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Acute Kidney Injury at ICU: Review Article

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ABSTRACT

Background: Acute kidney injury is a frequent consequence among intensive care unit cases with severe illness (ICU). It is currently considered that the incidence of acute kidney injury is substantially higher than previously anticipated, with over fifty percent of ICU cases having acute kidney injury at some stage during critical illness. More than fifty percent of ICU cases with acute kidney injury and multiorgan failure are reported to die. Those requiring renal replacement treatment (RRT) have a mortality rate of up to 80 percent. Acute kidney injury is defined by an abrupt, hours-to-days-long decline in kidney function, leading in the buildup of waste products.

Objective: This review article aimed to assess and examine Cases in ICU with acute kidney injury.

Methods: A comprehensive search was conducted in PubMed, Google Scholar, and Science Direct for information on acute kidney injury, ICU, Kidney, Liver function and RRT. However, only the most current or comprehensive study from May 2011 to November 2022 was considered. The authors also assessed references from pertinent literature. Documents in languages other than English have been disregarded since there are not enough resources for translation. Unpublished manuscripts, oral presentations, conference abstracts, and dissertations were examples of papers that were not considered to be serious scientific research.

Conclusion: Acute kidney injury is responsible for poor outcome in hospitalised cases. In critical cases, the underlying cause of acute kidney injury is renal hypoperfusion during shock episodes. Therefore, prevention is the most effective therapy.

Keywords: Acute kidney injury, ICU, Kidney, Liver function, RRT.

INTRODUCTION

Acute kidney injury is now recognized as a serious health issue affecting many people from all over the world and resulting in lower survival and can progress to CKD. It is frequently encountered alongside other acute diseases and is prevalent among severely unwell individuals. Acute kidney injury also plays a significant role, since it is substantially related with increased expenditures of care, worse outcomes, and reduced QOL following release from hospital. Impact and prognosis of acute kidney injury varies substantially based on severity, clinical context, concomitant variables, and geographic location [(1)].

Acute kidney injury is prevalent in the ICU, and its frequency is rising. The reported mortality of Cases in ICU with acute kidney injury differs between research based on the criteria of diagnosis and the patient population investigated. In the majority of studies, death rises proportionally with the severity of renal damage. Cases that require renal replacement therapy (RRT) have a fifty to seventy percent death rate. Although acute kidney injury necessitating RRT in the ICU is a well-known independent risk factor for in-hospital mortality, even minor increases in serum creatinine (SCr) are linked with an increased risk of death [(2)].

Acute kidney injury is characterised by quick reduction in glomerular filtration rate (GFR), which results in accumulation of metabolites from metabolism and fluid, electrolytes, and acid-base disturbance. A diverse syndrome characterised by hemodynamic abnormalities that disrupt normal renal perfusion and reduce GFR without injury to the parenchyma, partial or total blockage to UOP, resulting in glomerular, interstitial, tubule, or vascular malfunction. Hemodynamically induced pre-renal dysfunction and acute tubule necrosis (ATN) owing to ischemia-reperfusion damage, toxic exposure, or infection are the most prevalent causes of acute kidney injury in critically sick cases [(3)].

Epidemiology

Acute renal damage varies by population type and characteristics. Utilizing the existing 0.3 mg/ml change in SCr cutoff, available data indicates that the incidence of acute renal damage in hospitalised patients varies from three to fifty percent and from 10 to 70 percent in the ICU. A 2013 meta-analysis of the incidence of acute renal injury according to the kidney enhancing global outcomes stage system with a total of 3,585,911 persons indicated occurrence in 23 percent of all hospitalised cases [(1)].

