

## American College of Radiology ACR Appropriateness Criteria®

**Clinical Condition:** Renal Failure

**Variant 1:** Acute kidney injury (AKI), unspecified.

Radiologic Procedure	Rating	Comments	RRL*
US kidneys and bladder	9	Assess renal size and echogenicity. Exclude bilateral obstruction in high-risk groups. Doppler may be used to assess renal perfusion.	O
Percutaneous US-guided renal biopsy	6	Especially useful in acute inflammatory conditions such as nephritis. Perform a follow-up after US examination, if needed.	O
Tc-99m MAG3 scan kidney	4	This procedure may be useful if the creatinine level is high. Perform a follow-up after US examination, if needed.	☢☢☢
MRI abdomen without IV contrast	3	This procedure has a potential role in searching for sonographically unclear causes of ureteral obstruction. A nonenhanced MRI may be helpful in selected cases.	O
MRA abdomen without and with IV contrast	3	Because the eGFR and creatinine values are unreliable in the setting of AKI, caution should be used when administering intravenous gadolinium. Gadolinium-enhanced studies are very effective for renal artery evaluation.	O
MRA abdomen without IV contrast	3	This procedure can assess renal arterial or venous patency in rare instances when vascular stenosis or thrombosis may account for AKI.	O
Arteriography kidney	3	Potentially helpful in trauma evaluation for renal artery occlusion. Consider using aortography with CO <sub>2</sub> to avoid nephrotoxicity of the iodinated contrast.	☢☢☢
CT abdomen without IV contrast	3	Potentially helpful in trauma evaluation. Noncontrast helical CT is more sensitive than KUB for calculi. Evaluation of ureteral obstruction due to retroperitoneal diseases, masses, and tumors (hydronephrosis on US but an undetectable cause).	☢☢☢
CTA abdomen with IV contrast	3		☢☢☢
CT abdomen with IV contrast	2		☢☢☢
CT abdomen without and with IV contrast	2		☢☢☢☢
X-ray abdomen and pelvis (KUB)	2	To assess for calculi; however, it is insensitive for 30% of calculi.	☢☢
X-ray voiding cystourethrography	2	A VCUG may be indicated if a vesicoureteral reflux is suspected as a contributing factor in AKI.	☢☢
MRI abdomen without and with IV contrast	2	Potential role in search of sonographically unclear causes of ureteral obstruction.	O
<b>Rating Scale:</b> 1,2,3 Usually not appropriate; 4,5,6 May be appropriate; 7,8,9 Usually appropriate			*Relative Radiation Level

**Clinical Condition: Renal Failure**

**Variant 2: Chronic kidney disease (CKD).**

Radiologic Procedure	Rating	Comments	RRL*
US kidneys and bladder	9	Some authors have shown duplex Doppler to be successful in assessing RAS.	O
MRI abdomen without IV contrast	5	The potential role of this procedure is to search for sonographically unclear causes of ureteral obstruction. An unenhanced MRI may be helpful in selected cases. Perform a follow-up after US examination, if needed.	O
Percutaneous US-guided renal biopsy	5	This procedure is especially useful for distinguishing the different causes of proteinuria. Perform a follow-up after US examination, if needed.	O
CT abdomen without IV contrast	5	This procedure is potentially helpful in trauma for detecting calculi or to search for retroperitoneal mass/adenopathy as the cause of the obstruction. Perform a follow-up after US examination, if needed.	☢☢☢
MRA abdomen without and with IV contrast	4	Newer techniques with gadolinium are very effective for renal artery evaluation. Perform a follow-up after US examination, if needed.	O
MRA abdomen without IV contrast	4	If renal function is compromised, a nonenhanced MRA can be performed with good results. Perform a follow-up after US examination, if needed.	O
CTA abdomen with IV contrast	4	This procedure is effective in detecting RAS if nephrotoxicity is not a concern. Perform a follow-up after US examination, if needed.	☢☢☢
X-ray voiding cystourethrography	3	Use this procedure if reflux is suspected. It is particularly appropriate for use in children.	☢☢
CT abdomen with IV contrast	3		☢☢☢
CT abdomen without and with IV contrast	3		☢☢☢☢
MRI abdomen without and with IV contrast	3	The potential role of this procedure is to search for sonographically unclear causes of ureteral obstruction.	O
Tc-99m MAG3 scan kidney	2	Use this procedure to assess global and differential renal function and as a prognosis for recovery.	☢☢☢
X-ray abdomen and pelvis (KUB)	2	To assess for calculi; however, it is insensitive for 30% of calculi.	☢☢
Arteriography kidney	2	There is a problem of contrast nephrotoxicity with this procedure. CO <sup>2</sup> aortography is an option. Newer MRA techniques are preferred.	☢☢☢
<b>Rating Scale:</b> 1,2,3 Usually not appropriate; 4,5,6 May be appropriate; 7,8,9 Usually appropriate			<b>*Relative Radiation Level</b>

## RENAL FAILURE

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### **Summary of Literature Review**

#### **Introduction/Background**

Renal failure is defined as the inability of the kidney to maintain homeostasis leading to azotemia or the accumulation of nitrogenous wastes; however, exact biochemical or clinical criteria for this diagnosis are not clearly defined. Classically, renal failure is distinguished from renal insufficiency when renal function is abnormal but capable of sustaining essential bodily functions [1]. Renal failure is defined as anuric when the urine volume is <50 mL for 24 hours, oliguric when the volume is <500 mL for 24 hours; and nonoliguric when the volume is from 500 to 6,000 mL for 24 hours. Urine output above 6,000 mL is designated as polyuric [2]. Acute kidney injury (AKI) is the internationally preferred term to replace the term acute renal failure (ARF) [3]. The distinction between AKI and chronic renal failure (CRF), now termed chronic kidney disease (CKD), can often be made clinically [4]. End-stage renal disease (ESRD) implies CKD to a degree that life cannot be sustained long-term without dialysis.

