscript{27} Angiomyolipomas linked to tuberous sclerosis tend to be associated with enlarged kidneys and hemorrhage and might harbor intramass aneurysms (see Figure 5).\textsuperscript{14} Metanephrin adenomas might be more common among women with polycythemia (overproduction of red blood cells).\textsuperscript{27}

**Figure 5. Tuberous sclerosis.** Axial contrast-enhanced CT (CECT) image shows the kidneys to be enlarged and replaced by numerous cysts and soft-tissue renal masses. In the right kidney, a focus of fat (arrow in A) and a small intralemion aneurysm (arrow in B) are findings consistent with angiomyolipomas. The right kidney was subsequently removed because of hemorrhage and found upon examination to have been replaced by cysts and angiomyolipomas. The angiomyolipomas were composed mostly of smooth-muscle elements and very little fat. Reprinted with permission from Wood CG, Stromberg LJ, Harmath CB, et al. CT and MR imaging for evaluation of cystic renal lesions and diseases. Radiographics. 2015;35(1):140.
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Other than the heritable cystic diseases, relatively little is known about cyst epidemiology. More is known about the epidemiology of renal malignancies, which readily metastasize to distant organs and represent a life-threatening diagnosis. Malignant kidney tumors are frequently asymptomatic until they are advanced.\(^{29}\) However, since 1990, more asymptomatic early-stage kidney cancers are being diagnosed incidentally in the course of other imaging examinations.\(^{28}\)

The National Cancer Institute (NCI) estimated 61 560 Americans would be newly diagnosed with kidney and renal pelvis cancers in 2015, accounting for 3.7% of all new cancer cases.\(^{11}\) The median age of patients at diagnosis is 64 years.\(^{11}\) The 5-year survival rates are higher than 90% overall for those diagnosed when kidney cancers are localized but less than 12% for patients diagnosed after metastatic spread to distant organs.\(^{11}\) Kidney cancers account for 2.4% of cancer deaths.\(^{11}\)

Among adults, transitional cell carcinomas (TCCs) represent up to 10% of renal malignancies; these tumors occur in the renal pelvis rather than in the kidney parenchyma, which is where RCC tumors occur.\(^{24}\) RCC represents up to 90% of primary kidney cancers.\(^{20}\) It tends to spread through the capsule into the fat layer or beyond the kidney through renal vasculature, metastasizing into the adjacent adrenal gland, or to the liver, bones, lungs, or brain.\(^{29}\) Prognosis is poorer for people with lower socioeconomic status, possibly because these patients often receive a diagnosis with more advanced tumors.\(^{30}\) Overall, most patients (52%) with RCC have nonmetastatic disease at the time of diagnosis.\(^{28}\) The 5-year cancer-specific survival rates vary by tumor stage, from 97% for patients diagnosed with low-risk, localized tumors to 81% for intermediate-risk cases and 62% for high-risk cases.\(^{24}\) For patients diagnosed with metastatic disease, 5-year cancer-specific survival rates range from 8% to 41%.\(^{24}\)

Men are more likely to develop RCC, and among patients diagnosed with RCC, men have a lower survival rate than do women.\(^{39}\) Racial disparities exist in the incidence, treatment outcomes, and survival rates for RCC.\(^{35,39}\) Blacks have higher incidence rates and lower survival rates than whites, even after statistically controlling for sex, age, RCC subtype, tumor stage at diagnosis, and treatment.\(^{35}\) Among patients undergoing radical nephrectomy for RCC, black patients are more likely to experience major postoperative complications than white patients.\(^{32}\)

American Indians face an even higher risk of developing and dying from kidney cancers than black patients.\(^{34}\) Incidence rates for kidney and renal pelvis cancers are highest among blacks and American Indian populations, particularly men. Specifically, the rates are 25.1 per 100 000 black men and 24.8 per 100 000 for American Indian men.\(^{11}\) American Indians are more likely to die of kidney and renal pelvis cancers, with mortality rates of 8.7 per 100 000 per year compared with 5.7 per 100 000 for men of all races. The rates for American Indian women average 4.7 deaths per 100 000 each year compared with 2.5 per 100 000 for women of all races.\(^{11}\)

The incidence of kidney cancer rose more rapidly among American Indians than among whites between 1999 and 2009.\(^{34}\) During those years, the kidney cancer mortality rate among whites declined slightly and remained stable among American Indians.\(^{34}\) These trends reflect disparities in cancer incidence and mortality among American Indians, although disparities vary geographically and by cancer type.\(^{35}\) Such disparities might partly reflect different exposures to dietary and environmental risk factors for kidney and other cancers.\(^{35}\)

Well-established and modifiable risk factors for kidney cancer include cigarette smoking, obesity, and hypertension.\(^{36,37}\) Cigarette smoking increases the risk of developing kidney cancer by about 50% for men and 20% for women, with a strong dose-response pattern of increased risk with increased number of cigarettes smoked.\(^{36}\) It is believed that carbon monoxide from cigarette smoke increases RCC risk by inducing chronic renal hypoxia.\(^{36}\) Chemicals found in cigarette smoke also are genotoxic, meaning they can cause genetic mutations including those associated with tumorigenesis.\(^{36}\)

RCC risk appears to be reduced among people with diets rich in fruits and vegetables and among women with diets low in red and processed meat.\(^{36,38}\) More than half of Americans are overweight or obese, and obesity might be responsible for as much as 40% of RCC diagnoses in the United States.\(^{38}\) As with cigarettes, body weight above healthy levels has a dose-response relationship with RCC risk: for every additional 5 kg/m\(^2\)
increase in body mass index, the risk of RCC increases by 24% for men and 34% for women.³⁸ The biological basis for this association between RCC risk and obesity remains unclear, however.³⁹ In addition, one analysis of more than 1 million military recruits in Israel suggested that overweight status during late adolescence significantly increases RCC risk.³⁹

