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Acute Kidney Injuries: The Emergency Department's Approach

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Abstract

Acute kidney injury (AKI) is a widespread health problem that manifests as a variety of diseases. It has been linked to an increased risk of mortality and, in some cases, progression to chronic kidney disease, particularly in the elderly. AKI is associated with a number of other abnormalities, including metabolic acidosis and changes in body fluid volume. Although the epidemiology of AKI in hospitalised or critically ill populations is well understood and studied, the prevalence in the general emergency department (ED) population is unknown. As a result, acute AKI management and appropriate outpatient followup have not been optimised. This article provides a review of key renal disease principles as well as an evidence-based approach to AKI management in the emergency setting.

Key words: AKI • Injury • Abnormalities

Introduction

The population seen in the emergency or acute setting is frequently diverse, with complex medical histories and little access to primary care. One-third of community-acquired AKIs may be discovered in the emergency department. As a result, ED providers see a significant amount of AKI that can be treated if it is identified, managed, and discussed appropriately with these patients [1].

AKI has been defined in a variety of ways. To achieve universal agreement, the Kidney Disease: Improving Global Outcomes (KDIGO) group defined AKI in 2012 based on urine output and serum creatinine (SCr) concentration. An increase in SCr greater than or equal to 0.3 mg/dL within 48 hours, or an increase in SCr greater than 1.5 times baseline within 7 days, or a decrease in urine output for 6 hours are all considered to be elevated.

Description

RIFLE (risk, injury, failure, loss, end-stage) and Acute Kidney Injury Network (AKIN) are two other AKI classification systems that can be used. KDIGO has the advantage of covering parameters in AKIN and RIFLE, such as changes in creatinine levels within 48 hours or a decline in glomerular filtration rate (GFR) over 7 days. Prospective cohort studies show that all three criteria are effective tools for predicting mortality, with no significant differences between them.

AKI is defined as a sudden decrease in kidney function characterised by a decreased GFR as evidenced by an increased SCr or decreased urine output. AKI is further classified into three types: prerenal (a decrease in kidney blood flow), intrarenal (injury to the kidney parenchyma), and postrenal (obstructed urine flow). Intrarenal AKI is classified according to which part of the kidney is affected: the glomeruli, vasculature, or interstitium. The source of AKI must be determined because the cause and treatment differ depending on the type of insult. SCr concentration, on the other hand, may not provide an accurate, real-time assessment of AKI patients. Creatinine clearance, which is a close approximation of GFR, measures how well the glomeruli filter creatinine from the plasma. These levels may lag behind acute changes from baseline. Furthermore, SCr values are muddled by dilution, volume overload states, and creatinine production decreases during the acute phases of an illness [2,3]. Finally, muscle mass has been shown to affect relative SCr levels, which fluctuate in critical and acute care settings.

Other biomarkers, such as serum cystatin C, neutrophil gelatinase-associated lipocalin, and kidney injury molecule-1, are being studied as potential surrogates for acute kidney function for these reasons and to more accurately apply steady-state kinetics. However, SCr remains the most validated estimation of GFR to determine kidney function in the acute setting.

AKI can result in potentially fatal complications. Severe metabolic acidosis, hyperkalemia, and volume overload states such as pulmonary edoema or pericardial tamponade are among the complications. These complications can be identified using key elements from the history and physical examination. It is critical to determine whether kidney function abnormalities are normal or represent an acute injury. This is difficult to achieve in the emergency room; occasionally, the patient knows his or her baseline kidney function, or a baseline is determined through record review.

Determine the source of the injury once AKI has been identified. Before considering intrarenal sources, it is critical to rule out prerenal and postrenal sources. Prerenal causes occur after recent volume losses, such as haemorrhage, gastrointestinal or urinary fluid losses, and recent postoperative courses in which the patient was either hypotensive or exposed to iodinated contrast for imaging (although this is controversial). In patients with nocturia or urinary frequency, which may indicate prostatic or other obstruction and retention, postrenal sources, should be considered. Consider obstructive sources in cancer or trauma patients, as well as those with a solitary kidney. Because bilateral obstructive ureteral calculi are uncommon, any injury caused by obstructive ureteral calculi is considered severe.

Next, look for nephrotoxic agent exposure, especially in patients with low GFR. Antibiotics, such as vancomycin, aminoglycosides, nonsteroidal anti-inflammatory drugs, and angiotensin-converting enzyme (ACE) inhibitors/angiotensin receptor blockers are examples of common offending pharmacologic agents. Iodinated radiocontrast was once thought to be nephrotoxic; however, recent research has revealed that this link is not as strong as previously thought [4].

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Risk factors

There are numerous well-studied and well-defined risk factors for developing AKI that an emergency physician should evaluate in all patients, but especially those in critical condition. Despite advances in treatments such as dialysis, elderly patients (65 years or older) are more prone to AKI than younger patients and have a worse prognosis. Chronic diseases in older patients cause structural changes in the kidneys over time (discussed later). As a result, this vulnerable population has less kidney reserve and is more likely to develop AKI after an insult [5].