Many patients are first seen with markedly elevated serum creatinine of unknown duration, so that classification as AKI or CKD is not possible. The causes of kidney injury are separated into three categories: prerenal, intrarenal, and postrenal. Hypoperfusion is the cause of prerenal failure (including fluid loss, fluid sequestration, low cardiac output, and renal artery stenosis [RAS]). The causes of intrarenal failure include acute tubular necrosis (ATN) and interstitial, glomerular, or small-vessel disease. Obstruction (as well as distal renal tubular obstruction) is the usual postrenal cause of failure.

Renal function is often quantified by assessing serum creatinine. However, significant limitations of serum creatinine as an accurate measure of renal function include decreased muscle mass and poor nutritional status [5]. Glomerular filtration rate (GFR) is usually accepted as the best overall measure of renal function [6]. Creatinine clearance measures the ability of the glomerulus to filter creatinine from the plasma and approximates the GFR. In AKI, the creatinine clearance is usually <25 mL/min. The National Kidney Foundation and American Society of Nephrology recommend estimating GFR from serum creatinine. Two commonly used equations are the Modification of Diet in Renal Disease Study equation and the Cockcroft-Gault equation [7,8]. Both use serum creatinine in combination with age, sex, weight, and race data to estimate GFR. These equations are limited by the use of serum creatinine as a filtration marker, decreased accuracy at higher levels of estimated GFR, and non-steady-state conditions for the filtration marker when GFR is changing.

#### **Acute Kidney Injury**

AKI can be broadly defined as a sudden decrease in renal function resulting in azotemia. The definition of AKI is still under some debate. A recent consensus considers an abrupt (within 48 h) reduction in kidney function, currently defined as an absolute increase in serum creatinine >0.3 mg/dL (>25 μM/L), and a 50% increase or reduction in urine output (documented oliguria <0.5 mL/kg/h for >6 h) to constitute AKI (see [Appendix 1](#) for

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criteria and stages of AKI) [9]. It can develop in the setting of pre-existing renal insufficiency or can develop in a patient with previously normal kidneys [10].

Over 75% of patients with AKI will have either prerenal azotemia (PRA) or ATN (a parenchymal, intrarenal process) as the cause [4]. Prerenal causes of AKI relate to hypoperfusion or hypovolemia. A clinical suspicion of AKI usually leads to a fluid challenge with central monitoring and correction of the hypovolemic state, which corrects the renal failure. A common exception to this approach is the patient with heart failure or liver failure. A high ratio of blood urea nitrogen to creatinine has long been considered a marker of PRA. In addition, a characteristic laboratory finding in PRA is avid sodium retention, with a urine sodium concentration  $<20$  mEq/L [11]. A meta-analysis of various laboratory studies in an attempt to differentiate PRA from ATN revealed that most determinations (urine/plasma creatinine index, urine/plasma urea, or urinary sodium) are often nonspecific or unreliable [10]. Still, most experienced clinicians find that when urine output is  $<500$  mL for 24 hours, a determination of urinary fractional excretion of sodium is helpful.

In a patient with previously undiagnosed renal failure, the most helpful initial diagnostic test is to evaluate renal size using grayscale ultrasonography (US). If the kidneys are small and echogenic, the process is long-standing and unlikely to improve. Renal size is commonly determined from a measurement of renal length. Renal volume calculated by US provides a better measure of functioning renal parenchyma [12]; however, it is an imperfect measure because it includes the renal central sinus fat. In patients with CRF, the renal cortical thickness measured by US appears to be more closely related to the estimated glomerular filtration rate (eGFR) than renal length [13].

Sometimes, evaluation by US helps to identify a correctable cause of renal failure, such as obstruction, which is a postrenal cause of AKI. However, a number of studies have evaluated the use of US in the setting of acute renal insufficiency and have found it is generally unnecessary to exclude an obstruction unless 1) there is a clinical history that strongly supports an obstruction [14], such as flank pain, urolithiasis, or pelvic mass [15]; 2) the patient is deemed at high risk for obstruction [16]; or 3) if specific clinical factors stratify the patient into a high-risk group [17]. This applies irrespective of whether the patient requires treatment in an intensive care setting [18]. If hydronephrosis is present, retrograde or antegrade relief of the obstruction is usually performed. If there is no hydronephrosis and the patient does not have hypertension or other history to suggest RAS, then further workup of the small, echogenic kidneys is not warranted.

Obstruction may occur in the oncologic patient, the trauma patient, or the patient with a solitary kidney [19,20]. Bilateral, obstructing ureteral calculi are rare. More often in the older male patient, a bilateral ureteral obstruction may occur in a chronic bladder outlet obstruction by an enlarged prostate. In such cases, the patient may appear to be suffering from new onset renal failure; however, the deterioration in renal function has been ongoing but has remained clinically occult. Grayscale US is the most effective way to exclude a subacute or chronic obstruction. Regular grayscale US is not accurate in the minimally dilated obstructive situation, such as with retroperitoneal metastatic tumor or idiopathic retroperitoneal fibrosis, in which the ureter encasement interferes with peristalsis; in 1 series, 4%–5% of patients with an obstruction showed minimal or no upper-tract dilation [21]. Color Doppler US of the bladder can assist in detecting a urinary obstruction. Ureteral jets are streams of urine entering the bladder that can be detected with color Doppler US. The unilateral absence of a renal jet after 10 minutes of observation has an approximately 90% positive predictive value for a complete obstruction [22]. The bilateral absence of ureteral jets, however, is a normal finding. Also, in a partial obstruction, the ureteral jets can still be seen; a continuous low-level jet on the partially obstructed side is common [22].

The patient with an obstructed renal transplant can present with acute failure. Platt et al [23] found an elevated resistive index (RI) in 85% of transplanted kidneys with obstruction; a normal RI should argue strongly against an obstruction, unless a ureteral leak is also present. In addition to obstruction, an elevated RI can also be found in rejection and ATN; therefore, RI measurements are not reliable in the differential diagnosis of these entities. If US cannot determine the cause of the obstruction, MRI or nonenhanced CT can be obtained (retroperitoneal mass, lymphadenopathy, fibrosis, calculi, etc.).