A long-term history of hypertension appears to have a dose-response association with RCC risk, independent of obesity. Elevated blood pressure and increases in blood pressure over several years are associated with increased RCC risk, whereas blood pressure declines over time appear to be associated with decreased RCC risk—suggesting that hypertension could promote tumorigenesis.³⁶ As with cigarette smoking, a hypothesized biological mechanism for the association between RCC and hypertension is chronic renal hypoxia.³⁶

Associations with pain medications also have been studied. For example, in 1985, phenacetin-containing analgesics were removed from the market because of an association with increased RCC risk.³⁶,³⁷,⁴⁰ A 2014 meta-analysis of data pooled from 20 epidemiological studies conducted around the world found that acetaminophen and nonaspirin nonsteroidal anti-inflammatory drugs were associated with a significantly increased risk of kidney cancer.³⁹

A 2014 study that pooled data from 7 studies found RCC risk is associated with a history of renal calculi—but only among men.¹⁴ Occupational exposures that might increase RCC risk include refinery and petrochemical plant work and the chemical solvent trichloroethylene.³⁶,⁴²,⁴³ Occupational exposure to benzene, which is tied to other cancer types, might be a risk factor for RCC as well.⁴³

In addition to modifiable lifestyle, dietary, and occupational risk factors, certain medical conditions and procedures appear to increase the risk of developing renal masses. Specifically, patients with end-stage renal disease who undergo dialysis or kidney transplantation face up to 100 times higher risk of RCC than the general public; these patients also face an increased risk of cancer in other organs.¹³,¹⁴ Dialysis and transplantation also are associated with acquisition of renal cysts, which might contribute to the increased risk of kidney cancer.

### Genetic and Epigenetic Factors

Tuberous sclerosis is an autosomal dominant disease caused by mutations in either the TSC1 or TSC2 genes.¹² Most patients (up to 75%) develop nonmalignant angiomylipomas, which are solid masses composed of fat, vascular tissue, and smooth muscle cells.¹⁴ Patients also commonly develop simple renal cysts. There is reason to suspect that patients with tuberous sclerosis also are at increased risk for developing RCC, although the evidence has been mixed.¹⁴

Cancers, such as RCC, arise and progress alongside genetic alterations. Genes provide DNA-encoded instructions for the production of proteins. Alterations to DNA can affect gene expression and increase the risk of RCC. Other malignancies can include genetic mutations or epigenetic alterations such as the methylation of DNA.⁴⁶ Epigenetic changes do not change the DNA code of a gene; rather, they inhibit or silence expression of that gene.⁴⁷

Hereditary RCC syndromes have offered windows into the gene alterations that contribute to tumor formation and progression.⁴⁷,⁴⁸ VHL gene mutations are found both in patients diagnosed with VHL and those diagnosed with sporadic RCC, for example.⁴⁷ VHL is a tumor suppressor gene that, in the absence of mutations, regulates hypoxia inducible factor transcription proteins such as hypoxia inducible factor 1, alpha subunit (HIF1α).⁴⁷ In turn, HIF1α regulates vascular endothelial growth factor (VEGF), platelet-derived growth factor, and glucose transporter-1 protein.⁴⁷ VHL mutations disrupt this regulatory pathway, leading to overstimulation of growth factors and contributing to tumorigenesis.⁴⁷

Other genes involved in renal malignant tumor formation and progression include MET, FLCN, TSC1, TSC2, FH, and SDHB.⁴⁷ (In addition to these genes, mutations in several genes are associated with cancer in the kidneys and other organs: RAS genes, BRAF, TP53, PTEN, ERBB2, pRB, and CDKN2A.)⁴⁷ Mesenchymal-epithelial transition (MET) is a proto-oncogene involved in hereditary and sporadic forms of papillary RCC.⁴⁹ It produces a cell-surface receptor protein that helps to regulate cell division and development.⁴⁸ The function of the folliculin gene (FLCN) is not well understood but loss-of-function FLCN
Renal cysts are diagnosed with the use of a patient’s medical history and clinical examination, then imaging findings. Imaging findings that help with the differential diagnosis of renal cysts include:

- Whether one or both kidneys are involved.
- The size of the cyst.
- The co-occurrence of noncystic renal masses.
- Whether cysts are situated in the renal cortex, medulla, or renal pelvis.
- Whether cysts are densely distributed.
- The presence or absence of cysts in other abdominal organs such as the pancreas or liver.

RCC usually is asymptomatic until it has reached advanced stages and is suspected at an early stage only because of incidental imaging findings or lab tests. For some patients with advanced-stage RCC, presentation includes a classic triad of persistent flank or back pain, a palpable mass, and gross hematuria. Unexplained weight loss, anemia, fatigue, persistent fever not associated with an infection, and loss of appetite also might be symptoms of RCC. These symptoms are not specific to kidney cancer, however. A patient’s complete medical history is taken to check for risk factors and symptoms, and a physical examination is performed to identify palpable abdominal masses.

Medical history should include questions about the patient’s family history of cancer. Siblings of a person with RCC have more than twice the relative risk of RCC; having a history of relatives with RCC before age 50, or 2 or more family members with RCC, indicates a possible hereditary syndrome. If kidney cancer is suspected after the medical history and clinical examination, then the physician should order laboratory tests and imaging examinations. Laboratory tests are not diagnostic but can inform diagnosis, prognosis, and treatment planning. A complete blood count is performed, and results are frequently abnormal in the presence of RCC. If RCC tumor cells are excreting erythropoietin, the patient might have polycythemia or an elevated red blood cell count. Complete blood count results also help determine whether a patient is a candidate for surgery.