Numerous cohort studies have been conducted on critically sick cases to determine the prevalence of acute kidney injury in the ICU. According to conclusive statistics, the prevalence is as high as 70% in some groups. Cases with ICU-related acute kidney injury are younger, mostly males, and more likely than those with isolated acute kidney injury to have acute kidney injury coupled with multisystem organ failure. Sepsis is the most significant acknowledged risk factor for acute kidney injury in the ICU. Other significant risk factors include a history of DM, HTN, or CKD, concurrent use of vasopressors, and mechanical ventilation. The rates of RRT and mortality due to acute kidney injury are

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considerably greater in ICU cases compared to ward cases [4].

Community-acquired acute kidney injury, evident at admission to the ICU, and hospital-acquired acute kidney injury have been identified as two separate patterns of acute kidney injury in cases in ICU. Cases with hospital-acquired acute kidney injury had worse outcomes and more RRT requirements [5].

Pathophysiology

There are three basic etiologic groups for acute kidney injury: pre-renal, intrinsic, and post-renal. Pre-renal denotes to decrease blood supply to the kidney with no renal parenchyma disease, this kind of acute organ failure is prevalent in ICU cases. Acute urinary tract blockage differentiates between post-renal or obstructive acute kidney damage. Regarding internal malfunction, acute renal parenchyma damage develops, including ATN, acute interstitial, glomerular nephritis (AIN) (GN). Pre-renal, internal, and post-renal are terms used to classify pathophysiologic aspects, not diseases. Long considered synonymous with "hypovolemic acute kidney injury" and "fluid responsiveness s," the words "pre-renal acute kidney injury" and "transient acute kidney injury" should no longer be employed in this context [6].

1. "Prerenal" acute kidney injury

It is the most prevalent pathophysiological causes, accounting for 30–60 percent of all cases in the ICU. When the capability of the typical physiological responses to hypovolemia is surpassed, prerenal acute kidney injury occurs. This reaction begins with a fall in mean arterial pressure (mAP), which triggers baroreceptors that stimulate the sympathetic nervous system, RAAS, and the ADH-V. Renal sympathetic stimulation results in the constriction of afferent arterioles and the release of renin from the JXG apparatus. In addition, alterations in intrarenal hemodynamics induce renin secretion directly in response to hypovolemia. The net result is synthesis of angiotensin II, which causes renal arteriolar VC and a reduction in filtration rate to maintain circulation volume at normal levels by the production of smaller amount of urine deficient in salts, which is the net result of the overall effects of hypertension [7]. Decreased perfusion pressure (or elevated renal VP) and afferent arteriolar constriction reduce the glomerular capillary hydrostatic pressure and consequently lowering GFR. Prerenal acute kidney injury may be caused by ECF volume loss or alteration, reduced cardiac output, systemic VD, intrarenal VC, or high renal VP [8].

2. "Renal" acute kidney injury

Tubule, interstitial, glomerular, and vascular processes are typically used to classify on the basis of the afflicted nephron area. ATN is the most prevalent intrinsic cause, responsible for 85–90% intrinsic acute kidney injury in ICUs. Ischemia-reperfusion damage, nephrotoxic, and septic are the three main causes of ATN. ATN linked with sepsis has distinctive characteristics [9].

3. "Post-renal" acute kidney injury

Blockages occur for fewer than 5% of admissions to the ICU on average. Upper tract obstruction refers to obstructions that occur above the bladder. Upper tract blockage requires bilateral occlusion or unilateral obstruction in the presence of a single working kidney [10]. Cases with obstructive disease may exhibit anuria, normal or increased urine volume, or fluctuating urinary outflow obstruction with durations of anuria intermixed with flow of urine when renal pelvis pressure increases and overwhelms the obstruction. During the acute phase of blockage, intra-tubule pressure is greater than renal VP, removing it from the equation for net filtration pressure. When intra-tubule pressure hits mAP, the negative filtration pressure falls below the autoregulation limit and is frequently almost nil [11].

Diagnosis

It is diagnosed when SCR rises by 0.3 mg/dL (26.5 mol/L) within 48 hours, or when SCR rises to 1.5 times baseline within 7 days, or when urine volume is 0.5 mL/kg/h for 6 hours [12].