RAS and thrombosis are prerenal causes of AKI, but they are uncommon. The multicenter Program to Improve Care in Acute Renal Disease Study found that renal artery thrombosis, stenosis, or trauma accounted for 1% of the causes of AKI in 618 critically ill patients [24]. US can provide a measure of renal perfusion. Some have suggested that duplex Doppler US can supplant radionuclide scintigraphy, magnetic resonance angiography (MRA), or contrast angiography in evaluating the renal arteries; however, these results have not been reproduced in many centers. Newer MRA techniques offer improved images of the main and segmental renal arteries [25].

MRI can also provide direct assessment of renal blood flow [26]. Patients with normal or mildly decreased renal function may undergo contrast-enhanced MRA. However, if there is a risk for nephrogenic systemic fibrosis (NSF) among patients with reduced renal reserves, effective nonenhanced MRA techniques for evaluating the renal arteries are also available (eg, steady-state free precession using inflow inversion recovery or 3-D phase contrast) [27]. One caveat for patients with AKI is that eGFR or creatinine levels are unreliable when non-steady-state conditions exist. An MRI with contrast should be pursued with caution in any patient with AKI until it is clear that the kidney function is stable. Bilateral renal vein thrombosis is another vascular cause of AKI that can be diagnosed using Doppler US or MRI with vascular imaging techniques. However, it, too, is an uncommon cause of AKI; no cases were found in 748 AKI episodes in a prospective, multicenter epidemiological study from Spain [28]. Trauma presents a unique constellation of possible causes of AKI, such as renal artery occlusion, severe kidney trauma, and renal vein thrombosis occurring bilaterally or in a solitary kidney. Renal trauma is more fully addressed as a separate topic. (See the ACR Appropriateness Criteria® topic on “[Renal Trauma](#).”)

If the kidneys are of normal size on the initial US examination, with or without increased echogenicity, this may represent reversible renal failure, most often AKI. A number of publications from the late 1980s and early 1990s suggested the potential of Doppler US to improve the assessment of renal intrinsic dysfunction [29]. Changes in intrarenal arterial waveforms were shown to be associated with urinary obstruction, several intrinsic renal disorders, and renal vascular disease. One example is the use of Doppler imaging to distinguish acute prerenal failure from ATN (intrarenal failure). Unlike traditional grayscale US, which shows normal kidneys in most patients with ATN, duplex Doppler US shows an elevated RI ([peak systolic velocity – end diastolic velocity]/peak systolic velocity), abnormal >0.7) in 96% of patients with ATN. However, false-negative examinations can occur with nephrotoxic drug-induced ATN [20]. Also, ATN has a higher RI than prerenal ARF, but there is some overlap as 20% of patients with prerenal ARF had resistive indexes >0.75. These inconsistent findings and discouraging clinical experiences prompted most radiologists to abandon the RI [29], and renal biopsy remains important for evaluating intrinsic renal dysfunction.

Nephrotoxic drugs and ATN following prolonged shock, with the precipitation of hemoglobin and/or myoglobin in the tubules, are other causes of AKI that may lead to abnormal CT findings [2].

If prerenal and postrenal causes of AKI have been excluded, then an intrarenal cause is likely. Radionuclide scintigraphy with technetium-labeled mercaptoacetyltriglycine (MAG-3) has been studied in this setting, but it is currently not commonly performed. A progressive parenchymal accumulation without significant excretion is suggestive of ATN. An absent uptake suggests more serious conditions, such as acute cortical necrosis and acute glomerulonephritis. In AKI, GFR is more affected than renal blood flow; hence, Tc-99m diethylene-triamine-penta-acetic acid (DTPA) accumulation is decreased, and this agent is less able to distinguish acute from chronic renal disease. However, quantitative studies with the renal tubular agents, such as Tc-99m MAG-3, can be used [30,31]. These methods assess the effective renal plasma flow (ERPF) and the degree of renal function, and they also have prognostic significance. Patients with an ERPF >125 mL/min and good uptake usually recover completely or improve markedly. ATN, hepatorenal syndrome, and acute interstitial nephritis belong in the category of good prognosis. Patients with low uptake have a poor prognosis and eventually require dialysis or transplantation.

CT is used to evaluate the trauma patient and supplement technically unsatisfactory or equivocal US findings. Excretory urography has no role in investigating AKI.

### **Chronic Kidney Disease**

CKD often presents insidiously and is characterized by a steady decrease in GFR. The most common causes of CKD are diabetic nephropathy and hypertensive nephropathy. The causes of CKD that lead to ESRD and result in transplantation are (in order of decreasing frequency): chronic glomerulonephritis, diabetic nephropathy, hypertensive nephropathy, polycystic renal disease, chronic pyelonephritis, and renal calculi [27]. Hypertensive nephropathy in one study accounted for 25% of all patients with ESRD [32]. The most common causes of CKD in children are chronic glomerulonephritis and pyelonephritis [33]. US best differentiates between obstruction and intrinsic parenchymal disease. In children with small-scarred kidneys, voiding cystourethrography (VCUG) is performed to exclude vesicoureteral reflux. For adults with ESRD and urinary tract infection or calculi, evaluation with VCUG, urodynamics, and retrograde pyelography is also advised [27,34]. The National Kidney Foundation has defined the five stages of CKD based on GFR calculations [35].

Patients with CRF, especially those on dialysis, frequently develop multiple cysts. If a patient develops  $\geq 4$  cysts in each kidney, a diagnosis of acquired cystic renal disease is made. The duration and severity of CKD is a major risk factor in cyst development, as is the length of time a patient has received dialysis. A complication of acquired cystic renal disease is the development of renal cell carcinoma (RCC). RCC in this population is estimated to be 30 times more common than in the general population. The timing of screening for RCC and the modality used are somewhat controversial, as the rate of death related to RCC in CKD patients is low. Initial screening is recommended after patients are on dialysis 3–5 years [36]. One strategy is to first evaluate for cysts using US; if cysts are encountered, then CT can be performed yearly. Early enhanced CT (40-second [s] scan delay) is recommended rather than a later delay (120 s) to better detect tumors [37]. Alternatively, MRI and US can be used. Patients with CKD on temporary dialysis should not be given intravascular iodinated contrast media. The administration of gadolinium-based MR intravascular agents in patients with CKD is also restricted (see “Anticipated Exceptions”).