In addition to a complete blood count and urinalysis, blood chemistry panels might detect problems with renal function (eg, glomerular filtration rate or creatinine clearance rate) as well as liver enzyme or blood calcium biomarker levels, the latter of which is an indication for skeletal scintigraphy (nuclear imaging of the bone) to detect possible metastatic tumors. Diagnostic tumor biopsy is not performed as frequently with suspected kidney cancer as with other cancer types because modern diagnostic imaging usually is sufficient for determining whether surgery is needed. But when imaging results are indeterminate, follow-up CT or CECT imaging might be recommended. Percutaneous fine-needle or core biopsy with ultrasonography or CT guidance might be performed for microscopic pathology assessment of mass tissue.
Renal mass biopsy also is sometimes performed as part of a surveillance plan. Once cells are extracted, they might be assigned a Fuhrman grade (a scale of 1-4) based on the microscopic appearance of cell nuclei, with grade 1 cells looking similar to healthy renal cells. In addition to the contemporary approaches to renal mass evaluation, new diagnostic and prognostic molecular tools are under development that might change clinical practice. For example, the RCC-specific proteins aquaporin 1 (AQP1) and perilipin 2 (PLIN2) are undergoing clinical development as potential urinary biomarkers to help differentiate clear cell and papillary RCC tumors associated with increased AQP1 and PLIN2 levels from benign oncocytes or angiomyolipomas associated with urinary concentrations similar to those seen in healthy patients.

**Renal Cell Carcinoma Staging**

Aside from a patient’s age, comorbidities, and overall health status, tumor stage is the most important prognostic factor. Tumor stage is a description of the size and location of primary tumors and the extent to which they have invaded or spread to adjacent or distant tissues and organs. Tumors can spread by infiltrating adjacent tissues or when tumor cells enter the lymph system and travel through lymph ducts and vessels to other organs to metastatic tumors.

Several staging systems exist for RCC. The American Joint Committee on Cancer has endorsed the tumor, node, metastasis (TNM) staging system, which is the most widely used. TNM scores assist clinicians in assigning prognostic group status. T stage is a number representing the extent and size (greatest diameter) of the tumor (see Figure 6). For example, T1 is assigned to tumors less than or equal to 7 cm in dimension and limited to the kidney. T2 tumors are greater than 7 cm and limited to the kidney. T3 tumors extend into primary renal veins or adjacent (perinephric) tissues but not the adrenal gland. T4 tumors extend beyond the Gerota fascia. The N stage refers to the involvement of tumor cells in the regional lymph nodes. N0 means no regional lymph node involvement is evident. N1 represents involvement...
of one or more regional lymph nodes. M refers to the presence or absence of metastatic tumors, or those that have spread from the kidneys to distant organs. M0 means no distant organ or bone metastatic tumors exist, whereas M1 represents metastatic disease.6,56

Anatomic stage or prognostic groups represent a simplification of TNM staging, with stage I representing T1N0M0, for example, and stage IV representing T4 tumors with nodal involvement, or any T-stage tumor with distant metastasis (M1).6,56 Contiguous ipsilateral adrenal involvement of a primary tumor is classified as T4, whereas noncontiguous involvement of the ipsilateral adrenal gland is considered metastatic (M1).6,55

**Treatment Cysts**

Asymptomatic simple renal cysts rarely require intervention or follow-up.2,15 Complex cysts require CT imaging to determine whether cysts are progressing into renal malignancies.2 Sometimes cysts merit medical intervention. For example, when cysts rupture or are complicated by hemorrhage or infection, they can become symptomatic in rare instances causing hematuria and pain.2 Evidence suggests that enlarged or multiple simple renal cysts cause hypertension, and treatment can alleviate symptoms.2 However, such symptoms are more strongly associated with malignancies than cysts and are therefore an indication for diagnostic imaging to determine whether a patient has kidney cancer.2

Treatments for renal cysts include antimicrobial therapy (for infected cysts), surgical excision, or ultrasonography-guided or CT-guided cyst puncture and aspiration.2 Because cysts frequently recur after simple aspiration, cyst fluid aspiration commonly is accompanied by injections of sclerosing agents, usually ethanol, to damage cyst walls.2 Other sclerosing agents used for this purpose include glucose, phenol, bismuth phosphate, tetracycline and minocycline, carbon dioxide, acetic acid, and hypertonic saline, among others.2 Acetic acid appears to be more rapid and effective than ethanol at destroying cyst wall lipids and disrupting the structural integrity of the cyst wall collagen.2 However, more research is needed to validate the usefulness of aspiration and various sclerosing agents in cyst management.2 Heritable polycystic diseases usually are managed with infection control and treatment of pulmonary hypertension.2

**Malignant Solid Masses**

Solid malignant masses in the kidney require medical intervention. Localized, early-stage (I/II) renal cancers usually are curable with surgery.24 Once the cancer is identified and staged, a treatment plan is devised based on the patient’s overall health and preferences, treatment toxicity and adverse-effect profiles, and probable treatment outcomes.24 A treatment plan might involve a single treatment modality or a combination of therapies.

RCC can be treated with nephrectomy, either as open surgery or via laparoscopy or robotic-assisted laparoscopy. Nephrectomy is the surgical removal of the entire affected kidney, adrenal gland (unless the tumor is limited to the lower end of the kidney), and fatty layer in a single surgical procedure.24 Regional lymphadenectomy, or lymph node dissection, sometimes is performed during radical nephrectomy to help stage nodal involvement.24 Chemotherapy rarely is effective for RCC and usually is attempted only after other treatment modalities have not succeeded.24 Radiation therapy is not a primary treatment because renal tumors tend to be radioresistant. However, external-beam radiation therapy has neoadjuvant, adjuvant (postsurgical), and palliative roles alongside other treatments or therapies.6,24

The preferred treatment for early-stage RCC is partial or nephron-sparing nephrectomy involving removing tumor-affected portions of the kidney and sparing nonmalignant kidney tissue.24 As with radical nephrectomy, nephron-sparing surgery might be performed with the use of laparoscopy or robotic-assisted laparoscopy.24 Treatment options are more limited for patients with metastatic disease. In rare instances, however, curative nephrectomy might be attempted in patients diagnosed with RCC metastatic disease—but only if the metastasis involves a single or few tumors in distant organs that also can be surgically resected.24

