

Diagnostic approach to the patient with newly identified chronic kidney disease

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INTRODUCTION — Patients with kidney disease may have a variety of clinical presentations. Some have symptoms or signs that are directly referable to the kidney (such as hematuria) or to associated extrarenal manifestations (edema, hypertension, signs of uremia). Many patients are asymptomatic and are incidentally noted to have an elevated serum creatinine, which may have been (or upon further investigation is found to be) stable for years.

This topic reviews the evaluation of patients with newly identified chronic kidney disease (CKD).

The evaluation of patients who present with subacute or acute kidney injury (AKI) is discussed elsewhere. (See "[Diagnostic approach to adult patients with subacute kidney injury in an outpatient setting](#)" and "[Evaluation of acute kidney injury among hospitalized adult patients](#)".)

The management of CKD patients is discussed elsewhere. (See "[Evaluation of acute kidney injury among hospitalized adult patients](#)" and "[Overview of the management of chronic kidney disease in adults](#)".)

OVERVIEW — The evaluation of patients with an elevated creatinine of any duration includes:

- Careful history and physical examination. An important part of the history is the duration of the increased creatinine and any chronic exposures.
- Assessment of renal function by estimation of the glomerular filtration rate (GFR). Estimation of the GFR requires that patient is in steady state. (See "[Assessment of kidney function](#)".)
- Careful examination of the urine by both qualitative chemical tests and microscopic examination. The urinary findings narrow the differential. (See "[Urinalysis in the diagnosis of kidney disease](#)".)
- Radiographic imaging of the kidneys. (See "[Radiologic assessment of renal disease](#)".)
- Serologic testing and tissue diagnosis with renal biopsy if noninvasive evaluation is not sufficient for diagnosis. (See "[Glomerular disease: Evaluation and differential diagnosis in adults](#)".)

Disease duration — The determination of disease duration is an important aspect of the evaluation. Making this determination accurately requires the availability of older data for comparison. Knowing the disease duration helps to narrow the differential diagnosis of cause and to provide prognostic information to guide management.

The distinction between acute kidney injury (AKI), subacute kidney injury, and CKD is arbitrary, but the following definitions have been established by consensus panels:

- **Acute** – AKI is defined by a rise in the serum creatinine concentration or an abnormal urine output that has developed within hours to days. The criteria for AKI include an increase in serum creatinine by ≥ 0.3 mg/dL (27 micromol/L) relative to a known baseline value within 48 hours, or an increase to ≥ 1.5 times the baseline value within seven days, or a decrease in urine volume to < 3 mL/kg over six hours (Kidney Disease: Improving Global Outcomes [KDIGO]-AKI) ([table 1](#)).

AKI is most commonly diagnosed in hospitalized patients. (See "[Definition and staging criteria of acute kidney injury in adults](#)".)

- **Chronic** – The National Kidney Foundation Kidney Disease Outcomes Quality Initiative (NKF-KDOQI) and the KDIGO CKD guidelines define CKD as being present if GFR < 60 mL/min/1.73 m² or evidence of kidney damage such as albuminuria or abnormal findings on renal imaging have been present for three months or more. (See "[Definition and staging of chronic kidney disease in adults](#)", section on '[Definition of CKD](#)'.)
- **Subacute** – KDIGO guidelines proposed the term, acute kidney diseases and disorders (AKD), to encompass any decrement in renal function occurring in less than three months [1]. Disorders that evolve over more than 48 hours but generally in under three months are informally referred to as subacute kidney injury. There is considerable overlap in acute and subacute presentations.

We emphasize that terminology is less important than a clear understanding of the natural history of such diseases. The distinctions between subacute kidney injury and CKD and between subacute kidney injury and AKI can be arbitrary. As an example, a patient who presents with an elevated creatinine over 6 to 12 months or who has an acute deterioration of previously stable CKD is better evaluated as subacute kidney injury rather than CKD. (See "[Diagnostic approach to adult patients with subacute kidney injury in an outpatient setting](#)", section on '[Evaluation](#)'.)

The assessment of disease duration is best performed by comparing the current serum creatinine concentration and/or urinalysis with previous results. As an example, a patient with a current serum creatinine concentration of 4 mg/dL (354 micromol/L) and a value of 0.6 mg/dL (53 micromol/L) one month previously has acute or rapidly progressive disease. In contrast, the same patient with a prior serum creatinine concentration of 3.5 mg/dL (309 micromol/L) two years ago almost certainly has slowly progressive CKD.