• Assessment of intravascular volume (IVV)

The true difficulty is in determining the aetiology. The initial step is to evaluate the patient's fluid volume status. This may be performed by determining the fluid volume balance over the previous days, the blood pressure trend, and the IVV. IVV is inferred from the presence or absence of fluid responsiveness rather than measured directly. In ICUs, echocardiography that measures the diameter of the IVC and its fluctuation with respiration cycle has become increasingly accessible. Positive predictive value (PPV) for fluid responsiveness indicating IVV deficit was more than 90 percent in mechanical ventilation with sepsis and larger than 12 percent fluctuation in IVC diameter [13]. Changes in central venous pressure (CVP) might offer information regarding IV volume deficit. In an ICU, a drop of 1 mmHg in CVP during spontaneous inspiration or mechanical breath had a PPV of 84 percent and a NPV of 94 percent for response to fluids [14].

• Urine and serum biochemical analyses

Diagnostic indicators of the urinary tract may have confirmatory value. Concentrated urine with SG > 1.020, BUN/Cr > 20:1, urine Na 20 mEq/L, or low FeNa 1.0 percent is associated with fluid volume deficit or renal hypoperfusion indicative of prerenal acute kidney injury. In contrast, increasing SCR with FeNa > 2.0 percent strongly indicates renal cause. In septic cases with acute kidney injury, biochemical examination of urine utilising routine measures of Na, urea, and creatinine and calculating different indices of tubule function, FeNa, and FeUrea are inaccurate, or

therapeutically beneficial [16]. Fluid challenge restored oliguria in only half of individuals with oliguria, and neither urine Na, FeNa, nor FeUrea was an accurate predictor of renal response to challenge by fluids [17]. Therefore, these measures should be evaluated in combination with hemodynamic measurements and in the context of the clinical setting.

• Analysis of the urine

Microscopical sedimentation in urine can give a clue about the cause of kidney affection. In prerenal cause, urine sediment is typically diluted, with minor hyaline casts and little cell loss. ATN may be the cause based on finding epithelial cell casts and brownish granular casts. Casts of white blood cells are common in AIN and acute pyelonephritis. The presence of red cell casts indicates glomerular illness, such as GN or renal vasculitis. Not only may urine sediments assist distinguish prerenal from renal acute kidney injury, but they can also offer information about the location of nephron damage [2]

Fluid administration

• Fluid volume expansion

If a clinical examination reveals an IV volume deficit, optimising the patient's hemodynamic condition and correcting deficit volume should have a positive impact on renal function and aid in preventing the advancement of renal impairment. To minimise excessive fluid administration, resuscitation should be undertaken while monitoring dynamic changes in UOP, blood pressure, or CVP [18]. Investigation of 105 ICU cases demonstrated a linear relationship between elevated CVP levels and the onset or duration of damage. This happens due to increasing venous engorgement and a lowering filtration pressure, resulting in a reduction in GFR [19]. Observational research shows that low-chloride buffered crystalloids may be linked to a lower risk of pulmonary embolism [20]. This claim is supported by the unphysiological Cl^- concentration of normal saline (154 mEq/L), which can cause kidney VC, decreased GFR, and acidosis [21, 22]. Colloid solutions containing albumin can treat hypoalbuminemia, hypotension, and acute renal damage. In conjunction with vasopressors, infusion of albumin is also advised for patients with liver cirrhosis who have hepatorenal syndrome [23].

• Vasopressors

In sepsis with acute renal damage, noradrenaline is the most appropriate vasopressor, with a target mAP of 65–70 mmHg. In situations of chronic HTN, a higher target range of 80–85 mmHg is recommended. The target range for mAP of 65–70 mmHg is equally applicable to persons with hepatorenal syndrome. All vasoconstrictors used to treat hepatorenal syndrome should be coupled with albumin [24].