In one study [38], atherosclerotic renal artery stenosis (RAS) presenting as CKD occurred in 14% of patients  $>50$  years old. Reports on the ability of duplex Doppler US to detect RAS vary widely; some reports have shown it to be as high as 90%, whereas others have shown poor results [20,38-40]. Over one-third of patients evaluated with earlier Doppler methodology had an unsatisfactory examination [41]. With use of a posterior or posterolateral translumbar approach and analysis of intrarenal vessel waveforms, duplex Doppler US has been reported to detect significant ( $>70\%$ ) RAS as a cause of renal failure, with a sensitivity of 95% and specificity of 97% [42-44]. Examinations were almost always technically feasible and accomplished within 30 minutes [43]. The study by Kliever et al [45] found it effective in evaluating RAS, but only when the RAS was  $\geq 80\%$ . Usually, high-grade stenoses are associated with renal failure. A subsequent study was not able to reproduce results adequately to support the use of duplex Doppler US as a screening test for RAS [46]. Duplex Doppler US for diagnosis of RAS is very operator-dependent [47].

Renal scintigraphy with Tc-99m DTPA and an angiotensin-converting enzyme inhibitor (ACEI) has high sensitivity and specificity in detecting RAS in patients with normal or near-normal renal function. Its use is also reported in patients with renal insufficiency [48,49]. However, it becomes less accurate in patients with CKD, because DTPA is a pure glomerular agent and there is a variable response to ACEI in patients with low baseline renal function (eg, GFR  $<15$  mL/min) [50]. It is of value to only a subset of all potential renovascular patients and is of limited value in patients with significant azotemia, bilateral RAS, or RAS to a single, functioning kidney. Therefore, it is no longer recommended by the American College of Cardiology/American Heart Association guidelines [51].

MRA is able to demonstrate, with high sensitivity and specificity, atherosclerotic narrowing of the orifice and proximal renal artery [52-54]. Aortic or proximal renal artery disease is the usual culprit when atherosclerosis causes renal failure, making MRA a helpful imaging modality [55]. Newer, ultrafast MRA techniques using intravenous gadolinium agents during breath-held imaging provide excellent images of the entire renal artery and often the segmental branches [25,56,57]. Gadolinium agents have less nephrotoxicity than conventional iodinated contrast media and, therefore, may be used in cases of mild renal insufficiency when contrast-enhanced imaging is necessary [58,59]. Furthermore, several nonenhanced MRA techniques are available and can be used to evaluate the renal arteries in patients with severe renal insufficiency without the risk of NSF [60].

Following recent advances in multidetector CT (MDCT) technology, CT angiography (CTA) has emerged as an effective alternative to MRA and duplex US for evaluating RAS, if the intravascular administration of contrast media is not contraindicated [61,62]. Both MRA and CTA, although not as sensitive as digital subtraction angiography (DSA), have been shown to be better than duplex US and radionuclide captopril renography. Currently, CTA has better spatial resolution and shorter examination times than MRA. It can also determine the extent of calcified atheromatous plaques that are not seen on MRA. The disadvantages of CTA include the radiation exposure and risk for renal damage in patients with compromised renal function [63-66].

Conventional angiography with iodinated contrast material and DSA technique should be carefully considered because of the risk of contrast nephrotoxicity. Some institutions use carbon dioxide as the contrast agent, and thereby avoid nephrotoxicity. If intra-arterial contrast is used judiciously, the ability to treat any encountered stenosis with balloon angioplasty or renal artery stent placement at the time of diagnosis makes angiography an option in patients with a high clinical suspicion for RAS. However, this approach may be appropriate in a limited number of patients, as revascularization was found to impart substantial risks but no worthwhile clinical benefit in

806 patients [67]; in 140 patients randomized to medication only or medication with stenting, there was no statistically significant difference in the progression of renal failure over 2 years [68].

Urinary obstruction as a cause of CKD is best evaluated by US. If azotemia is secondary to obstructive uropathy, hydronephrosis will almost always be demonstrable. US has sensitivity approaching 100% in moderate to severe hydronephrosis. There may be a false-positive rate  $\leq 26\%$ , caused by such entities as vesicoureteral reflux, full bladder, renal sinus cysts, and normal vessels in the renal sinus; however, vascular structures causing confusion can be resolved with duplex Doppler US or color Doppler US [69]. When the kidneys fail secondary to a chronic obstruction, resistive indexes may return to normal [21].

Newer and future techniques for detecting renal function in patients with renal failure include determining the clearance of small doses (10 mL) of low osmolar contrast media and dynamic MRI with gadolinium DTPA [56,70-75].

The nephrology literature contains several reports stressing the importance of preserving residual renal function (RRF) in patients on peritoneal dialysis or hemodialysis, who may require intravascular administration of contrast media for diagnostic purposes. The preservation of RRF, even after initiating dialysis, has been shown to result in better survival, electrolyte and fluid balance, nutritional status, and quality of life and has shown a decrease in morbidity and the need for fewer dialysis sessions (fewer or shorter dialysis sessions are cost effective). The recommendation is to continue to protect the RRF in patients on peritoneal dialysis or hemodialysis by balancing the risks versus benefits derived from using intravascular contrast media [76-78]. It should be emphasized that if a patient is on temporary (on demand) dialysis, the use of such contrast media is usually withheld. The standard of practice is to administer intravascular contrast media to patients on permanent dialysis only when indicated. This is supported by evidence that no accelerated loss of RRF was observed after the administration of intravascular contrast media in peritoneal dialysis patients undergoing diagnostic studies [79,80].