When a previous urinalysis, serum creatinine concentration, and/or radiographic study are unavailable, certain findings from the history and physical examination may suggest the duration of disease [2]. As examples:

- The recent onset of symptoms or signs, such as sudden onset of anasarca and discolored urine, suggests an acute process.
- Marked oliguria (urine output < 500 mL/day) or anuria in a patient not on maintenance dialysis indicates an acute process since prolonged oliguria/anuria does not occur in slowly progressive CKD (even if advanced).
- A progressive increase in the serum creatinine concentration on a daily basis after the initial evaluation indicates an acute process while a stable value suggests CKD.
- Imaging showing small kidneys provides definitive evidence of chronicity. However, the presence of normal-sized kidneys does **not** exclude chronicity, since some causes of CKD such as diabetic nephropathy are associated with preserved kidney size. Renal parenchyma echogenicity (normally less echogenicity than of healthy liver parenchyma), if markedly increased, suggests nonspecific diffuse renal disease. Increased echogenicity combined with relatively small kidneys further supports diagnosis of

CKD [3-5]. (See ["Radiologic assessment of renal disease"](#).)

- Radiologic evidence of renal osteodystrophy such as subperiosteal bone resorption or loss of bone density at the distal third of the clavicles suggests CKD. (See ["Overview of chronic kidney disease-mineral and bone disorder \(CKD-MBD\)"](#).)

Other findings are less helpful. As an example, anemia due to erythropoietin deficiency is a common (though not absolute) finding in CKD, but many acute diseases cause both hemolysis or bleeding and AKI or subacute injury. Although hyperphosphatemia commonly affects CKD patients, it may also be seen in AKI or subacute kidney injury, so it does not distinguish acute from chronic disease. The lack of presence of anemia or hyperphosphatemia does not exclude the presence of CKD.

MAJOR CAUSES AND CLASSIFICATION OF KIDNEY DISEASE — The traditional approach to kidney disease has been to categorize the clinical etiology as prerenal (decreased renal perfusion pressure), intrinsic renal (pathology of the vessels, glomeruli, or tubules-Interstitial), or postrenal (obstructive). CKD may result from disease processes in any of these categories.

Prerenal disease — Chronic prerenal disease occurs in patients with ongoing heart failure or cirrhosis with persistently decreased renal perfusion, which increases the propensity for intrinsic kidney injury, such as acute tubular necrosis (ATN). Causes of acute or subacute prerenal injury are discussed elsewhere. (See ["Diagnostic approach to adult patients with subacute kidney injury in an outpatient setting"](#), section on ["Prerenal disease"](#).)

Intrinsic renal vascular disease — The most common chronic renal vascular disease is nephrosclerosis, which initially involves the blood vessels but ultimately damages the glomeruli and tubulointerstitium. (See ["Clinical features, diagnosis, and treatment of hypertensive nephrosclerosis"](#) and ["Hypertensive complications in black patients"](#), section on ["Risk of hypertensive complications"](#).)

Renal vascular diseases such as renal artery stenosis from atherosclerosis or fibromuscular dysplasia may, over the course of months or years, cause ischemic nephropathy, characterized by glomerulosclerosis and tubulointerstitial fibrosis [6]. (See ["Clinical features, diagnosis, and treatment of hypertensive nephrosclerosis"](#) and ["Clinical manifestations and diagnosis of chronic kidney disease resulting from atherosclerotic renal artery stenosis"](#) and ["Clinical presentation, evaluation, and treatment of renal atheroemboli"](#).)

Intrinsic glomerular disease — Chronic glomerular disease may be classified as nephritic or nephrotic.

- A nephritic pattern is suggested by an abnormal urine microscopy with red blood cell (RBC) casts and dysmorphic red cells, occasionally white blood cells (WBCs), and a variable degree of proteinuria [7,8]. (See ["Glomerular disease: Evaluation and differential diagnosis in adults"](#).)
- A nephrotic pattern is associated with proteinuria, usually in the nephrotic range (>3.5 g per 24 hours), and an inactive urine microscopic analysis with few cells or casts. (See ["Glomerular disease: Evaluation and differential diagnosis in adults"](#).)

Some patients cannot be easily assigned to one of these two categories.

