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Approach to Acute Kidney Injury: Diagnosis and Management

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Abstract

Acute kidney injury is a critical but commonly occurring medical condition that presents with a sudden decline in kidney function. This comprehensive review article provides an in-depth examination of the risk elements, etiology, diagnosis, management, and preventive approach of AKI. The causes that contribute to the development of AKI include prerenal, intrinsic renal, and postrenal. The diagnostic approach to AKI includes clinical, laboratory, and imaging studies to evaluate the root cause analysis and to find out the severity of kidney injury. Timely and accurate diagnosis is crucial for initiating appropriate management strategies. The treatment strategies may include fluid and electrolyte management, medication adjustments, nutritional support, and renal replacement therapy. The prospect of recovery diverges as it relies on the individual factors, reasons, and gravity of the condition. This review highlights the importance of raising awareness among healthcare professionals and the public about AKI, early recognition of risk factors, and prompt management. Further research is needed to explore novel therapeutic approaches and refine existing management guidelines for this critical condition.

Keywords

Acute Kidney Injury, Glomerulonephritis, Acute Tubular Necrosis, Diabetes, Hypertension, Chronic Kidney Disease, Hemodialysis

1. Introduction

Acute Kidney Injury (AKI), once often dismissed as a transitory condition, has emerged as a serious and potentially life-threatening medical challenge. This condition has gained prominence not only due to its rising incidence but also due to its significant impact on patient outcomes, healthcare resources, and

overall healthcare costs. The causes of AKI are as diverse as their clinical presentations. It can arise from various factors, including inadequate blood flow to the kidneys and direct damage to kidney tissue, adverse effects of certain medications, obstruction in the urinary tract, secondary to complications of medical conditions such as heart failure, hypotension, sepsis, etc. In recent years, AKI is no longer seen as an isolated event but is recognized as a systemic disorder affecting beyond the realm of nephrology such as cardiology, critical care, surgery, and more. This paradigm shift has prompted a reevaluation of diagnostic criteria, treatment strategies, and preventative measures. By understanding the intricate mechanisms that underlie this condition, clinicians and researchers can pave the way for earlier detection, targeted interventions, and improved patient outcomes [1] [2].

2. Epidemiology

The worldwide yearly AKI prevalence rate is reported as 13.3 million with approximately 2 million deaths. The prevalence included approximately. 23.9% adults and 13.8% children death rate. Prevalence is also high in kidney failure patients and higher in developing countries with approx. 11.3 million cases per Year [3].

In the United States, per CDC report acute kidney injury total number of cases has risen from 953,926 in 2000 to 1,823,054 in 2006 and 3,959,560 in 2014. For the same period, the rate of AKI hospitalizations has risen from 23.1 to 55.3 per 1000 persons, which is a 139% rise in diabetes patients as compared to a rise of 230% in patients with no diabetes, from 3.5 to 11.7 per 1000 persons. Comparatively lower rates of occurrences were seen in the dialysis patient population. In the US the yearly expenditure for acute kidney injury cases is $10 billion. The expenditure is directly related to the complicated nature of AKI. Studies reported that worldwide during respective hospitalization 1 in 5 adults and 1 in 3 children were impacted with AKI. Therefore, awareness of AKI should be spread among the public, government officials, and healthcare professionals [4].

3. Discussion

In the discussion, we explore deeper into AKI risk factors, causes, diagnosis, management, and preventative approach for improved patient outcomes. Clinicians must be vigilant to recognize subtle changes in kidney function and consider the broader clinical context. Some of the biomarkers that have been researched for AKI are Neutrophil gelatinase-associated lipocalin, Cell cycle arrest biomarkers, liver-type fatty acid binding protein, Interleukin-18, kidney injury molecule-1 possibly do early AKI detection [5]. Using these biomarkers with traditional markers like serum creatinine could revolutionize AKI diagnosis, enabling intervention before irreversible damage occurs. A holistic approach involving nephrologists, intensivists, cardiologists, and surgeons, is essential to comprehensively address the intricacies of AKI. The economic burden of AKI on

healthcare systems is substantial, necessitating a focus on preventative measures to ameliorate both clinical and financial implications is crucial [5] [6].

4. Risk Factors for Acute Kidney Injury

Acute kidney injury can occur in a wide range of patients and situations. Several risk factors increase the likelihood of developing AKI. Identifying these risk factors can help healthcare professionals take preventative measures and closely monitor patients at higher risk.