Ischemia caused by volume depletion, surgical procedures, sepsis or acute infections, and nephrotoxic agents are all risk factors for developing AKI. Chronic underlying diseases such as hypertension, diabetes, congestive heart failure, atherosclerosis, chronic kidney disease, and obstructive (postrenal) uropathy are examples of medical conditions that put patients at risk for developing AKI. Further complications, such as hospital delay, ventilator use, and concomitant diseases, have been significantly different in patients with these risk factors who develop AKI.

Causes

More than 75% of AKI patients have prerenal azotemia or acute tubular necrosis (ATN). Fluid loss, fluid sequestration, decreased cardiac output, or renal artery narrowing can all lead to prerenal failure. ATN, interstitial, glomerular, and small vessel disease are all intrarenal causes. Postrenal causes are those that occur as a result of obstruction. Ultrasound can detect masses, hydronephrosis, ureteral dilatation, and ureteral jets entering the bladder.

In summary, the pathophysiology of acute glomerulonephritis involves autoimmune-mediated inflammation that leads to glomerular apparatus fibrosis. In post-streptococcal glomerulonephritis, for example, antibodies formed against streptococcal antigen adhere to glomeruli during infection. Inflammatory pathways initiate complement and coagulation cascades, resulting in cell lysis, fibrin deposition, and, ultimately, damage to the glomeruli's native structure. Long-term intraglomerular hypertension and the destruction of normal anatomy can result in permanent scarring [6].

This review will not go into detail about the various types of acute glomerulonephritis. Postinfectious glomerulonephritis, IgA nephropathy, microscopic polyangiitis, and lupus nephropathy are a few examples. Several specialised laboratory tests, such as complement levels, help nephrologists make final diagnoses. The laboratory studies can be deferred to a specialist and are not life-threatening in an emergency. However, an emergency physician should consider the following clinical manifestations in patients who present with nephrotic syndrome: hypertension, periorbital swelling or lower leg edoema, and AKI.

Acute glomerulonephritis treatment includes supportive care as well as immunosuppressive therapies to counteract the autoimmune cascade that causes intrinsic renal damage. Early intervention in the ED with blood pressure control and, in some cases, dialysis can be lifesaving. Controlling blood pressure is critical in the acute setting because it can help slow the decline in GFR or proteinuria. Although ACE inhibitors have antiproteinuric and antifibrotic effects, they can cause further renal dysfunction in the short term. In the chronic phase, ACE inhibitors are commonly used (ie, chronic kidney disease). Use of diuretics and management of hyperkalemia, acidosis, uremia, and fluid overload are also supportive measures.

Currently, the treatment of the sepsis patient is supportive, with fluid resuscitation remaining the hallmark of care. Recently, studies comparing early goaldirected therapy to standard care in septic shock have failed to demonstrate a benefit to protocolized resuscitation with early goal directed therapy. The promise trial found no statistically significant differences in outcomes. It is critical for the emergency physician to understand these mechanisms and which patient-related risk factors, such as age, diabetes, or underlying renal insufficiency, in combination with drug interactions, can help prevent AKI secondary to medications. There are far too many drugs to name, and many of them cause AKI via multiple injury pathways. AKI is caused by a combination of drugs. Many patients, for example, are on calcium channel blockers for hypertension, but for an acute respiratory process, a macrolide may be prescribed. There is a clear link between concurrent administration of these drugs and an increased risk of AKI hospitalisation. Surveillance in the emergency department with pharmacists who can check for nephrotoxic medication and real-time AKI analysis may result in less harm.

Diagnostic examination

Laboratory values are used to diagnose AKI. Urinalysis, urine sediment, and urine chemistries should all be evaluated [7]. High blood urea nitrogen to creatinine ratio is a sign of prerenal azotemia. Because sodium is inappropriately retained in this setting, a urine sodium concentration of less than 20 mEq/L is also indicative of a prerenal cause. An ultrasound of the kidneys and postvoid bladder volumes is recommended if obstruction is suspected. Urine electrolytes and urine osmoles should be investigated further as intrarenal causes. Kidney biopsy is performed at the nephrologist's discretion and is typically reserved for patients suspected of having an intrarenal cause. Systemic lupus erythematosus, glomerulonephritis, and ATN are examples of these etiologies.

Conclusion

In an emergency situation, a referral to nephrology or urology services may be indicated. Consultation for emergent dialysis is most commonly appropriate for life-threatening acidosis, medically unresponsive electrolyte imbalances, toxic ingestion, volume overload, or uremia. AKIs suspected to be caused by intrinsic kidney disease, such as acute glomerulonephritis, should be seen by a nephrologist (typically as an inpatient). Consult a urologist if you suspect you have AKI from a postrenal cause, such as obstructive nephrolithiasis. Interventional radiology, for example, may be available on an institution-specific basis for procedures such as nephrostomy tube placement.

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Conflict of Interest

There are no conflicts of interest by author.

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