### **Renal Biopsy**

A percutaneous renal biopsy is used to diagnose renal dysfunction when pre- and postrenal causes have been excluded. It is essential in the diagnosis of glomerular, vascular, and tubulointerstitial diseases of the kidney [81]. Indications for native renal biopsy include further evaluation of proteinuria, microscopic hematuria, renal manifestations of systemic disease, and unexplained renal failure [82]. Biopsies are performed using real-time US guidance and a 16- or 18-gauge automated biopsy device. Specimens are obtained from the lower pole of the kidney with the biopsy device directed to traverse the renal cortex. Perinephric hemorrhage is the most common complication and is typically self-limited and subclinical. Gross hematuria has been reported to be as common as 5%–7% [83], but in a series of 8,573 renal biopsies in adults it occurred in only 1.9% of patients [84]. Other complications include pseudoaneurysm and arteriovenous fistula. Patients with increased serum creatinine are at an increased risk for bleeding complications [85]. It has also been reported that an age >60 years, systolic blood pressure >160 mm Hg, and ARF can increase complication rates [84].

### **Summary**

- US is the first imaging study for evaluating the patient with previously undiagnosed renal failure. It helps the clinician separate chronic ESRD from potentially reversible kidney injury by defining renal size, echogenicity, the presence or absence of hydronephrosis, and presence of cystic renal disease. US with duplex Doppler can be used by experienced laboratory personnel to screen for RAS.
- Radionuclide scintigraphy provides an assessment of global and differential renal function and potential reversibility of renal failure, but it is not believed to be generally useful in clinical decision making.
- CT is of value for ruling out stone disease, surveying the retroperitoneum for masses in patients with suspected postrenal causes of dysfunction, and the periodic evaluation of native kidneys in patients with ESRD who are at risk of developing renal cell carcinoma. Although DSA continues to be the gold standard in detecting RAS, MDCT CTA can be an alternative, noninvasive, effective diagnostic tool in patients who can receive intravascular iodinated contrast media.
- In hypertensive patients or in those with extensive peripheral atherosclerotic vascular disease, MRA with or without gadolinium contrast (depending on the GFR level), is useful for detecting RAS when duplex Doppler US is negative or nondiagnostic. MRI may also be useful in screening native kidneys with cystic changes of ESRD to detect suspected renal cell carcinomas.



- A percutaneous US-guided renal biopsy yields tissue for pathological examination in patients with intrinsic renal dysfunction, such as glomerular, vascular, or tubulointerstitial diseases.

### Relative Radiation Level Information

Potential adverse health effects associated with radiation exposure are an important factor to consider when selecting the appropriate imaging procedure. Because there is a wide range of radiation exposures associated with different diagnostic procedures, a relative radiation level (RRL) indication has been included for each imaging examination. The RRLs are based on effective dose, which is a radiation dose quantity that is used to estimate population total radiation risk associated with an imaging procedure. Patients in the pediatric age group are at inherently higher risk from exposure, both because of organ sensitivity and longer life expectancy (relevant to the long latency that appears to accompany radiation exposure). For these reasons, the RRL dose estimate ranges for pediatric examinations are lower as compared to those specified for adults (see Table below). Additional information regarding radiation dose assessment for imaging examinations can be found in the ACR Appropriateness Criteria® [Radiation Dose Assessment Introduction](#) document.

Relative Radiation Level Designations		
Relative Radiation Level*	Adult Effective Dose Estimate Range	Pediatric Effective Dose Estimate Range
○	0 mSv	0 mSv
⊕	<0.1 mSv	<0.03 mSv
⊕⊕	0.1-1 mSv	0.03-0.3 mSv
⊕⊕⊕	1-10 mSv	0.3-3 mSv
⊕⊕⊕⊕	10-30 mSv	3-10 mSv
⊕⊕⊕⊕⊕	30-100 mSv	10-30 mSv

\*RRL assignments for some of the examinations cannot be made, because the actual patient doses in these procedures vary as a function of a number of factors (eg, region of the body exposed to ionizing radiation, the imaging guidance that is used). The RRLs for these examinations are designated as “Varies”.

### Supporting Documents

For additional information on the Appropriateness Criteria methodology and other supporting documents go to [www.acr.org/ac](http://www.acr.org/ac).

### References

1. Davidson AJ, Hartman DS. *Radiology of the kidney and urinary tract*. 2nd ed. Philadelphia, Pa: WB Saunders Co; 1994.
2. Stene JK. Renal failure in the trauma patient. *Crit Care Clin*. 1990;6(1):111-119.
3. Molitoris BA, Levin A, Warnock DG, et al. Improving outcomes of acute kidney injury: report of an initiative. *Nat Clin Pract Nephrol*. 2007;3(8):439-442.
4. Rose BD. *Pathophysiology of renal disease*. 2nd ed. New York: McGraw-Hill; 1987.
5. Becker JA. Evaluation of renal function. *Radiology*. 1991;179(2):337-338.
6. National Kidney Foundation: Frequently Asked Questions About GFR Estimates. Available at: [http://www.kidney.org/professionals/kls/pdf/12-10-4004\\_KBB\\_FAQs\\_AboutGFR-1.pdf](http://www.kidney.org/professionals/kls/pdf/12-10-4004_KBB_FAQs_AboutGFR-1.pdf). Accessed October 10, 2012.
7. Cockcroft DW, Gault MH. Prediction of creatinine clearance from serum creatinine. *Nephron*. 1976;16(1):31-41.
8. Levey AS, Coresh J, Greene T, et al. Using standardized serum creatinine values in the modification of diet in renal disease study equation for estimating glomerular filtration rate. *Ann Intern Med*. 2006;145(4):247-254.
9. Ugurel MS, Hayakawa M. Implications of post-gadolinium MRI results in 13 cases with posterior reversible encephalopathy syndrome. *Eur J Radiol*. 2005;53(3):441-449.
10. Kellen M, Aronson S, Roizen MF, Barnard J, Thisted RA. Predictive and diagnostic tests of renal failure: a review. *Anesth Analg*. 1994;78(1):134-142.



