Clinical Validation Guidelines

RENAL FAILURE

Definition: abrupt decrease in kidney function that occurs usually within a period of hours or days.

Diagnostic Criteria [1,2]: To clinically validate the following diagnoses the listed criteria must be met AND the diagnosis must be documented by a physician in the medical records.

(N18.1-18.6, 18.9) Chronic kidney disease: kidney damage for > 3 months, defined by structural or functional abnormalities of the kidney, with or without a decreased glomerular filtration rate (GFR) that can lead to decreased GFR (See Supplements B and C). When the creatinine on admission remains elevated at about the same level during hospitalization, it suggests CKD rather than AKI.[1,4]

(N17.9) Acute renal failure (ONE or more of the following Criteria #1, Criteria #2 or Criteria #3 is needed to make the diagnosis).

1) Increase in serum creatinine (SCr) level to ≥ 1.5 x baseline (historical or measured) that is known or presumed to occur in last 7 days, **OR**
2) Increase in SCr ≥ 0.3 mg/dl comparing two separate levels, the second done within 48 hours or less of the first, **OR**
3) Urine output <0.5 ml/kg/hr for 6 hours or more

Acute renal/kidney failure and acute kidney injury are synonymous terms.[1]

The diagnosis of AKI depends on what the normal baseline value or an individual patient is, not the reference range for the lab test.[1]

When baseline creatinine is unknown, the lowest serum creatinine level obtained during a hospitalization is used to diagnose (and stage) AKI. [3] The identified baseline cannot be higher than the lowest currently measured SCr level.[3]

The criteria apply to patients with and without chronic kidney disease (CKD). However, there must be a documented baseline for the CKD. [3]

(N17.0) Acute renal failure with tubular necrosis (ALL of the following Criteria #1- Criteria #5 are needed to make the diagnosis).

1) Must meet criteria for acute kidney injury (see above), **AND**
2) Takes more than 72 hours for renal function (measured by creatinine levels) to return to or near baseline, **AND**

3) Return of creatinine levels to or near baseline occurs following effective IV fluid resuscitation/hydration, **AND**
4) There is a documented triggering event leading to the increase in serum creatinine (SCr) from baseline. Depending on the nephrotoxic exposure, the rise in creatinine may occur one to two days after the exposure (e.g. with IV radiocontrast) or be more delayed (e.g. with aminoglycosides).
5) **Additionally**, any patient with the diagnosis of acute tubular necrosis would be expected to have a documented treatment approach including the ordering of appropriate diagnostic lab studies (serial serum creatinine levels, urine studies with presence of muddy casts or epithelial cells, a urinalysis with urine sodium concentration >40 meq/L or fractional excretion of sodium (FENa) >2%), documentation of IV fluid resuscitation/hydration, review of medications and adjustments to renal doses, correction of offending causes (removal of nephrotoxic agents), placement of a Foley catheter with recording of strict ins and outs, or a nephrology consult. In extreme cases, dialysis may be required.

The distinction between pre-renal AKI and ATN is based on the clinical circumstances leading to AKI and the speed of the creatinine response to IV fluid resuscitation.[1]

Common Causes of Renal failure[2]:

- Pre-renal AKI:
  - Pre-renal: renal failure caused by decreased blood flow (hypoperfusion) to kidneys. This can occur due to hemorrhage, dehydration or hypotension. There is no true renal pathology. This is the most common type of renal failure and is almost always corrected with the administration of IV fluids.
  - Renal (intra-renal): renal failure caused by an injury to the kidney cells themselves by ischemia, infarction, necrosis or disease. The most common form of intra-renal AKI is acute tubular necrosis (ATN). Other common causes of intra-renal AKI are glomerulonephritis, hypertensive kidney disease, diabetic nephropathy and acute interstitial nephritis.
  - Post-renal: renal failure due to an obstructive process of the ureters or bladder. Common causes include kidney stones, neoplasms, adhesions, neuropathic bladder and surgical complications.
- ATN: acute kidney injury due to an ischemic or toxic injury to the renal tubular cells. The distinction between ATN and AKI is based on 1) the cause of the kidney injury and 2) the response time to IV fluid resuscitation. Common causes are IV contrast (always ATN, not AKI), sepsis, prolonged hypotension, nephrotoxic medications (NSAIDS, vancomycin, chemotherapy drugs) and rhabdomyolysis with myoglobinuria.

Clinical Indicators of Renal Failure:

- AKI: usually urine sodium concentration < 20 mEq/L and FENA <1%.
- ATN: usually urine sodium concentration >40 mEq/L, FENA >2%.
- BUN should not be used as an indicator of AKI since it can be elevated for many other reasons.
- Refer to nursing notes and intake and output (I&O) flow sheets to evaluate for the KDIGO urine output criteria of <0.5 ml/kg/hr. If low urine output is a concern, the patient may have an indwelling urinary catheter or urometer to accurately capture hourly urine output.
- Presence of dehydration or prolonged hypotension.
- Use of nephrotoxic medications and contrast substances.
- Nephrology consult and/or initiation of dialysis.