Intrinsic tubular and interstitial disease — The most common chronic tubulointerstitial disease is polycystic kidney disease (PKD). Other chronic etiologies include nephrocalcinosis (most often due to hypercalcemia and/or hypercalciuria), sarcoidosis, Sjögren's syndrome, reflux nephropathy in children and young adults, and medullary cystic kidney disease in families with a pattern of autosomal dominant inheritance. (See ["Course and treatment of autosomal dominant polycystic kidney disease"](#) and ["Nephrocalcinosis"](#) and ["Renal disease in Sjögren's syndrome"](#) and ["Clinical presentation, diagnosis, and](#)

[course of primary vesicoureteral reflux](#)" and ["Autosomal dominant tubulointerstitial kidney disease \(medullary cystic kidney disease\)"](#) and ["Renal disease in sarcoidosis".](#))

There is increased recognition of relatively high prevalence of CKD of unknown cause among agricultural workers from Central America and parts of Southeast Asia. (See ["Mesoamerican nephropathy".](#))

Subacute tubulointerstitial diseases are discussed elsewhere. (See ["Diagnostic approach to adult patients with subacute kidney injury in an outpatient setting", section on 'Intrinsic tubular and interstitial disease'.](#))

Postrenal (obstructive nephropathy) — Chronic obstruction may be due to prostatic disease or abdominal/pelvic tumor with mass effect on ureter(s). Retroperitoneal fibrosis is a rare cause of chronic ureteral obstruction (see ["Clinical manifestations and diagnosis of retroperitoneal fibrosis"](#)). If untreated, obstructive nephropathy leads to irreversible tubulointerstitial fibrosis (ie, intrinsic disease). (See ["Clinical manifestations and diagnosis of urinary tract obstruction and hydronephrosis".](#))

CLINICAL MANIFESTATIONS — CKD patients may present with symptoms and signs resulting directly from diminished kidney function. These include edema, hypertension, and/or decreased urine output. However, many patients have no clinical symptoms. In such patients, kidney disease is detected by laboratory tests that are obtained as part of an evaluation of an unrelated disorder.

Depending on the duration and severity of CKD, patients may also present with symptoms and/or signs of prolonged renal failure, including weakness and easy fatigability, anorexia, vomiting, mental status changes, and seizures.

The total absence of urine (anuria) is never observed with CKD or subacute kidney injury alone and always indicates at least some component of acute kidney injury (AKI). However, anuria may be present among patients with acute superimposed on chronic kidney disease, such as is observed in a patient with chronic obstruction who develops acute urinary retention. Anuria occurs as a result of severe/prolonged shock, bilateral urinary tract obstruction, pregnancy-related cortical necrosis, or bilateral renal arterial obstruction (eg, dissecting aortic aneurysm). (See ["Evaluation of acute kidney injury among hospitalized adult patients", section on 'Clinical manifestations'.](#))

The major laboratory findings in patients with CKD include an increased serum creatinine concentration and increased urea (blood urea nitrogen [BUN]). Other common laboratory abnormalities include anemia, hyperphosphatemia, hyperkalemia, metabolic acidosis, hypocalcemia, and elevated parathyroid hormone (PTH) but not invariably.

The degree to which these abnormalities are present depends on the severity of renal dysfunction. Hyperphosphatemia is uncommon among patients with estimated glomerular filtration rate (eGFR) >45 mL/min/1.73 m². PTH, on the other hand, may be mildly elevated even with a mild reduction of eGFR (ie, 50 to 60 mL/min/1.73 m²). (See ["Overview of chronic kidney disease-mineral and bone disorder \(CKD-MBD\)", section on 'Overview'.](#))

A urinalysis may show albuminuria and/or an abnormal urine microscopy. (See ["Urinalysis in the diagnosis of kidney disease".](#))

Radiographic findings (eg, multiple renal cysts suggestive of polycystic kidney disease [PKD]) may be observed on imaging performed for some other reason.

EVALUATION — The extent of the evaluation generally depends on the severity and trajectory of creatinine abnormality and on the results of urine tests and renal imaging (often ultrasonography). Patients with a glomerular filtration rate (GFR) that does not change over sequential measurements, minimal or no

proteinuria, and no cellular elements on urine microscopy undergo a limited evaluation, and a renal biopsy is rarely performed. In such patients, the cause of CKD is usually not identified with certainty. By contrast, patients with significant proteinuria and glomerular hematuria or sterile pyuria often undergo renal biopsy to determine cause, even if the estimated GFR (eGFR) remains unchanged. Abnormalities in renal imaging may warrant urologic evaluation and urodynamic studies. (See ["Radiologic assessment of renal disease"](#).)