11. Adcox MJ, Collins B, Zager RA. The differential diagnosis of acute renal failure. *Contemp Issues Nephrol.* 1992;25:73-117.
12. Emamian SA, Nielsen MB, Pedersen JF, Ytte L. Kidney dimensions at sonography: correlation with age, sex, and habitus in 665 adult volunteers. *AJR Am J Roentgenol.* 1993;160(1):83-86.
13. Beland MD, Walle NL, Machan JT, Cronan JJ. Renal cortical thickness measured at ultrasound: is it better than renal length as an indicator of renal function in chronic kidney disease? *AJR Am J Roentgenol.* 2010;195(2):W146-149.
14. Stuck KJ, White GM, Granke DS, Ellis JH, Weissfeld JL. Urinary obstruction in azotemic patients: detection by sonography. *AJR Am J Roentgenol.* 1987;149(6):1191-1193.
15. Gottlieb RH, Weinberg EP, Rubens DJ, Monk RD, Grossman EB. Renal sonography: can it be used more selectively in the setting of an elevated serum creatinine level? *Am J Kidney Dis.* 1997;29(3):362-367.
16. Ritchie WW, Vick CW, Glocheski SK, Cook DE. Evaluation of azotemic patients: diagnostic yield of initial US examination. *Radiology.* 1988;167(1):245-247.
17. Licurse A, Kim MC, Dziura J, et al. Renal ultrasonography in the evaluation of acute kidney injury: developing a risk stratification framework. *Arch Intern Med.* 2010;170(21):1900-1907.
18. Keyserling HF, Fielding JR, Mittelstaedt CA. Renal sonography in the intensive care unit: when is it necessary? *J Ultrasound Med.* 2002;21(5):517-520.
19. Platt JF. Duplex Doppler evaluation of native kidney dysfunction: obstructive and nonobstructive disease. *AJR Am J Roentgenol.* 1992;158(5):1035-1042.
20. Platt JF, Rubin JM, Ellis JH. Acute renal failure: possible role of duplex Doppler US in distinction between acute prerenal failure and acute tubular necrosis. *Radiology.* 1991;179(2):419-423.
21. Spital A, Valvo JR, Segal AJ. Nondilated obstructive uropathy. *Urology.* 1988;31(6):478-482.
22. Burge HJ, Middleton WD, McClennan BL, Hildebolt CF. Ureteral jets in healthy subjects and in patients with unilateral ureteral calculi: comparison with color Doppler US. *Radiology.* 1991;180(2):437-442.
23. Platt JF, Ellis JH, Rubin JM. Renal transplant pyelocaliectasis: role of duplex Doppler US in evaluation. *Radiology.* 1991;179(2):425-428.
24. Mehta RL, Pascual MT, Soroko S, et al. Spectrum of acute renal failure in the intensive care unit: the PICARD experience. *Kidney Int.* 2004;66(4):1613-1621.
25. Tello R, Thomson KR, Witte D, Becker GJ, Tress BM. Standard dose Gd-DTPA dynamic MR of renal arteries. *J Magn Reson Imaging.* 1998;8(2):421-426.
26. Dagher PC, Herget-Rosenthal S, Ruehm SG, et al. Newly developed techniques to study and diagnose acute renal failure. *J Am Soc Nephrol.* 2003;14(8):2188-2198.
27. Kabler RL, Cerny JC. Pre-transplant urologic investigation and treatment of end stage renal disease. *J Urol.* 1983;129(3):475-478.
28. Liano F, Pascual J. Epidemiology of acute renal failure: a prospective, multicenter, community-based study. Madrid Acute Renal Failure Study Group. *Kidney Int.* 1996;50(3):811-818.
29. Tublin ME, Bude RO, Platt JF. Review. The resistive index in renal Doppler sonography: where do we stand? *AJR Am J Roentgenol.* 2003;180(4):885-892.
30. Fresco GF, DiGiorgio F, Curti GL. Simultaneous estimation of glomerular filtration rate and renal plasma flow. *J Nucl Med.* 1995;36(9):1701-1706.
31. Taylor A, Jr., Manatunga A, Morton K, et al. Multicenter trial validation of a camera-based method to measure Tc-99m mercaptoacetyl triglycine, or Tc-99m MAG3, clearance. *Radiology.* 1997;204(1):47-54.
32. Zucchelli P, Zuccala A. The diagnostic dilemma of hypertensive nephrosclerosis: the nephrologist's view. *Am J Kidney Dis.* 1993;21(5 Suppl 2):87-91.
33. Frankel DG, Narla D. Imaging of children with chronic renal failure. *J Pediatr.* 1996;129(2):s33-38.
34. Confer DJ, Banowsky LH. The urological evaluation and management of renal transplant donors and recipients. *J Urol.* 1980;124:305-310.
35. Currie S, Hadjivassiliou M, Craven IJ, Wilkinson ID, Griffiths PD, Hoggard N. Magnetic resonance imaging biomarkers in patients with progressive ataxia: current status and future direction. *Cerebellum.* 2013;12(2):245-266.
36. Taylor AJ, Cohen EP, Erickson SJ, Olson DL, Foley WD. Renal imaging in long-term dialysis patients: a comparison of CT and sonography. *AJR Am J Roentgenol.* 1989;153(4):765-767.
37. Takebayashi S, Hidai H, Chiba T, Takagi H, Koike S, Matsubara S. Using helical CT to evaluate renal cell carcinoma in patients undergoing hemodialysis: value of early enhanced images. *AJR Am J Roentgenol.* 1999;172(2):429-433.