The probability of AKI increases with advancing age, especially in elderly individuals who may have multiple comorbidities and a decline in kidney function such as; Chronic kidney disease, uncontrolled diabetes, hypertension, cardiovascular conditions, such as a coronary artery, and heart failure, sepsis and severe infections, hypovolemia, nephrotoxic medications or nephrotoxic substances, major surgeries, and certain medical procedures can strain the kidneys, intravenous contrast media used in imaging studies, advanced liver disease, multiple organ failure, urinary tract obstructions, such as kidney stones or tumors, can factors can lead to AKI [7] [8] [9].

4.1. Etiology of AKI

The etiology of acute kidney injury is generally categorized into three types; Prerenal, intrinsic renal, and postrenal causes. Each category encompasses various conditions that can lead to the sudden decline in kidney function characteristic of AKI [2] [10] [11].

4.2. Prerenal Causes of AKI

Prerenal causes of AKI are conditions that reduce blood flow to the kidneys, leading to a decrease in kidney function. These conditions occur before the kidneys and are not directly related to intrinsic kidney damage. Prerenal AKI is often reversible if the underlying cause is identified and treated promptly. Some common prerenal causes of AKI include the ones below [2] [10] [11].

Hypovolemia can occur due to severe dehydration, excessive fluid loss from vomiting, diarrhea or excessive sweating, or inadequate fluid intake. Hypotension can result from various conditions, including severe infections, heart failure, severe bleeding, or anaphylactic shock. The condition causing decreased cardiac output such as severe arrhythmia or heart failure can lead to reduced blood flow to the kidneys. Renal artery stenosis: Real artery stenosis leads to hypoperfusion of the kidneys: This can occur in situations of shock, where there is widespread vasodilation and inadequate blood supply to the organs. Medication such as some hypertension medications NSAID—nonsteroidal anti-inflammatory drugs can cause prerenal AKI by affecting renal blood flow. Sepsis can result in significant changes in blood flow and cause prerenal AKI. Advanced liver disease, such as cirrhosis, can lead to a decrease in the effective circulating blood volume. Aortic dissection causes disturbances in blood flow including reduced

blood flow to the kidneys. Renovascular diseases affecting the blood vessels in the kidneys, such as atherosclerosis or fibromuscular dysplasia.

4.3. Intrinsic Renal Causes of AKI

These are conditions that directly affect the kidney tissues, damage, and rapidly weaken their functionality resulting in AKI. Some common intrinsic renal causes of AKI include below [2] [11] [12]:

Acute Tubular Necrosis (ATN) is a type of AKI. It occurs when there is damage to the tubular cells in the kidneys and it is often triggered by ischemia or nephrotoxic substances such as certain medications or contrast dyes used in imaging studies. Glomerulonephritis is an inflammatory process in the glomeruli that can lead to AKI. Interstitial nephritis is an inflammation of the spaces between the kidney tubules caused by infections, autoimmune medications such as NSAIDs, and antibiotics. Acute interstitial nephritis is a certain type of interstitial nephritis that develops rapidly and is often caused by an allergic reaction to certain medications [13]. Renal artery or vein thrombosis: blood clots in the renal arteries or veins can decrease blood flow to the kidneys.

Thrombotic microangiopathies are a group of disorders that cause small blood clots to form in the tiny blood vessels of the kidneys, leading to AKI. Examples include Hemolytic—uremic syndrome (HUS), thrombotic thrombocytopenic purpura (TTP). HUS is manifested by the destruction of red blood cells and reduced platelet count, and it usually occurs due to certain types of bacterial infections, such as *E. coli.*

Acute kidney injury related to systemic diseases is a complication of systemic diseases such as sepsis, lupus nephritis, or vasculitis.

Acute glomerulonephritis is a specific type of glomerulonephritis that develops suddenly and can be caused by infections, immune disorders, or other underlying conditions.

4.4. Postrenal Causes of AKI

Postrenal causes of AKI refer to conditions or obstructions that occur in the urinary tract after the kidneys. These obstructions prevent the normal urinary flow from the kidneys to the bladder leading to the buildup of pressure in the renal system, affecting kidney function. Some common postrenal causes of AKI include [2] [11] [12].