For patients who are not candidates for nephrectomy, local tumor ablation sometimes is attempted with local anesthesia.24 Ablative modalities include cryotherapy
and radiofrequency ablation in which a thin needle or probe is used to deliver very low or very high temperatures to tumor tissue.24

Among patients with advanced RCC (stage III/IV), biologic or immunotherapy treatments might be attempted.24 Immunotherapy treatments are favored for advanced RCC and can slow the growth of renal tumors—or even cause them to shrink temporarily—but are not curative.24 These agents can block specific proteins or signaling pathways to disrupt tumor growth or tumor recruitment of new vasculature and access to the body's blood supply.24 Bevacizumab slows the growth of tumor neoangiogenesis, and medications, such as pazopanib and sorafenib, block both neoangiogenesis and tumor growth.24 The agents temsirolimus and everolimus block the mTOR protein signaling pathway, which can hasten tumor growth through the accumulation of tumor-promoting oncoproteins (eg, HIF1α and VEGF) that are involved in tumor recruitment of new vasculature and improved access to nutrients from the patient's bloodstream.24,47,49

Biologic therapies, or immunotherapies, are cytokine agents that improve the body's immune system attacks on malignant cells. Biologic therapies used for renal cancer include interleukin-2 (IL-2) and interferon-α.24 For a small number of cases, IL-2 can shrink tumors, but the therapy entails a severe and sometimes fatal toxicity profile.24 Interferon-α is less toxic but also less effective than IL-2 and sometimes is used in combination with bevacizumab to improve the response rate.10,24

Most transitional cell carcinomas (TCCs) can be cured at early stages; treatment typically involves radical nephrectomy.24 The ipsilateral ureter and often the ureter-attached portion of the urinary bladder also are excised.24 Neoadjuvant chemotherapy also might be administered before surgery as part of TCC treatment.24 Catheter arterial embolization might be performed before surgery to minimize bleeding.24

Active Surveillance

Active surveillance of renal masses might be recommended before or after treatment of small renal masses.57-59 Post-treatment surveillance with CT imaging typically follows nephron-sparing surgery or tumor ablation to detect new primary tumors or recurrent tumors in remaining renal tissues.59 More frequently, active surveillance refers to deferred medical intervention while the growth rates of small, localized renal masses (< 4 cm) are assessed over time, typically every 4 to 6 months for 2 years and every 6 to 12 months thereafter.24,57,58 Small, localized renal masses frequently are indolent and unlikely to become metastatic or life-threatening if treatment is delayed; only 20% of small renal masses show high malignant potential.58,60 Proponents of active surveillance argue this allows time for routine imaging in lieu of immediate and potentially unnecessary treatment.57, 61 Delayed treatment preserves kidney tissue and function and avoids potential complications associated with treatment, particularly among elderly patients or those with comorbidities or poor overall health status.24,57,61 Biopsy sometimes is performed to confirm whether a small mass is malignant and surveillance is warranted.24,61

Despite the growing popularity of active surveillance, standardized protocols and guidelines are not available, and evidence that it benefits patients and improves quality of life or survival is lacking or in early stages.57,61 Reliable clinical predictors of an individual small tumor’s malignant potential are not well established.48 Nephrometric systems such as the RENAL score have been developed, and early studies suggest these scores correlate with renal masses’ annual growth rates.62,63

The RENAL score supports quantification of renal masses to guide treatment decisions. RENAL refers to:

- Radius – the maximal cross-section of a renal mass expressed in centimeters.
- Exophytic or endophytic – exophytic refers to tumor outward growth beyond its epithelial origin; endophytic refers to inward growth.
- Nearness – of the mass to the renal collecting system or sinus.
- Anterior or posterior position in the kidney – a subcategory of “A” is “h,” for hilar tumor involvement in the main renal artery or vein.
- Location of the tumor – relative to the superior/anterior midline of the kidney.

Visit www.nephrometry.com for more on the RENAL scoring system.
Renal Imaging Overview

The history of renal imaging is as old as medical radiography itself, with the first examinations performed in the late 1800s. By the mid-1900s, uroradiologists administered iodinated contrast agents to image the kidneys with the use of intravenous (IV) urography and IV pyelography, along with mass puncture or biopsy, to diagnose renal masses. However, mass puncture frequently failed with smaller masses. Diagnostic exploratory surgery was common when patients were symptomatic and mass puncture had been unsuccessful. Arteriography was used widely in the mid-1960s, and by the 1970s ultrasonography was used to assess renal masses. In the 1970s, ultrasonography helped physicians to differentiate cystic from solid renal masses—an application for which it remains a choice imaging modality.

CT technology revolutionized medical imaging throughout the 1980s and 1990s. By the early 2000s, technical advances in CT imaging, such as the development of multidetector scanners, largely replaced IV urography, and CT imaging became recognized as a standard preliminary diagnostic modality for many renal imaging applications.

Today, ultrasonography, CECT, magnetic resonance (MR) imaging, and nuclear medicine imaging, including positron emission tomography (PET), are appropriate to some degree for different facets of renal mass diagnosis, staging, monitoring, and surveillance. MDCT is more widely available, faster, and less expensive than MR, and its narrow collimation improves acquisition of 3-D data sets for volumetric visualization of complex anatomies such as renal vasculature. Like ultrasonography, MDCT can differentiate cystic from solid renal masses reliably; MDCT also can help identify specific types and subtypes of renal masses and kidney cancer.

The American College of Radiology’s (ACR) appropriateness criteria for assessing indeterminate renal masses ranks contrast and noncontrast abdominal CT highly, with a 9 out of 10 score for the diagnostic imaging of renal masses. This ranking means that CT and CECT imaging are recommended as the first choices for follow-up imaging assessment of indeterminate renal masses. The only concern listed, preventing a 10 out of 10 ACR appropriateness score, is patient exposure to ionizing radiation from the CT examination.