• Diuretics

Previous research has demonstrated that extended usage of Furosemide improves prognosis despite a rise in SCR (25-27). A study indicated that oliguric patients with a UOP of at least 100 ml/h after receiving a test dose of 1.0–1.5 mg furosemide per kilogram had a decreased likelihood of progressing to a more severe stage of renal disease [28]. In a multicenter, randomised, controlled investigation of adult patients, low-dose furosemide (0.4 mg/kg loading dose followed by 0.05 mg/kg/h continuous infusion) had no effect on incidence, recovery, or the need for RRT [29]. Recently many scientific societies do not recommend using furosemide in acute kidney damage. Furosemide should be given to cases with acute kidney damage and hypervolemia, such as those with cardiorenal syndrome and decompensated HF. In this situation, diuretics have not been observed to cause toxicity [30].

Replacement therapy

RRT is the foundation of supportive therapy for severe cases. It allows the elimination of hypervolemia and electrolytes that build during renal failure is one of the goals of RRT. Intermittent dialysis (IHD), continuous renal replacement treatment (CRRT), and the prolonged intermittent RRT (PIRRT; alternatively extended duration dialysis [EDD] are the available RRT modalities [31]. During RRT, solute removal may proceed by diffusion along a concentration gradient from blood over a semipermeable membrane into dialysate, or via convective transport of solute across the membrane during filtration. The composition of the dialysis solution resembles the typical electrolyte structure of ECF and produces balance in the circulation by balancing solute concentrations [32].

The CRRT may employ diffusive dialysis, convective hemofiltration, or a mix of the two. Dialysate flow rate (DFR) is the primary distinction between intermittent and continuous dialysis, in addition to treatment time. DFR (usually 500–800 mlper minute) are equivalent to or larger than blood DFRs in intermittent dialysis, allowing for fast solute removal. In continuous dialysis, the DFR (usually 15–30 mlper minute) is slower than the blood flow rate but more effective due to prolonged duration of dialysis, allowing virtual equilibration of LMW solutes like urea between the blood and dialysate. Thus, the solute clearance for solutes with LMW is comparable to the DFR [32].

Continuous hemofiltration generates a high Fr and administers physiologic replacement fluid at the same pace. Negative fluid balance (ultrafiltration) is achieved by providing fewer milliliters per hour (often between 50 and 400 ml/h). The elimination of solutes happens only by convection, and the clearance is about equivalent to the ultraFr. Convective transport is essentially restricted by the pore size of the membrane, hence hemofiltration enables more effective clearance

of greater MW (>500–15,000 KD) solutes than convective transport [33]. Although it has been hypothesised that the removal of greater molecular weight solutes with hemofiltration as opposed to dialysis would be clinically advantageous, clinical investigations have not supported this hypothesis. Due to its longer duration, CRRT requires a lower net ultraFr than IHD to achieve the same daily fluid removal. In general, CRRT is seen as causing less hemodynamic instability than standard IHD. PIRRT is a variation of traditional IHD that use lower blood and DFR while extending the treatment time to 8–16 hours [34].

Conventional criteria for initiating RRT are hypervolemia not overcome with diuretics, electrolytes and pH abnormalities unresponsive to correction and cardiac or CNS affection. Most cases in the ICU are not hospitalized long enough to exhibit the majority of symptoms. RRT is precious for some circumstances, however its utility in other situations where the modifications do not pose an urgent threat to life is dubious [35].

CONCLUSIONS

Acute kidney injury is responsible for poor outcome in hospitalised cases. In critical cases, the underlying cause of acute kidney injury is renal hypoperfusion during shock episodes. Therefore, prevention is the most effective therapy. In addition, hemodynamic optimization and the reduction of offending variables are essential. There are several RRT techniques available, and they should be complementary. The optimal beginning time varies on the demands of each patient, and benefits and risks should be weighed regularly during each patient's progression.

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