Among all patients, the medical history should be carefully reviewed. Longstanding diabetes and severe hypertension are common causes of CKD. A history of severe peripheral vascular disease and cardio- and cerebrovascular disease may suggest renovascular disease. (See ["Clinical manifestations and diagnosis of chronic kidney disease resulting from atherosclerotic renal artery stenosis"](#).)

A prior history of acute kidney injury (AKI), particularly if severe (ie, dialysis requiring), may suggest a cause of CKD (ie, in the absence of other causes that may be revealed by history), even if the patient describes sufficient recovery to stop dialysis. (See ["Kidney and patient outcomes after acute kidney injury in adults"](#), section on 'Kidney outcomes'.)

A careful history may also reveal exposure to lead, harsh physiological environments, or other nephrotoxins. (See ["Lead nephropathy and lead-related nephrotoxicity"](#) and ["Balkan endemic nephropathy"](#) and ["Nephropathy induced by aristolochic acid \(AA\) containing herbs"](#) and ["Cisplatin nephrotoxicity"](#) and ["Chemotherapy nephrotoxicity and dose modification in patients with renal insufficiency: Conventional cytotoxic agents"](#) and ["Chemotherapy nephrotoxicity and dose modification in patients with renal insufficiency: Molecularly targeted agents"](#) and ["Mesoamerican nephropathy"](#).)

Family history should be explored for familial diseases such as polycystic kidney disease (PKD), uromodulin kidney disease, or glomerulonephritides such as C3 glomerulonephritis or immunoglobulin A (IgA) nephropathy. (See ["Autosomal dominant tubulointerstitial kidney disease \(medullary cystic kidney disease\)"](#) and ["C3 glomerulopathies: Dense deposit disease and C3 glomerulonephritis"](#), section on 'Pathogenesis' and ["Pathogenesis of IgA nephropathy"](#), section on 'Genetic predisposition' and ["Diagnosis of and screening for autosomal dominant polycystic kidney disease"](#).)

Medications should be carefully reviewed, including historical medications. A history of prolonged use of [lithium](#), certain Chinese herbs, or analgesic combination agents may suggest a chronic interstitial lesion that caused CKD.

A constellation of symptoms and signs may suggest a particular set of disorders. Edema, heavy proteinuria, and little or no hematuria suggest a nonproliferative glomerular disease such as diabetic nephropathy.

Initial testing should include a serum creatinine for the estimation of the GFR, reagent strip urinalysis (dipstick) with urine microscopy, and the quantification of urine protein or albumin (by random or "spot" protein-to-creatinine ratio or albumin-to-creatinine ratio) ([algorithm 1](#)). The use of total protein versus albumin is debatable. A more detailed discussion is presented elsewhere (see ["Assessment of urinary protein excretion and evaluation of isolated non-nephrotic proteinuria in adults"](#)). Manual urine microscopy for the assessment of urine sediment is best performed by an experienced operator.

We perform a renal ultrasound in all patients with an increased serum creatinine of unclear duration.

For patients who are at higher risk for multiple myeloma, we obtain a serum protein electrophoresis (SPEP) and urine protein electrophoresis (UPEP), with immunofixation, and a serum light chain assay at the time of the initial evaluation. Patients who are considered at higher risk for myeloma include all patients who are >40 years of age who have a documented increase in the serum creatinine within three to six months and no other obvious cause for increased creatinine, such as nonsteroidal anti-inflammatory drug (NSAID) use. Patients who have other manifestations consistent with myeloma are also considered at high risk regardless

of whether the creatinine increase is documented to be within three to six months; such manifestations include hypercalcemia, bone pain, radiographic lesions, or anemia that is disproportionate to CKD and otherwise unexplained. In one retrospective study, testing had a higher yield for paraprotein-related kidney disease when CKD was more advanced on presentation (ie, eGFR <45 mL/min/1.73 m²) or when hypercalcemia (calcium >10.7 mg/dL) or anemia (hemoglobin <10.6 g/L) was present [9]. (See ["Clinical features, evaluation, and diagnosis of kidney disease in multiple myeloma and other monoclonal gammopathies"](#), section on 'Evaluation of kidney disease in patients with multiple myeloma or other monoclonal gammopathy'.)