MR sometimes is used as a follow-up examination when CT imaging of a renal mass proves to be indeterminate. PET-CT sometimes is used to simultaneously display mass anatomy and metabolism and to stage RCC. PET skeletal scintigraphy might be performed when metastatic renal tumors are suspected to have spread to the bones.

CT Assessment of Renal Masses

For renal masses that require follow-up imaging or medical intervention, it is important to identify which lesions are candidates for active surveillance and which might be malignant. CT scanning is the imaging modality of choice for helping physicians distinguish among lesions larger than 1 cm in maximum diameter. Some authors caution that CT has limited utility in characterizing renal masses smaller than 1 cm.

Patient Preparation

Having a history of allergy to iodinated contrast agents puts a patient at higher risk of experiencing a reaction to the contrast material used in CECT, and alternative imaging modalities should be considered for these patients. Allergy-like reactions to contrast media entail 5 times the risk of a subsequent contrast-allergy reaction. Although it is no longer recommended that patients be asked about their history of dairy, seafood, or shellfish allergies, as there is not reliable evidence these histories predict adverse reactions to iodinated contrast agents, history of a previous contrast reaction is important. Patients who have significant allergies, such as history of severe reactions or several allergies, should be questioned further. Contrast-induced nephrotoxicities from iodinated contrast agents are detailed in the ACR Manual on Contrast Media.

To access the manual online, visit www.asrt.org/asrt/hWloXn.

Oral or IV hydration can ensure contrast media excretion and minimize the risk of nephrotoxicity from IV contrast agents. This is particularly important for patients with chronic kidney disease or renal
insufficiency who face an increased, albeit small, risk of contrast-induced nephropathy. MR or CT without contrast or other alternatives to CECT imaging might be considered for these patients. However, if CECT is determined to be the best method to obtain necessary clinical information about a renal mass, then oral or IV hydration for fluid volume expansion should be completed before the CECT examination and the minimum possible volume of contrast material used.  

**Contrast-Enhanced Computed Tomography**

CECT helps radiologists differentiate solid tumors from cystic masses, which tend to be radiographically hyperdense, and CECT yields visual information useful for renal mass characterization, staging, and treatment planning. Renal CECT can be performed on older single-detector and contemporary MDCT scanners. MDCT scan acquisition times are much faster than single-detector models and offer superior z-axis spatial resolution and volumetric image precision. CECT involves image acquisition during several phases of contrast-bolus enhancement and excretion. The phases of renal enhancement are:

- **Arterial phase** – 15 to 25 seconds after IV contrast administration.
- **Corticomedullary or late arterial phase** – 25 seconds after IV contrast bolus injection. Nephronic glomerulus filtration of contrast agent causes cortical renal enhancement, which shows renal mass vascularity and detailed volumetric mapping of renal venous vasculature, an important part of planning partial nephrectomy.
- **Venous phase** – 55 seconds after IV contrast injection.
- **Nephrographic or parenchymal venous phase** – 80 to 120 seconds after IV contrast injection. Contrast material has reached the collecting ducts by this point, enhancing renal parenchymal tissues.
- **Excretory or late phase** – 180 seconds after IV contrast injection. Contrast bolus excretion from the renal parenchyma enhances the renal pelvis and ureters. Excretory phase scan data shows tumor associations with the collection tubules, renal pelvis, and ureters for planning of nephron-sparing partial nephrectomy.  

The exact timing of the phases depends on the IV contrast injection rate, but a protocol described by professors at the Johns Hopkins University School of Medicine assumes IV injection of 100 mL to 120 mL of nonionic contrast material (iohexol) at a rate of 3 mL/s, 0.75-mm collimation, and 0.75-mm slice thickness scan acquisitions using a 16-row-detector CT scanner. CECT protocols for renal mass assessment vary among institutions. For example, a separately reported CECT protocol for MDCT scanners involves 150 mL of IV contrast injected at a rate of no less than 3 mL/s, with scan acquisitions at 40 seconds and 100 seconds (for corticomedullary and nephrographic phase scans), reconstructed to 4-mm slices. An unenhanced (precontrast) CT scan of the kidneys is acquired before CECT scanning begins to provide baseline or comparison images to the CECT scans.

As the contrast agent moves through the renal tissues, scan acquisitions can be obtained repeatedly to capture information about different phases of renal enhancement. Different phasic CECT imaging protocols are in use, using the distinct phases of renal enhancement to address different pathologies. At least 3 acquisition sequences are acquired to detect and characterize renal lesions: the precontrast image, the late arterial phase acquisition, and the delayed (excretory) phase acquisition. Some institutions limit CECT to these 3 phases of image acquisition to reduce patients’ CECT-related doses of ionizing radiation. Other renal imaging protocols also involve acquisition of scan data during the corticomedullary, nephrographic, and excretory phases of enhancement.