Urinary tract obstruction, blockages in the urinary tract can result from kidney stones, blood clots, tumors, or other masses that obstruct the urinary flow. Enlarged prostate such as benign enlargement of the prostate obstructing the urethra and causing difficulties in urinary outflow, leading to AKI. Ureteral obstruction like ureteral strictures, congenital abnormalities, or external compression can cause obstruction of the ureters. Bladder outlet, obstruction such as bladder stones, tumors, or neurogenic bladder dysfunction can block the outflow of urine from the bladder and cause AKI. Pelvic malignancies originating from the pelvic organs, such as cervical cancer, prostate cancer, or colorectal cancer,

can invade or compress the nearby urinary structures, leading to postrenal AKI. A urethral obstruction such as urethral strictures or other abnormalities in the urethra can obstruct the outflow of urine from the bladder.

It's crucial to recognize the risk factors and take appropriate measures to prevent AKI whenever possible. Close monitoring, early intervention, and proper management of underlying conditions are critical in reducing the occurrence and intensity of AKI in high-risk patients. Additionally, patient education about these risk factors can empower individuals to take proactive steps to protect their kidney health.

4.5. Signs and Symptoms of AKI

AKI-related signs and symptoms differ according to the intensity of the condition and the underlying cause. Below are some of the common signs and symptoms [14].

Decreased urinary output is one of the hallmark signs of AKI. In severe cases, urine output may be minimal or even absent. Swelling and fluid retention could appear in the face, legs, feet, ankles. Declining renal function accumulates toxins and waste products in the blood, leading to fatigue and weakness.

Nausea, vomiting, loss of appetite, and weight loss. Pulmonary edema leads to shortness of breath. Buildups of waste products and electrolyte imbalances can affect brain function, leading to confusion, disorientation, and even coma in severe cases. Pericardial effusion can cause chest pain or pressure.

High blood pressure, irregular heartbeat, electrolyte imbalances, and particularly high potassium levels can lead to abnormal heart rhythms. A proper medical evaluation and differential diagnosis is necessary to diagnose AKI accurately.

4.6. Stages of AKI

Based on the intensity of kidney dysfunction AKI is commonly staged using the following criteria. The appropriate management and predicting outcomes can be determined accordingly. RIFLE criteria or AKIN criteria or KDIGO criteria. These staging systems assess kidney function while utilizing the value of serum creatinine and urine output.

KDIGO criteria are widely used, and they are as follows:

AKI Stage 1: Serum creatinine 1.5 - 1.9 times than baseline or elevated ≥ 0.3 mg/dL in 48 hours, or 6 - 12 hours urinary output < 0.5 mL/kg/hour.

AKI Stage 2: Serum creatinine elevated 2.0 - 2.9 times than baseline or 12 hours urinary output < 0.5 mL/kg/hour.

AKI Stage 3: Serum creatinine elevated ≥ 3.0 times than baseline or ≥4.0 mg/dL or commencement of renal replacement treatment, or patients < 18 years, reduction in eGFR to <35 ml/min per 1.73 m^2 or urinary output < 0.3 mL/kg/hour for 24 hours or anuria for 12 hours.

RIFLE Criteria (Acute kidney injury; risk, injury, failure, loss, ESKD);

AKI Stage 1: Serum creatinine increased 1.5 times than baseline or GFR decreased > 25% or 6-12 hours urinary output < 0.5 mL/kg/hour.

AKI Stage 2: Serum creatinine increased 2 times than baseline or GFR decreased > 50% or 12 hours urinary output < 0.5 mL/kg/hour.

AKI Stage 3: Serum creatinine increased > 3 times than baseline or increased ≥ 4.0 mg/dL with an acute rise > 0.5 mg/dl or eGFR decreased > 75% or urinary output < 0.3 mL/kg/hour for 24 hours or anuria for 12 hours.

AKIN Criteria (Acute Kidney Injury Network) was developed in 2007 as an update to the RIFLE criteria, and it emphasizes changes in serum creatinine within a shorter time frame;

AKI Stage 1: Serum creatinine increased 1.5 - 1.9 times than baseline or increased ≥ 0.3 mg/dL in 48 hours, or 6 - 12 hours urinary output < 0.5 mL/kg/hour.

AKI Stage 2: Serum Creatinine increased 2 to 3 times than baseline or 12 hours urinary output < 0.5 mL/kg/hour.

AKI Stage 3: Serum creatinine increased >300% times than baseline or increased ≥ 4.0 mg/dL with an acute rise of ≥ 0.5 mg/dl or commencement on renal replacement treatment or urinary output < 0.3 mL/kg/hour for 24 hours or anuria for 12 hours [12].

