ACUTE KIDNEY INJURY: ETIOLOGY AND TREATMENT

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Abstract. *Acute kidney injury (AKI), previously called acute renal failure (ARF), denotes a sudden and often reversible reduction in kidney function, as measured by glomerular filtration rate (GFR). AKI is part of a range of conditions summarized as acute kidney diseases and disorders (AKD), in which slow deterioration of kidney function or persistent kidney dysfunction is associated with an irreversible loss of kidney cells and nephrons, which can lead to chronic kidney disease (CKD). New biomarkers to identify injury before function loss await clinical implementation. AKI and AKD are a global concern. In low-income and middle-income countries, infections and hypovolaemic shock are the predominant causes of AKI. In high-income countries, AKI mostly occurs in elderly patients who are in hospital, and is related to sepsis, drugs or invasive procedures. Infection and trauma-related AKI and AKD are frequent in all regions. The large spectrum of AKI implies diverse pathophysiological mechanisms. AKI management in critical care settings is challenging, including appropriate volume control, nephrotoxic drug management, and the timing and type of kidney support. Fluid and electrolyte management are essential. As AKI can be lethal, kidney replacement therapy is frequently required. AKI has a poor prognosis in critically ill patients. Long-term consequences of AKI and AKD include CKD and cardiovascular morbidity. Thus, prevention and early detection of AKI are essential.*

Keywords: *Acute kidney injury, glomerular filtration rate, chronic kidney disease, hypovolaemic shock, high-income countries, pathophysiological mechanisms.*

ОСТРОЕ ПОВРЕЖДЕНИЕ ПОЧЕК: ЭТИОЛОГИЯ И ЛЕЧЕНИЕ

Аннотация. *Острое повреждение почек (ОПП), ранее называвшееся острой почечной недостаточностью (ОПН), означает внезапное и часто обратимое снижение функции почек, измеряемое по скорости клубочковой фильтрации (СКФ). ОПН является частью ряда состояний, обобщенных как острые заболевания и расстройства почек (ОПН), при которых медленное ухудшение функции почек или стойкая дисфункция почек связаны с необратимой потерей почечных клеток и нефронов, что может привести к хронической болезни почек (ХБП). Новые биомаркеры для определения повреждения до потери функции ожидают клинического внедрения. ОПН и ОПН являются глобальной проблемой. В странах с низким и средним уровнем дохода основными причинами ОПН являются инфекции и гиповолемический шок. В странах с высоким уровнем дохода ОПН чаще всего возникает у пожилых пациентов, находящихся в больнице, и связано с*

сепсисом, приемом лекарств или инвазивными процедурами. ОПН и ОПН, связанные с инфекцией и травмой, часто встречаются во всех регионах. Широкий спектр ОПН подразумевает разнообразные патофизиологические механизмы. Лечение ОПН в условиях интенсивной терапии является сложной задачей, включая надлежащий контроль объема, управление нефротоксичными препаратами, а также выбор времени и типа поддержки почек. Управление жидкостью и электролитами имеет важное значение. Поскольку ОПН может быть летальным, часто требуется заместительная почечная терапия. ОПН имеет плохой прогноз у пациентов в критическом состоянии. Долгосрочные последствия ОПН и ОБП включают ХБП и сердечно-сосудистую заболеваемость. Таким образом, профилактика и раннее выявление ОПН имеют важное значение.

Ключевые слова: *острое повреждение почек, скорость клубочковой фильтрации, хроническое заболевание почек, гиповолемический шок, страны с высоким уровнем дохода, патофизиологические механизмы.*

Introduction

Acute kidney injury (AKI) is a syndrome. It is an important complication in patients admitted to hospital (15–20% of all hospitalisations) and in patients in the intensive care unit (ICU) where its prevalence can sometimes exceed 55%. Despite its complexity, AKI is traditionally seen as a single disease or classified according to semianatomical categories (pre-renal, intrinsic, and postrenal AKI) in reference to the kidney.

This simplistic taxonomy is now giving way to more specific syndromic descriptions including among others hepatorenal, cardiorenal, nephrotoxic, and sepsis-associated AKI. This increased specificity is because of increasing evidence that these syndromes have a unique pathophysiology and treatment.