A written standard operating procedure for renal mass assessment should be available and consulted carefully at each institution. It is important that slice collimation, milliampere second, peak kilovoltage, and pitch be the same for scan acquisitions of precontrast and contrast-enhanced image acquisitions. The 16-row MDCT CECT protocol involves precontrast acquisition of kidney scans, followed by a late-arterial phase scan acquisition 25 seconds after contrast injection. Next, a delayed or excretory phase scan acquisition of the abdomen and pelvis is timed at between 180 and 240 seconds from contrast injection. MDCT workstation postprocessing options allow rapid, nearly real-time maximum intensity projection.
display of scan data, as well as multiplanar reconstruction and volume rendering.\textsuperscript{67} Corticomedullary-phase CECT images are used for volumetric display of renal vasculature.\textsuperscript{73} Because renal masses do not harbor intact nephrons, the degree of contrast enhancement reflects the degree of vascularization.\textsuperscript{67} In most cases, this readily allows differentiation of fluid-filled, avascular cysts from vascularized renal malignancies.\textsuperscript{67} Therefore, cysts do not exhibit contrast enhancement and instead exhibit CT attenuation values smaller than 20 HU.\textsuperscript{67} A CT attenuation value increase of 10 HU or more is the traditional definition of enhancement indicating that a renal mass is solid rather than cystic.\textsuperscript{74} However, authors have noted that this definition was proposed in 1986, before helical and MDCT scanners were widely available.\textsuperscript{73} At some centers the 10 HU threshold is still used; elsewhere, renal mass enhancement is now defined as an increase of 15 HU to 20 HU.\textsuperscript{73,75}

Complicating matters further, small cysts sometimes exhibit pseudoenhancement, possibly because of image reconstruction artifacts related to beam-hardening effects on data from renal parenchyma.\textsuperscript{73,76} Pseudoenhancement is more pronounced with 64-detector MDCT scanners than with 16-detector scanners and occurs more often with renal cysts smaller than 2 cm.\textsuperscript{76}

Renal Cell Carcinoma

CECT is the preferred diagnostic imaging modality for RCC; it is associated with an RCC diagnostic accuracy rate higher than 95%.\textsuperscript{77} Contrast and noncontrast MDCT imaging also are used in RCC staging, active surveillance, and post-treatment monitoring.\textsuperscript{55,77}

Oral contrast frequently is used alongside IV contrast injection.\textsuperscript{77} Well-vascularized RCC tumors exhibit maximum enhancement during the corticomedullary phase.\textsuperscript{67} Corticomedullary and nephrographic phase scan acquisitions ensure both hypervascular cortical and hypovascular medullary tumors are well enhanced.\textsuperscript{77} Corticomedullary phase images help physicians distinguish RCC tumors from aneurysms.\textsuperscript{55}

Contrast-enhanced images are compared with precontrast unenhanced images, and multiphasic enhancement patterns are examined to help characterize RCC tumors and to rule out benign angiomyolipomas, oncocytomas, and hyperdense cysts.\textsuperscript{55,77} Nephrographic phase images display RCC tumors as less dense than adjacent parenchyma. Smaller RCC tumors tend to be homogeneous with smooth and distinct margins.\textsuperscript{77} Larger tumors frequently have necrotic central foci, a more lobular-appearing margin, and in many cases, calcifications.\textsuperscript{77} Tumors smaller than 3 cm in diameter are often best seen during the venous phase or excretory phase.\textsuperscript{87}

Ideally, RCC diagnostic and staging images should show:

- The number, sizes, and locations of tumors.
- Tumor invasion of the fat layer, adrenal glands, or renal sinus.
- Vasculature and vascular invasion.
- Involvement of adjacent organs.
- Metastatic tumors in distant organs.\textsuperscript{55}

The involvement of the renal vein or inferior vena cava is important information for surgical planning.\textsuperscript{55} In addition, presurgery nodal staging to avoid unnecessary nodal dissection requires detailed visualization of regional lymph nodes.\textsuperscript{55}

RCC can be mistaken for TCC, especially when the RCC occurs in the renal pelvis.\textsuperscript{79} Attenuation differences during the corticomedullary and nephrographic phases of enhancement might help differentiate clear cell RCC from TCC.\textsuperscript{79} Multiphasic enhancement patterns and phase-specific CT features also can provide clues about a tumor’s RCC subtype histology but cannot alone offer definitive subtype diagnosis.\textsuperscript{55,80,81} For example, corticomedullary phase images might help in differentiating poorly vascularized papillary RCC from clear cell RCC.\textsuperscript{55} Papillary RCC tumors tend to exhibit low peak enhancement overall, but peak enhancement during the nephrographic and excretory phases, whereas clear cell RCC tumors exhibit high attenuation and peak enhancement during the corticomedullary phase.\textsuperscript{81} Oncocytomas exhibit peak enhancement in nephrographic-phase images.\textsuperscript{81} It was proposed in 2014 that RCC subtypes can be differentiated using only corticomedullary phase CECT images to a degree comparable to that possible using multiphasic imaging, but that retrospective study involved 79 patients and additional research is needed.\textsuperscript{82}
**Bosniak Classification System**

The 1986 Bosniak classification system for renal cysts uses CT image features, such as morphology, calcifications, cyst wall appearance, and contrast enhancement, to distinguish simple, benign cysts from more complex masses that might require monitoring or medical intervention (see Table). Precontrast and nephrographic phase CECT images are compared to assess enhancement of the distinguishing features of renal mass anatomy for scoring purposes.

The Bosniak system classifies cysts into 5 categories that ascend toward potential for malignancy. Bosniak category I and II cysts are benign. Bosniak category IIIF cystic masses are malignant in 11% to 25% of cases, according to 2 recent studies; Bosniak III category cystic renal lesions are malignant in 54% of cases. Bosniak category I cysts are fluid-filled benign cysts that have Hounsfield unit values similar to water (0-20 HU), with hairline-thin walls that lack septations and calcifications. Minimally complicated Bosniak cysts have calcification of the cyst wall or septa and perceived but not measurable hairline-thin septal enhancement. Homogeneous hyperattenuating (> 20 HU) nonenhancing lesions 3 cm or less are considered Bosniak II cysts. The hyperattenuating cyst wall that extends beyond the parenchyma should be smooth appearing (see Figure 7).

Bosniak IIIF cysts require follow-up diagnostic imaging with CT or MR because of the high probability these masses are malignant. This category includes hyperattenuating nonenhancing cysts with diameters larger than 3 cm that do not extend beyond the renal parenchyma.

Bosniak III cyst walls might be thick and not smooth, and these cysts can contain septations with measurable enhancement of between 45 HU and 75 HU (see Figure 8). These cysts might represent malignancies, hemorrhage, or renal abscess. Because of the risk of cancer, Bosniak III cysts are treated as an indication for surgery.