The recognition of monoclonal gammopathy by serum or urine electrophoresis or by abnormal ratio of light chains in a patient with kidney disease of uncertain etiology may prompt renal biopsy for definitive diagnosis. Discussions of monoclonal gammopathy of undetermined significance (MGUS), myeloma, and amyloidosis are presented elsewhere. (See ["Clinical presentation, classification, and causes of membranoproliferative glomerulonephritis"](#), section on 'Monoclonal gammopathies' and ["Clinical features, evaluation, and diagnosis of kidney disease in multiple myeloma and other monoclonal gammopathies"](#) and ["Epidemiology, pathogenesis, and etiology of kidney disease in multiple myeloma and other monoclonal gammopathies"](#) and ["Renal amyloidosis"](#).)

The results of the urinalysis and ultrasound generally direct the remainder of the diagnostic evaluation ([algorithm 1](#)).

Patients who have evidence of obstruction on ultrasound may require further investigation and intervention to relieve the obstruction and determine the cause. (See ["Clinical manifestations and diagnosis of urinary tract obstruction and hydronephrosis"](#).)

Patients who have a urinalysis and/or albumin-to-creatinine (or protein-to-creatinine) ratio that suggests a glomerular or interstitial lesion should be further evaluated based upon the specific finding on urinalysis or based upon determination of abnormal proteinuria. (See ["Urinalysis in the diagnosis of kidney disease"](#) and ["Glomerular disease: Evaluation and differential diagnosis in adults"](#).)

Patients with sterile pyuria should be evaluated for interstitial nephritis. (See ["Clinical manifestations and diagnosis of acute interstitial nephritis"](#), section on 'Diagnosis'.)

Patients who are high risk for renovascular disease (hyperlipidemia, cigarette smoking, age greater than 50 years, coronary artery disease or peripheral arterial disease in other vascular beds) may undergo evaluation for bilateral renal artery stenosis. (See ["Clinical manifestations and diagnosis of chronic kidney disease resulting from atherosclerotic renal artery stenosis"](#).)

Patients who have a progressive increase in the serum creatinine should undergo a biopsy unless imaging shows significant evidence of chronicity. Among patients with small, echogenic kidneys, the biopsy is most likely to demonstrate chronic changes and very unlikely to yield information that could be used to inform therapy. Such patients should be followed closely and treated conservatively. (See ["Overview of the management of chronic kidney disease in adults"](#), section on 'Slowing the rate of progression' and ["Overview of the management of chronic kidney disease in adults"](#), section on 'Treatment of the complications of renal failure'.)

Renal biopsy is discussed in more detail elsewhere (see ["Indications for and complications of renal biopsy"](#)). Additional testing prior to biopsy may also be suggested by the history. As an example, determining lead levels may be indicated among patients with a history of lead exposure. (See ["Lead nephropathy and lead-related nephrotoxicity"](#).)

SOCIETY GUIDELINE LINKS — Links to society and government-sponsored guidelines from selected

countries and regions around the world are provided separately. (See ["Society guideline links: Chronic kidney disease in adults"](#).)

INFORMATION FOR PATIENTS — UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topics (see ["Patient education: Chronic kidney disease \(The Basics\)"](#) and ["Patient education: Acute kidney injury \(The Basics\)"](#))
- Beyond the Basics topics (see ["Patient education: Chronic kidney disease \(Beyond the Basics\)"](#) and ["Patient education: Dialysis or kidney transplantation — which is right for me? \(Beyond the Basics\)"](#) and ["Patient education: Hemodialysis \(Beyond the Basics\)"](#) and ["Patient education: Peritoneal dialysis \(Beyond the Basics\)"](#) and ["Patient education: Protein in the urine \(proteinuria\) \(Beyond the Basics\)"](#) and ["Patient education: Split urine collection for orthostatic proteinuria \(Beyond the Basics\)"](#))