Etiology

The impetus for glomerular filtration is the pressure difference between the glomerulus and Bowman's space. This pressure gradient is affected by the renal blood flow and is under the direct control of the combined resistances of afferent and efferent vascular pathways. For most causes of AKI, renal blood flow reduction is a common pathologic pathway for declining GFR.

The pathophysiology of AKI has traditionally been divided into three categories: prerenal, intrinsic renal, and postrenal. Each of these categories has many different associated causes, and some causative factors of AKI have overlapping mechanisms of injury.

The prerenal form of AKI is due to any cause of reduced blood flow to the kidney. This may be part of systemic hypoperfusion resulting from hypovolemia or due to selective hypoperfusion of the kidneys, such as resulting from renal artery stenosis or aortic dissection.

However, tubular and glomerular function tends to be initially normal. A few examples of prerenal AKI mechanisms are listed below:

1. Hypovolemia: hemorrhage, severe burns, and gastrointestinal fluid losses such as diarrhea, vomiting, and high ostomy output.

2. Hypotension from decreased cardiac output: cardiogenic shock, massive pulmonary embolism, acute coronary syndrome.

3. Hypotension from systemic vasodilation: septic shock, anesthesia administration.

4. Renal vasoconstriction: NSAIDs, amphotericin B, calcineurin inhibitors, hepatorenal syndrome.

5. Glomerular efferent arteriolar vasodilation (causing intraglomerular hypotension): ACE inhibitors, angiotensin receptor blockers.

Intrinsic renal causes include conditions that affect the glomerulus or tubule, such as acute tubular necrosis and acute interstitial nephritis. This underlying glomerular or tubular injury is associated with the release of vasoconstrictors from the renal efferent pathways.

Prolonged renal ischemia, sepsis, and nephrotoxins are the most common causes. It is worth mentioning that prerenal injury can convert into a renal injury if the offending factor's exposure is prolonged enough to cause cellular damage. A few examples of this mechanism are listed below:

1. Acute tubular necrosis (ATN): ischemia from prolonged prerenal injury; drugs such as aminoglycosides, amphotericin B, and pentamidine; iodinated contrast; rhabdomyolysis; intravascular hemolysis

2. Acute interstitial nephritis (AIN): Drugs such as beta-lactam antibiotics, penicillins, NSAIDs, proton pump inhibitors (PPIs), and 5-ASA; infection; autoimmune conditions (systemic lupus erythematosus [SLE], IgG-related disease); and hereditary AIN.

3. Glomerulonephritis: anti-glomerular basement membrane disease, immune complex-mediated diseases (post-infectious glomerulonephritis, cryoglobulinemia, IgA nephropathy, IgA vasculitis).

4. Intratubular obstruction: monoclonal gammopathy, hemolytic anemia, and toxins such as ethylene glycol.

Postrenal etiology for AKI includes obstructive causes, which lead to congestion and urinary backflow of the filtration system, leading to a shift in the filtration driving forces. A noteworthy fact is that a unilateral obstruction may not always present as AKI, especially if the obstruction is gradual, because a normal working contralateral kidney may compensate for the function of the affected kidney.

Pathological disturbances can occur within 2 hours of obstruction, starting with decreased filtration at the level of the glomerulus due to increased upper urinary tract pressure. This results

in decreased renal perfusion, inflammation, tubular atrophy, and interstitial fibrosis. Eventually, bladder atony, periglomerular fibrosis, chronic interstitial nephritis can develop. The most common etiology of postrenal AKI is bladder outlet obstruction, which is often due to prostatic hypertrophy in older men, pelvic masses in older women, and nephrolithiasis in younger patients.

Renal calculi can present in the renal calyces, renal pelvis, bladder. Size and location are the determining factors of AKI, and this is a significant etiology in those with a solitary kidney.

Struvite and cystine stones grow especially rapidly and commonly cause obstruction.

Tumors, blood clots, and neurogenic bladder cause mechanical ureteral outlet obstruction.