The staging of AKI is a part of the evaluation and management of AKI, which involves identifying the cause and providing appropriate treatment to improve kidney function and prevent further kidney damage, it is not the same as the diagnosis of the underlying cause.

4.7. Diagnosis of AKI

The diagnosis of AKI is a mixture of clinical evaluation, laboratory tests, and imaging studies. The aim is to identify the underlying cause of AKI and determine its severity. Detailed medical history rules out any recent illnesses, medications, surgeries, or exposures to nephrotoxic substances. Physical exams assess signs and symptoms of AKI reduced urinary output, fatigue, changes in mental status, and swelling. Lab testing: urinalysis, blood Tests: serum creatinine: elevated creatinine is characteristic of kidney injury. Comparing current levels with previous records can help determine the severity of AKI. Blood urea nitrogen (BUN): Elevated BUN levels are another indicator of kidney dysfunction. Electrolytes: Abnormalities in electrolyte levels may be present due to impaired kidney function.

Imaging Studies: Imaging may be used to identify any structural abnormalities or obstructions in the urinary tract. Common imaging tests include ultrasound, CT scan, or MRI. Renal Biopsy (if necessary): In certain cases, a biopsy can help diagnose specific kidney diseases if the cause of AKI is unclear. Urine Output Monitoring: Monitoring urine output is essential in diagnosing AKI. A significant decrease in urine output over a defined period is a significant sign of AKI [9] [15] [16].

4.8. Urine Examination

Urinalysis and urine microscopy findings can provide valuable information

about the underlying etiologies of acute kidney injury. Different etiologies of AKI may be present with characteristic changes in the urine that can aid in diagnosing the cause. Prerenal AKI: Urine Osmolality: High urine osmolality (>500 mOsm/kg) due to concentrated urine as a compensatory response to reduced blood flow to the kidneys. Urine Sodium: Low urine sodium concentration (<20 mEq/L) as the kidneys retain sodium to maintain blood volume and perfusion. Urine Sediment: Typically, normal, or minimal sediment without significant cellular or granular casts. Acute Tubular Necrosis (ATN): Urine Osmolality: Low urine osmolality (<350 mOsm/kg) due to the impaired tubular reabsorption of water and solutes. Urine Sodium: High urine sodium concentration (>40 mEq/L) due to the inability of damaged tubules to reabsorb sodium. Urine Sediment: Microscopic examination may reveal renal tubular epithelial cells, granular casts, and rarely red and white blood cells. Glomerulonephritis: Urine Sediment: Microscopic examination may show dysmorphic red blood cells and casts, proteinuria.

Interstitial Nephritis: Urine Sediment: Microscopic examination may reveal white blood cells, eosinophils, and occasionally red blood cells. Renal Vasculitis: Urine Sediment: Microscopic examination may show dysmorphic red blood cells and casts, proteinuria. Obstructive Nephropathy (Postrenal AKI): Microscopic examination may reveal crystals, white blood cells, and red blood cells. The presence of obstructing casts such as calcium oxalate or uric acid may also be seen. It is important to note that these urinalysis and urine microscopy findings are not always specific to a single etiology, and different etiologies can have overlapping features. Interpreting the urinalysis and urine microscopy findings in reference to clinical evaluation and laboratory and imaging results is critical to accurately diagnose the cause of AKI and provide appropriate treatment [17] [18] [19].

4.9. Management of AKI

The management of AKI often involves a team of healthcare professionals, including nephrologists, critical care specialists, internists, and other specialists as needed. The first step is to determine the cause and address it. This may include stopping any medications that could be contributing to kidney injury, treating infections or sepsis, relieving urinary tract obstructions, managing fluid and electrolyte imbalances, and addressing other underlying conditions such as heart failure or liver disease. Maintaining a balance of fluids is essential and it could include fluid resuscitation to recuperate blood supply to the kidneys. However, excessive fluid administration should be avoided to prevent volume overload and worsening of AKI. Close monitoring and management of electrolyte levels are crucial to prevent complications like hyperkalemia or acidosis. Medicinal treatment choices are based on the etiology and intensity of AKI. To improve urinary output diuretics could be initiated in certain cases, while other medications may be used to control blood pressure, treat infections, or manage specific complications. A carefully planned diet may be provided to support kidney

function and reduce stress on the kidneys. This may include restricting certain nutrients like protein, phosphorus, and potassium. In severe cases when the kidneys are unable to adequately filter waste products and maintain fluid and electrolyte balance, dialysis may be necessary. AKI can lead to various complications, such as fluid overload, electrolyte imbalances, and infections. Preventive measures are taken to avoid these complications and promote recovery. Patients may require supportive care, including careful monitoring of vital signs, urine output, and kidney function. Monitoring allows healthcare providers to assess the response to treatment and make adjustments as needed. To prevent further kidney damage, it is essential to avoid medications or substances that can be harmful to the kidneys, especially in patients with compromised kidney function [6] [16] [17] [18].