SUMMARY AND RECOMMENDATIONS

- Chronic kidney disease (CKD) is defined as being present if the glomerular filtration rate (GFR) is <60 mL/min/1.73 m² or evidence of kidney damage such as albuminuria or abnormal findings on renal imaging have been present for three months or more. (See ['Overview'](#) above.)
- CKD patients may present with symptoms and signs resulting directly from diminished kidney function. These include edema and hypertension. However, many patients have no clinical symptoms. In such patients, kidney injury is detected by laboratory tests that may have been obtained as part of an evaluation of an unrelated disorder. (See ['Clinical manifestations'](#) above.)
- The major laboratory findings in patients with CKD include an increased serum creatinine concentration and increased urea (blood urea nitrogen [BUN]). Other common laboratory abnormalities include anemia, hyperphosphatemia, hyperkalemia, metabolic acidosis, hypocalcemia, and elevated parathyroid hormone (PTH) but not invariably. (See ['Clinical manifestations'](#) above.)
- The evaluation of patients with CKD includes careful history and physical examination, assessment of renal function by estimation of the GFR, examination of the urine by both qualitative chemical tests and microscopic examination, radiographic imaging of the kidneys, and serologic testing and tissue diagnosis with renal biopsy if noninvasive evaluation is not sufficient for diagnosis. We follow a stepwise approach ([algorithm 1](#)). (See ['Overview'](#) above and ['Evaluation'](#) above.)

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GRAPHICS

Criteria for acute kidney injury

	RIFLE [1]	AKIN [2]	KDIGO [3]
Diagnostic criteria*			
		Increase in serum creatinine of ≥ 0.3 mg/dL or $\geq 50\%$ within 48 hours OR Urine output of < 0.5 mL/kg/hour for > 6 hours	Increase in serum creatinine of ≥ 0.3 mg/dL within 48 hours or $\geq 50\%$ within 7 days OR Urine output of < 0.5 mL/kg/hour for > 6 hours
Staging criteria			
Risk (RIFLE) or stage 1 (AKIN/KDIGO)	Increase in serum creatinine of 50 to 99% OR Urine output of < 0.5 mL/kg/hour for 6 to 12 hours	Increase in serum creatinine of ≥ 0.3 mg/dL or 50 to 100% OR Urine output of < 0.5 mL/kg/hour for 6 to 12 hours	Increase in serum creatinine of ≥ 0.3 mg/dL or 50 to 99% OR Urine output of < 0.5 mL/kg/hour for 6 to 12 hours
Injury (RIFLE) or stage 2 (AKIN/KDIGO)	Increase in serum creatinine of 100 to 199% OR Urine output of < 0.5 mL/kg/hour for 12 to 24 hours	Increase in serum creatinine of > 100 to 200% OR Urine output of < 0.5 mL/kg/hour for 12 to 24 hours	Increase in serum creatinine of 100 to 199% OR Urine output of < 0.5 mL/kg/hour for 12 to 24 hours
Failure (RIFLE) or stage 3 (AKIN/KDIGO)	Increase in serum creatinine of $\geq 200\%$ OR Increase in serum creatinine by > 0.5 mg/dL to > 4.0 mg/dL OR Urine output of < 0.3 mL/kg/hour for > 24 hours or anuria for > 12 hours OR Initiation of renal replacement therapy	Increase in serum creatinine of $> 200\%$ OR Increase in serum creatinine by > 0.5 mg/dL to ≥ 4.0 mg/dL OR Urine output of < 0.3 mL/kg/hour for > 24 hours or anuria for > 12 hours OR Initiation of renal replacement therapy	Increase in serum creatinine of $\geq 200\%$ OR Increase in serum creatinine of ≥ 0.3 mg/dL to ≥ 4.0 mg/dL [¶] OR Urine output of < 0.3 mL/kg/hour for ≥ 24 hours or anuria for ≥ 12 hours OR Initiation of renal replacement therapy
Loss (RIFLE)	Need for renal replacement therapy for > 4 weeks		
End stage (RIFLE)	Need for renal replacement therapy for > 3 months		

RIFLE: risk, injury, failure, loss, ESRD; AKIN: Acute Kidney Injury Network; KDIGO: Kidney Disease: Improving Global Outcomes; ESRD: end-stage renal disease.

* AKIN and KDIGO provided both diagnostic and staging criteria. RIFLE provided a graded definition of AKI that is implicit in the staging criteria.