**Table**

<table>
<thead>
<tr>
<th>Bosniak Classification of Renal Cysts</th>
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<tr>
<td><strong>Lesion Type</strong></td>
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*Abbreviations: CT, computed tomography; HU, Hounsfield unit; MR, magnetic resonance.

³ Hyperattenuating Bosniak II and IIIF cysts have no septations or calcifications.
Bosniak IV cysts almost always are malignant and contain enhancing soft-tissue components.\textsuperscript{14,73} Surgery is indicated for these lesions.\textsuperscript{14,73}

**Radiogenomics**

Radiogenomics refers to the use of CT and other medical imaging to assess gene mutations associated with masses.\textsuperscript{85,90} The term radiogenomics also is used to describe efforts to identify gene variants associated with increased or decreased risk of radiation therapy–associated toxicities, but this is not the definition used in this article.

Multiphasic CECT scan features can help physicians differentiate RCC subtypes. The emerging field of radiogenomics promises to take RCC evaluation a step further, using mass imaging feature associations to identify genetic mutations associated with a particular tumor.\textsuperscript{85-90}

In an era of cost controls and targeted anticancer agents and gene therapies, radiogenomics could support efforts in cancer genomics as a valuable clinical tool for informing diagnostic and treatment decision making and determining patient prognosis.\textsuperscript{90}

Radiogenomics is possible because gene and protein expression patterns can affect the imaging features of a tumor.\textsuperscript{90} Whereas anatomic imaging traditionally reveals imaging associations with histopathology, radiogenomics researchers seek clinically meaningful “association maps” between image features and specific gene or protein biomarkers, sometimes called molecular phenotypes.\textsuperscript{86} For example, preliminary findings suggest pretreatment CT image features, such as tumor margin definition, nodular tumor enhancement, intratumor vascularity, and renal vascular invasion, are associated with specific mutations in clear cell RCC tumors.\textsuperscript{85} Enhancement patterns in multiphasic MDCT also are associated with loss of chromosome 8p in clear cell RCC tumors.\textsuperscript{85}

Tumor size, calcification, hyperattenuation on unenhanced MDCT, and staging information such as lymph node involvement and distant metastasis could be helpful in distinguishing papillary RCC from RCC tumors in children and adolescents that harbor chromosome Xp11.2 translocation and transcription factor binding to IGHM enhancer 3 (TFE3) gene fusion. These tumors are associated with an especially poor prognosis.\textsuperscript{85,89} Conflating these 2 types of RCC leads to suboptimal assessment of patient prognosis and disease management.\textsuperscript{85} Radiogenomic screening of RCC tumors for imaging features associated with Xp11.2 translocation and TFE3 gene fusion could help avoid unnecessary immunohistochemical testing for patients unlikely to have these gene mutations.\textsuperscript{87}
Although early findings have been promising, the field of radiogenomics is young. Proponents expect the field to improve the prognostic value of diagnostic imaging and result in more meaningful surveillance and treatment-response imaging.

**Metastatic Disease**

Estimates vary, but up to 33% of patients have metastatic disease at the time of RCC diagnosis. Until recently, metastatic disease often prompted clinicians to abandon efforts to prolong a patient’s life in favor of palliative care. However, treatment with newer, targeted agents, such as bevacizumab, sunitinib, and interferon, can prolong the lives of these patients. Metastatic renal tumors frequently are found in the lungs, bones, liver, and brain.

Several imaging modalities can help physicians characterize the location and number of metastatic RCC tumors, but MDCT-based CECT that includes arterial-phase scan acquisition is emerging as a versatile modality for this task. Pulmonary RCC metastases occur in 45% of patients diagnosed with metastatic RCC; the pulmonary lesions are seen on MDCT as solitary or multiple nodules, sometimes as large as 5 cm in diameter (see Figure 9).

Bone metastasis occurs in 30% of patients diagnosed with RCC metastatic disease, but unlike pulmonary metastases, bone tumors usually are symptomatic, associated with painful fractures. Bone metastases appear on MDCT images as bone-destroying foci in limbs, vertebrae, ribs, or the pelvis. RCC metastasis to the bones usually involves more than one metastatic tumor. Liver metastasis occurs in 20% of RCC patients overall; these tumors are displayed on MDCT images as hypervascular foci (see Figure 10). RCC metastases in the brain affect fewer than 10% of patients. Most patients diagnosed with brain metastases also have metastatic tumors in other organs. Brain metastases usually appear as solitary, enhancing nodules.

**Conclusion**

CECT plays a central role in the diagnostic imaging, characterization, classification, and staging of renal masses in adults. Dual-energy CT techniques under development might overcome the problem of

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**Figure 9.** Pulmonary metastases from RCC. A. Coronal CT image of the lungs shows a 5-cm paramediastinal metastatic tumor (arrow) located in the left inferior lobe. A lesion of this size is known as a “cannonball” metastasis. Smaller metastases (arrowheads) also are seen in the apical segment of the left inferior lobe. B. Axial CT image shows multiple small metastatic tumors in both lungs (arrowheads). One of these lesions, in the middle lobe in the subpleural location (arrow), shows cavitation after antiangiogenic therapy. Used with permission from Brufau BP, Cerqueda CS, Villalba LB, Izquierdo RS, González BM, Molina CN. Metastatic renal cell carcinoma: radiologic findings and assessment of response to targeted antiangiogenic therapy by using multidetector CT. Radiographics. 2013;33(6):1695.