4.10. Etiology for ATN (Acute Tubular Necrosis)

ATN is a category of AKI manifested by injury to the tubular cells of the kidneys. Several medications can be associated with the development of ATN, either due to their direct toxic effects on renal tubules or because of hypoperfusion to the kidneys. For example, NSAIDs such as diclofenac and naproxen can lead to ATN, especially when used in high doses or for prolonged periods, the risk is higher in patients with kidney disease or dehydration. aminoglycoside antibiotics such as gentamicin, tobramycin, and amikacin, intravenous contrast media used in imaging studies, ACE/ARB especially in patients with underlying renal artery stenosis or volume depletion, cyclosporine and tacrolimus immunosuppressive drugs, used in organ transplantation and certain autoimmune conditions, chemotherapy agents such as cisplatin and methotrexate, radiocontrast dyes, and certain antiviral medications such as acyclovir and foscarnet, can cause ATN when dosing is not adjusted on an individual basis. The risk of ATN from these medications varies between individuals and is influenced by factors such as age, pre-existing kidney function, hydration status, and the presence of other medical conditions. Before initiating these medications, the Healthcare providers carefully assess the risks and benefits of these medications while considering the known risk elements for ATN. Strict monitoring of kidney function and adjusting medication dosages, when necessary, can help mitigate the risk of ATN in susceptible patients [11] [19].

4.11. Medications Require Dose Adjustment or Discontinuation in AKI

Following are some key drug classes that can cause kidney injury that typically requires dose adjustment or discontinuation in AKI.

Aminoglycoside antibiotics such as gentamicin, tobramycin, and amikacin, NSAIDs such as Ibuprofen, and naproxen, ACE, and ARB, thiazide and loop diuretics, antiviral medications such as acyclovir, ganciclovir, and foscarnet, diabetic medication such as metformin, intravenous contrast agents used in imaging studies, lithium included in the treatment of the bipolar disorder, immuno-

suppressive agents like cyclosporine and tacrolimus, chemotherapy agents such as cisplatin and methotrexate. It is critical to thoroughly evaluate kidney functionality and consider dose adjustments or medication changes when managing patients with AKI. Individualized dosing based on kidney function and close monitoring are critical to avoid further harm and optimize patient outcomes [6] [18] [19] [20].

4.12. Prognosis of AKI

The prognosis of AKI depends on several elements which include the patient's individual factors such as underlying medical conditions, the cause and grade of AKI and the promptness of diagnosis and treatment, and the overall health of the patient. The outcomes of AKI could be the complete recovery of kidney function or progressing toward CKD or ESRD requiring long-term dialysis treatment or renal transplant. In patients with several comorbid conditions, AKI can lead to short-term and long-term morbidity and mortality [21] [22] [23].

4.13. Follow Up on AKI

The key aspects of the follow-up process for AKI [21]. Regular monitoring of Serum creatinine, urine output, fluid, and electrolyte balance can help prevent complications and support kidney function. Medication review and adjustment as needed. Nutrition and diet guidance to manage specific nutrients, such as protein, potassium, and phosphorus, to support kidney function and prevent further damage. Lifestyle modifications may be recommended to manage risk factors such as high blood pressure, and diabetes, or avoid nephrotoxic substances. EGFR and UACR assessment to test kidney function and recovery. Educating patients and their caregivers about AKI, its causes, prevention, and the importance of follow-up care is crucial to optimize their understanding and involvement in their care and identify and manage any risk factors that could lead to AKI recurrence in the future. Some patients may fully recover kidney function and require only periodic monitoring, while others may need long-term management if AKI leads to chronic kidney disease. Regular follow-up and adherence to the healthcare provider's recommendations are essential for optimizing kidney health and overall well-being [16] [20] [21].

Conflicts of Interest

The author has no conflict of interest.

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