Differential Diagnoses for Renal Failure:

(N15.1) Contrast-induced nephropathy without ATN: impairment of renal function defined by either a 25% increase in SCr from baseline or a 0.5 mg/dL increase in absolute SCr value within 48-72 hours after IV contrast administration. The decreased renal function should be an acute onset, usually within two or three days. There should be no other contributing cause for the renal failure. SCr levels usually will peak between two to five days and normalize within 14 days. If it takes >72 hours to resolve it should be clarified as ATN. In cases where the creatinine returns promptly (within 48 hours or less) to baseline, it is probably not contrast-induced but rather "pre-renal" AKI.

(N17.1) Vasomotor nephropathy: a now uncommon term used for conditions that cause renal dysfunction due to an imbalance or impairment in renal vasomotor control. It is not caused by intrinsic renal injury. It is also coded as ATN (N17.1).

(T86.19) Kidney transplant dysfunction, failure and rejection: if a kidney transplant patient is admitted with AKI due to dehydration. This code captures the fact that the function of the transplanted kidney is affected by the acute renal failure, but the transplant itself has not failed.

References:

1) Tang, C., Pinson, R. (2023). CDI Pocket Guide (16th Edition).
    a. Acute Kidney Injury, pages 77-81.
    b. Acute Tubular Necrosis, pages 82-83.
2) Prescott, L., James, M. (2023). ACDIS Pocket Guide: The Essential CDI Resource. Pages 367-375.
    a. Acute Kidney Injury, pages 381-387.
    b. Acute Tubular Necrosis, pages 388-389.
3) Kidney International Supplements. (2012). Definition and Classification of AKI. *Kidney Int Suppl*, 2(1), 19-36. https://kdigo.org/wp-content/uploads/2016/10/KDIGO-2012-AKI-Guideline-English.pdf.
4) NICE Clinical Guidelines 182. *Chronic Kidney Disease: Early Identification and Management of Chronic Kidney Disease in Adults in Primary and Secondary Care*. London: National Institute of Health and Care Excellence; 2014.
5) https://read.nxtbook.com/provider_education_tools/icd_10/partnership_guide_2023/chronic_kidney_disease_ckd_.html

SUPPLEMENTS:

(A)Kidney Disease: Improving Global Outcomes (KDIGO) staging classification for acute kidney injury:

T1 KDIGO 2012-based Definition of Acute Kidney Injury

Diagnostic criteria for AKI	
• Increase in serum creatinine by ≥0.3 mg/dL (26.5 µmol/L) within 48 hrs; or • Increase in serum creatinine to ≥1.5 times baseline, known or presumed to have occurred in the past 7 days; or • Urine volume <0.5 mL/kg/h for 6 hours	
AKI Staging	
AKI Stage I	• Increase ≥0.3 mg/dL (26.5 µmol/L); or • Increase to 1.5-1.9 times from baseline; or • Urine volume <0.5 mL/kg/h for 6-12 hours
AKI Stage II	• Increase to 2.0-2.9 times from baseline; or • Urine volume <0.5 mL/kg/h for ≥12 hours
AKI Stage III	• Increase to ≥3.0 times from baseline; or • Serum creatinine ≥4.0 mg/dL (≥354 µmol/L); or • Initiation of renal replacement therapy; or • Decrease in eGFR to <35 mL/min/1.73m^2 in patients <18 years; or • Urine volume <0.3 mL/kg/h for ≥24 hours; or • Anuria for ≥12 hours

(B) Diagnostic Criteria for Chronic Kidney Disease[4]:

One of the following needs to be present for at least 3 months:

a) Decreased eGFR (<60 mL/min/1.73 m^2)

b) One or more marker of kidney damage:

i. Albuminuria (urinary albumin-to-creatinine ratio [ACR] ≥30 mg/g [3 mg/mmol])
ii. Structural abnormalities (from imaging)
iii. Urine sediment abnormalities (hematuria, red or white blood cell casts, oval fat bodies or fatty casts, granular casts, and renal tubular epithelial cells)
iv. Electrolyte and other abnormalities due to tubular disorders
v. Histological abnormalities
vi. Previous history of kidney transplantation

(C) Stages of Chronic Kidney Disease[5]:

CKD Stages		
ICD-10-CM CODE	**CODE DESCRIPTION**	**GFR**
N18.1	Chronic kidney disease, stage1	90 or above
N18.2	Chronic kidney disease, stage 2, (mild)	60-89
N18.30	Chronic kidney disease, stage 3 unspecified (moderate)	
N18.31	Chronic kidney disease, stage 3a	45-89
N18.32	Chronic kidney disease, stage 3b	30-44
N18.4	Chronic kidney disease, stage 4 (severe)	15-29
N18.5	Chronic kidney disease, stage 5	<15
N18.6	Chronic kidney disease, stage 5 requiring dialysis	Use additional code to identify dialysis status (Z99.2)
N18.6	End Stage Renal Disease (ESRD	
N18.9	Unspecified chronic kidney disease	
Note: Acute renal failure is reported with codes from category N19.		