38. Scoble JE, Maher ER, Hamilton G, Dick R, Sweny P, Moorhead JF. Atherosclerotic renovascular disease causing renal impairment--a case for treatment. *Clin Nephrol.* 1989;31(3):119-122.
39. Stansby G, Hamilton G, Scoble J. Atherosclerotic renal artery stenosis. *Br J Hosp Med.* 1993;49(6):388-395, 398.
40. Taylor DC, Kettler MD, Moneta GL, et al. Duplex ultrasound scanning in the diagnosis of renal artery stenosis: a prospective evaluation. *J Vasc Surg.* 1988;7(2):363-369.
41. Berland LL, Koslin DB, Routh WD, Keller FS. Renal artery stenosis: prospective evaluation of diagnosis with color duplex US compared with angiography. Work in progress. *Radiology.* 1990;174(2):421-423.
42. Middleton WD. Doppler US evaluation of renal artery stenosis: past, present, and future. *Radiology.* 1992;184(2):307-308.
43. Schwerk WB, Restrepo IK, Stellwaag M, Klose KJ, Schade-Brittinger C. Renal artery stenosis: grading with image-directed Doppler US evaluation of renal resistive index. *Radiology.* 1994;190(3):785-790.
44. Stavros AT, Parker SH, Yakes WF, et al. Segmental stenosis of the renal artery: pattern recognition of tardus and parvus abnormalities with duplex sonography. *Radiology.* 1992;184(2):487-492.
45. Kliewer MA, Tupler RH, Carroll BA, et al. Renal artery stenosis: analysis of Doppler waveform parameters and tardus-parvus pattern. *Radiology.* 1993;189(3):779-787.
46. Kliewer MA, Tupler RH, Hertzberg BS, et al. Doppler evaluation of renal artery stenosis: interobserver agreement in the interpretation of waveform morphology. *AJR Am J Roentgenol.* 1994;162(6):1371-1376.
47. Aitchison F, Page A. Diagnostic imaging of renal artery stenosis. *J Hum Hypertens.* 1999;13(9):595-603.
48. Chen CC, Hoffer PB, Vahjen G, et al. Patients at high risk for renal artery stenosis: a simple method of renal scintigraphic analysis with Tc-99m DTPA and captopril. *Radiology.* 1990;176(2):365-370.
49. Setaro JF, Chen CC, Hoffer PB, Black HR. Captopril renography in the diagnosis of renal artery stenosis and the prediction of improvement with revascularization. The Yale Vascular Center experience. *Am J Hypertens.* 1991;4(12 Pt 2):698S-705S.
50. Davidson RA, Wilcox CS. Newer tests for the diagnosis of renovascular disease. *Jama.* 1992;268(23):3353-3358.
51. Hirsch AT, Haskal ZJ, Hertzner NR, et al. ACC/AHA 2005 guidelines for the management of patients with peripheral arterial disease (lower extremity, renal, mesenteric, and abdominal aortic): executive summary a collaborative report from the American Association for Vascular Surgery/Society for Vascular Surgery, Society for Cardiovascular Angiography and Interventions, Society for Vascular Medicine and Biology, Society of Interventional Radiology, and the ACC/AHA Task Force on Practice Guidelines (Writing Committee to Develop Guidelines for the Management of Patients With Peripheral Arterial Disease) endorsed by the American Association of Cardiovascular and Pulmonary Rehabilitation; National Heart, Lung, and Blood Institute; Society for Vascular Nursing; TransAtlantic Inter-Society Consensus; and Vascular Disease Foundation. *J Am Coll Cardiol.* 2006;47(6):1239-1312.
52. Debatin JF, Spritzer CE, Grist TM, et al. Imaging of the renal arteries: value of MR angiography. *AJR Am J Roentgenol.* 1991;157(5):981-990.
53. Kim D, Edelman RR, Kent KC, Porter DH, Skillman JJ. Abdominal aorta and renal artery stenosis: evaluation with MR angiography. *Radiology.* 1990;174(3 Pt 1):727-731.
54. Zuccala A, Zucchelli P. Ischemic nephropathy: diagnosis and treatment. *J Nephrol.* 1998;11(6):318-324.
55. Myers DI, Poole LJ, Imam K, Scheel PJ, Eustace JA. Renal artery stenosis by three-dimensional magnetic resonance angiography in type 2 diabetics with uncontrolled hypertension and chronic renal insufficiency: prevalence and effect on renal function. *Am J Kidney Dis.* 2003;41(2):351-359.
56. Bakker J, Beek FJ, Beutler JJ, et al. Renal artery stenosis and accessory renal arteries: accuracy of detection and visualization with gadolinium-enhanced breath-hold MR angiography. *Radiology.* 1998;207(2):497-504.
57. Christensson A. Renovascular disease and renal insufficiency--diagnosis and treatment. *Scand J Urol Nephrol.* 1999;33(6):400-405.
58. Bellin MF, Deray G, Assogba U, et al. Gd-DOTA: evaluation of its renal tolerance in patients with chronic renal failure. *Magn Reson Imaging.* 1992;10(1):115-118.
59. Prince MR, Arnoldus C, Frisoli JK. Nephrotoxicity of high-dose gadolinium compared with iodinated contrast. *J Magn Reson Imaging.* 1996;6(1):162-166.
60. Miyazaki M, Lee VS. Nonenhanced MR angiography. *Radiology.* 2008;248(1):20-43.
61. Aspelin P, Aubry P, Fransson SG, Strasser R, Willenbrock R, Berg KJ. Nephrotoxic effects in high-risk patients undergoing angiography. *N Engl J Med.* 2003;348(6):491-499.