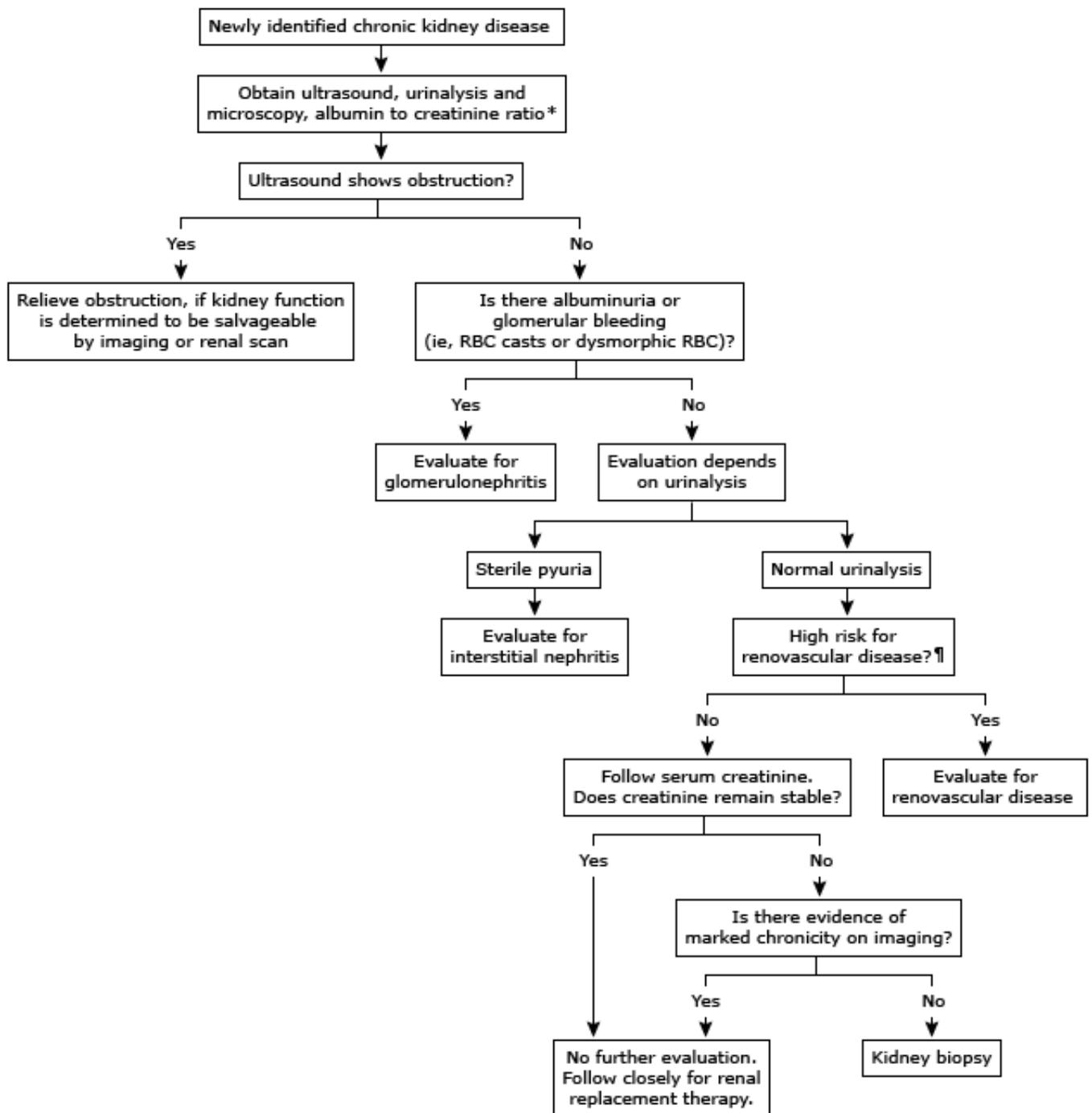
¶ In patients <18 years, stage 3 AKI is also defined by KDIGO as a decrease in estimated glomerular filtration rate (eGFR) to <35 mL/min/1.73 m².

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Graphic 83168 Version 11.0

Overview of the evaluation of newly identified chronic kidney disease



RBC: red blood cell; SPEP: serum protein electrophoresis; UPEP: urine protein electrophoresis; NSAID: nonsteroidal anti-inflammatory drug.

* Among patients at higher risk for multiple myeloma, we obtain SPEP, UPEP with immunofixation, and serum free light chains at the time of initial evaluation. Higher-risk patients are those >40 years who have a documented increase in creatinine over three to six months and have no other obvious cause for kidney dysfunction such as NSAID use or exposure to contrast.

¶¶ Risk factors for renovascular disease include hyperlipidemia, cigarette smoking, age >50 years, coronary artery disease, peripheral arterial disease in other vascular beds.

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