**Figure 10.** Liver metastases of RCC, with additional pancreas lesion. A. Axial arterial-phase CT image shows 3 hypervascular lesions (arrows) in the liver that were not visible on a previous CT image (not shown) and that are compatible with stage M1 disease. B. Axial nephrographic-phase CT image fails to show these lesions. Because hypervascular lesions cannot be seen during the nephrographic phase but can be easily seen during the arterial phase, it is essential to include the arterial phase in the diagnostic protocol and follow-up for patients with RCC. Another hypervascular lesion in the head of the pancreas (arrowhead in A) is more easily identified during the arterial phase, compared with the nephrographic phase of enhancement. Nearly all patients with RCC liver metastases also have metastases in other locations. Used with permission from Brufau BP, Cerqueda CS, Villalba LB, Izquierdo RS, González BM, Molina CN. Metastatic renal cell carcinoma: radiologic findings and assessment of response to targeted antiangiogenic therapy by using multidetector CT. Radiographics. 2013;33(6):1697.
renal cyst pseudoenhancement in MDCT-based CECT imaging and reduce imaging-related radiation doses to patients. The field of renal mass imaging is likely to advance rapidly during the genomics and personalized medicine era. Biomarkers undergoing testing and development might help with differential diagnosis of renal masses identified in imaging examinations.

Subtype differentiation of renal masses with the use of diagnostic CT techniques also will likely improve. Radiogenomics-based associations between diagnostic imaging of renal masses and their genetic mutations could offer clinicians timely information about treatment options and prognosis with which to guide individualized treatment plans.

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References


324CT

Directed Reading

Computed Tomography of Renal Masses in Adults


91. Bru financially supported by the National Science Foundation, awards 1304252 and 1304838.
1. The kidneys help maintain the body's homeostasis of:
   a. pH.
   b. electrolytes.
   c. fluid.
   d. 1 and 2
   e. 1 and 3
   f. 2 and 3
   g. 1, 2, and 3

2. Healthy adult kidneys filter up to _______ L of blood daily.
   a. 102
   b. 122
   c. 142
   d. 162

3. Which of the following statements is true about the nephrons?
   a. Each nephron contains a corpuscle but no tubule.
   b. Nephrons are filtering units.
   c. Nephrons produce important hormones.
   d. There are approximately 1000 nephrons in the kidneys.

4. Cysts are the most common form of renal mass found during abdominal imaging.
   a. true
   b. false

5. _______ is an example of a developmental cyst.
   a. Polycystic kidney disease
   b. Multicystic dysplasia
   c. Tuberous sclerosis
   d. Von Hippel-Lindau syndrome
12. Modifiable risk factors for kidney cancer include:
   1. cigarette smoking.
   2. obesity.
   3. hypertension.
   a. 1 and 2
   b. 1 and 3
   c. 2 and 3
   d. 1, 2, and 3

13. A diet rich in ______ might reduce the risk of RCC.
   a. red meats
   b. fruits and vegetables
   c. whole grains
   d. organic foods

14. Patients with end-stage renal disease who undergo dialysis are ______ times more likely to develop RCC than the general public.
   a. 2
   b. 10
   c. 50
   d. 100

15. Most patients with tuberous sclerosis develop which of the following?
   a. TCC
   b. nonmalignant angiomyolipomas
   c. clear cell carcinoma
   d. oncocytomas

16. According to the article, epigenetic hypermethylation of unmutated ______ genes appears to be associated with sporadic RCC risk.
   a. TSC
   b. VHL
   c. H1F
   d. BRAF
17. Early stages of RCC are typically asymptomatic and might only be discovered through incidental imaging findings and lab tests.
   a. true
   b. false

18. The Fuhrman grade scores cell nuclei on a scale of 1 to:
   a. 3.
   b. 4.
   c. 5.
   d. 6.

19. Stage T3 RCC tumors extend into _______ or adjacent (perinephric) tissues but not the adrenal glands.
   a. primary renal veins
   b. arterioles
   c. ureters
   d. Gerota fascia

20. According to the article, which sclerosing agent is usually employed during cyst fluid aspiration?
   a. tetracycline
   b. carbon dioxide
   c. ethanol
   d. acetic acid

21. The preferred treatment for early-stage RCC is:
   a. nephrectomy.
   b. chemotherapy.
   c. immunotherapy.
   d. brachytherapy.

22. Temsirolimus and everolimus block which signaling pathway?
   a. RAS
   b. mTOR
   c. DAPK1
   d. MET

23. The ______ phase of enhancement begins 80 to 120 seconds after intravenous contrast injection.
   a. arterial
   b. corticomedullary
   c. venous
   d. nephrographic

24. Which of the following phases are acquired for detecting and characterizing renal lesions in contrast-enhanced computed tomography renal imaging?
   1. late arterial
   2. precontrast
   3. excretory
   a. 1 and 2
   b. 1 and 3
   c. 2 and 3
   d. 1, 2, and 3

25. Which of the following is true regarding renal mass contrast enhancement?
   a. Traditionally, enhancement of 5 HU or more defined a cyst as solid rather than cystic.
   b. Renal masses harbor intact nephrons, giving no clues on vascularization.
   c. With helical and multidetector scanning, centers now use thresholds of 10 HU or 15-20 HU.
   d. The American College of Radiology defined the appropriate Household unit threshold as 25 HU.

26. Pseudoenhancement occurs most often with renal cysts smaller than _______ cm.
   a. 1
   b. 2
   c. 3
   d. 4
27. ______ RCC tumors tend to exhibit low peak enhancement overall, but peak enhancement occurs during the nephrographic and excretory phases.
   a. Clear cell  
   b. Papillary  
   c. Chromophobe  
   d. Spindle

28. ______ RCC tumors exhibit high attenuation and peak enhancement during the corticomedullary phase.
   a. Clear cell  
   b. Papillary  
   c. Chromophobe  
   d. Spindle

29. Bosniak IIF cysts are hyperattenuating nonenhancing cysts with diameters larger than ______ cm.
   a. 3  
   b. 4  
   c. 5  
   d. 6

30. Bosniak ______ cysts are almost always malignant.
   a. I  
   b. II  
   c. III  
   d. IV