62. Jo SH, Youn TJ, Koo BK, et al. Renal toxicity evaluation and comparison between visipaque (iodixanol) and hexabrix (ioxaglate) in patients with renal insufficiency undergoing coronary angiography: the RECOVER study: a randomized controlled trial. *J Am Coll Cardiol*. 2006;48(5):924-930.
63. Glockner JF, Vrtiska TJ. Renal MR and CT angiography: current concepts. *Abdom Imaging*. 2007;32(3):407-420.
64. Leiner T, de Haan MW, Nelemans PJ, van Engelshoven JM, Vasbinder GB. Contemporary imaging techniques for the diagnosis of renal artery stenosis. *Eur Radiol*. 2005;15(11):2219-2229.
65. van Helvoort-Postulart D, Dirksen CD, Nelemans PJ, et al. Renal artery stenosis: cost-effectiveness of diagnosis and treatment. *Radiology*. 2007;244(2):505-513.
66. Vasbinder GB, Nelemans PJ, Kessels AG, et al. Accuracy of computed tomographic angiography and magnetic resonance angiography for diagnosing renal artery stenosis. *Ann Intern Med*. 2004;141(9):674-682; discussion 682.
67. Wheatley K, Ives N, Gray R, et al. Revascularization versus medical therapy for renal-artery stenosis. *N Engl J Med*. 2009;361(20):1953-1962.
68. Bax L, Woittiez AJ, Kouwenberg HJ, et al. Stent placement in patients with atherosclerotic renal artery stenosis and impaired renal function: a randomized trial. *Ann Intern Med*. 2009;150(12):840-848, W150-841.
69. Scola FH, Cronan JJ, Schepps B. Grade I hydronephrosis: pulsed Doppler US evaluation. *Radiology*. 1989;171(2):519-520.
70. Dalla-Palma L, Panzetta G, Pozzi-Mucelli RS, Galli G, Cova M, Meduri S. Dynamic magnetic resonance imaging in the assessment of chronic medical nephropathies with impaired renal function. *Eur Radiol*. 2000;10(2):280-286.
71. Furukawa A, Murata K, Morita R. [Evaluation of renal function using Gd-DTPA dynamic MR imaging]. *Nihon Igaku Hoshasen Gakkai Zasshi*. 1996;56(5):264-274.
72. Gaspari F, Perico N, Ruggenenti P, et al. Plasma clearance of nonradioactive iohexol as a measure of glomerular filtration rate. *J Am Soc Nephrol*. 1995;6(2):257-263.
73. Knesplova L, Krestin GP. Magnetic resonance in the assessment of renal function. *Eur Radiol*. 1998;8(2):201-211.
74. Laissy JP, Benderbous S, Idee JM, Chillon S, Beaufile H, Schouman-Claeys E. MR assessment of iodinated contrast-medium-induced nephropathy in rats using ultrasmall particles of iron oxide. *J Magn Reson Imaging*. 1997;7(1):164-170.
75. Sterner G, Frennby B, Hultberg B, Almen T. Iohexol clearance for GFR-determination in renal failure--single or multiple plasma sampling? *Nephrol Dial Transplant*. 1996;11(3):521-525.
76. Chandna SM, Farrington K. Residual renal function: considerations on its importance and preservation in dialysis patients. *Semin Dial*. 2004;17(3):196-201.
77. Dittrich E, Puttinger H, Schillinger M, et al. Effect of radio contrast media on residual renal function in peritoneal dialysis patients--a prospective study. *Nephrol Dial Transplant*. 2006;21(5):1334-1339.
78. Sterner G, Frennby B, Mansson S, Ohlsson A, Prutz KG, Almen T. Assessing residual renal function and efficiency of hemodialysis--an application for urographic contrast media. *Nephron*. 2000;85(4):324-333.
79. Moranne O, Willoteaux S, Pagniez D, Dequiedt P, Boulanger E. Effect of iodinated contrast agents on residual renal function in PD patients. *Nephrol Dial Transplant*. 2006;21(4):1040-1045.
80. Weisbord SD, Bernardini J, Mor MK, et al. The effect of coronary angiography on residual renal function in patients on peritoneal dialysis. *Clin Cardiol*. 2006;29(11):494-497.
81. Whittier WL, Korbet SM. Renal biopsy: update. *Curr Opin Nephrol Hypertens*. 2004;13(6):661-665.
82. Korbet SM. Percutaneous renal biopsy. *Semin Nephrol*. 2002;22(3):254-267.
83. Vassiliades VG, Bernardino ME. Percutaneous renal and adrenal biopsies. *Cardiovasc Intervent Radiol*. 1991;14(1):50-54.
84. Tondel C, Vikse BE, Bostad L, Svarstad E. Safety and complications of percutaneous kidney biopsies in 715 children and 8573 adults in Norway 1988-2010. *Clin J Am Soc Nephrol*. 2012;7(10):1591-1597.
85. Whittier WL, Korbet SM. Timing of complications in percutaneous renal biopsy. *J Am Soc Nephrol*. 2004;15(1):142-147.

The ACR Committee on Appropriateness Criteria and its expert panels have developed criteria for determining appropriate imaging examinations for diagnosis and treatment of specified medical condition(s). These criteria are intended to guide radiologists, radiation oncologists and referring physicians in making decisions regarding radiologic imaging and treatment. Generally, the complexity and severity of a patient's clinical condition should dictate the selection of appropriate imaging procedures or treatments. Only those examinations generally used for evaluation of the patient's condition are ranked. Other imaging studies necessary to evaluate other co-existent diseases or other medical consequences of this condition are not considered in this document. The availability of equipment or personnel may influence the selection of appropriate imaging procedures or treatments. Imaging techniques classified as investigational by the FDA have not been considered in developing these criteria; however, study of new equipment and applications should be encouraged. The ultimate decision regarding the appropriateness of any specific radiologic examination or treatment must be made by the referring physician and radiologist in light of all the circumstances presented in an individual examination.

#### Appendix 1: Classification/Staging System for AKI [9]

Stage	Creatinine Criteria	Urine Output Criteria
1	↑ Serum Creatinine of >0.3 mg/dl or increase to ≥150% - 200% from baseline	<0.5ml/kg/hr for > 6 hrs
2	Increase serum creatinine to >200%-300% from baseline	<0.5ml/kg/hr for >12 hrs
3	Increase serum creatinine to >300% from baseline (or serum creatinine ≥4.0mg/dl with an acute rise of at least 0.5 mg/dl)	<0.3ml/kg/hr x 24 hrs or anuria x 12 hr