Blood clots can be a result of bladder or urinary tract malignancy.

Urethral obstruction is the most common cause of prostate enlargement in older men. The obstruction can also be caused by retroperitoneal fibrosis, pregnancy, fecal impaction, pelvic organ prolapse.

Clinical presentation

Kidney disease is usually a silent condition. Except for urinary tract obstruction, it does not cause pain or any specific signs or symptoms. Patients can therefore present in two ways.

First, a patient might present with an acute illness such as sepsis,or be exposed to a condition known to be associated with AKI such as major surgery.Importantly, such patients might not present to the ICU and it is therefore essential that clinicians working outside the ICU are aware of the clinical presentation of kidney disease and specifically AKI. In ideal circumstances, a premorbid assessment of kidney function within the past 4 months might be available and changes from this baseline state can be detected by measuring serum creatinine or urinary output.

Second, a patient might present with abnormal kidney function of unknown duration and the clinician has to then decide if the condition is AKI, CKD, or both. This scenario can pose a substantial clinical dilemma particularly if the patient's medical history, including baseline renal function, is not well documented. Indeed, baseline renal function often has to be inferred using various sources of information including the medical history, kidney size using imaging, presence or absence of albuminuria, and the history of serum measurements of serum creatinine over time. A decrease in serum creatinine after hospital admission might indicate that AKI had occurred before admission.

Prevention of AKI

The first principle of AKI prevention is to treat its cause or trigger. The second principle is to ensure that further insults are avoided.

Systemic haemodynamics should be optimised so that, irrespective of the trigger, further damage does not occur and adequate renal perfusion and perfusion pressure are maintained. If

intravascular volume is compromised, it must be rapidly restored by administration of intravenous fluids.

Avoidance of other nephrotoxic drugs is another important step in preventing AKI or shortening its course. However, no specific drug-based intervention has been consistently and reproducibly shown to be kidney protective. Thus, there is no established pharmacotherapy for AKI. Among patients undergoing cardiac surgery, off-pump coronary artery bypass grafting has been shown to attenuate renal injury. However, the magnitude of effect is small and the inferior quality of grafting is a major concern.

Treatment

Many cases overlap between prerenal and ATN types of AKI. The best way to determine if the AKI is prerenal or not is a fluid challenge. If there is no contraindication, all patients with acute renal dysfunction should receive a fluid challenge. This requires close monitoring of urine output and renal function. If the renal function improves with fluid, this indicates prerenal AKI.

Acute tubular necrosis and other intrarenal causes are often slow to recover and can take weeks to months for complete recovery of renal function. Diuretics may be required during the oliguric phase of ATN if significant volume overload develops. It is also important to avoid further kidney insults, such as nephrotoxic drugs. In addition, many medications must be renally adjusted once a patient develops AKI. Dietary ingestion of potassium and phosphorus should also be monitored.

If hyperkalemia develops, it needs to be managed expeditiously. Approaches to lower potassium in the body include:

Dietary restriction

Insulin, IV dextrose, and beta-agonists

Potassium-binding resins

Calcium gluconate to stabilize the cardiac membrane if EKG changes are present

Dialysis for nonresponsive hyperkalemia

Some AKI patients tend to develop volume overload, which should be corrected as early as possible to avoid pulmonary and cardiac complications. Euvolemic state can be achieved with the help of diuretics, which is a cornerstone in managing such patients. Usually, high doses of IV furosemide are needed to correct volume overload in AKI patients; however, it plays no role in converting oliguric AKI to non-oliguric AKI.

Conclusion

AKI is undergoing substantial evolution in terms of definition and classification, understanding of pathophysiological mechanisms, and interaction with other disciplines and organ systems. Epidemiology describes an increasing incidence partly due to a more thorough clinical evaluation and detection.The management of patients with AKI has improved together with the improvements in hospital and intensive care quality, supported in part by sophisticated technology of extracorporeal organ support, a more personalised pharmacological therapy, and a standardised and protocolised management of physiological endpoints. In many areas, controversies still exist but consensus has been reached in several protocols and treatments so that true benchmarking and quality control are possible.

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