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Review Article

RECENT ADVANCE TREATMENT OF ACUTE KIDNEY INJURY- AN OVERVIEW

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Abstract

Objective: Acute Kidney Injury (AKI) is a critical medical condition characterized by a rapid decline in renal function, often associated with significant morbidity and mortality. This comprehensive review article provides an in-depth analysis of the current state of knowledge regarding AKI, focusing on recent advancements in its understanding and management. The review begins with an overview of the epidemiology, etiology, and risk factors of AKI, emphasizing the growing prevalence and the impact of various comorbidities on its incidence. It then delves into the pathophysiological mechanisms underlying AKI, including ischemia-reperfusion injury, inflammation, oxidative stress, and mitochondrial dysfunction, shedding light on the complex interplay of these factors. Recent developments in diagnostic tools for early AKI detection are extensively discussed, highlighting the potential for improved outcomes through timely intervention. Furthermore, the article explores emerging therapeutic strategies, such as fluid management, nephrotoxic drugs and agents, Haemodynamic management, renal replacement therapy and extracorporeal support, and extracorporeal support, emphasizing the need for personalized treatment regimens. In addition to medical interventions, a comprehensive overview of preventive measures and strategies to mitigate AKI risk, particularly in high-risk populations, is provided. The importance of a multidisciplinary approach involving nephrologists, intensivists, and primary care physicians is underscored throughout the review. By synthesizing the latest research findings and clinical practices, this review aims to contribute to the ongoing efforts to enhance our understanding of AKI, improve early diagnosis, and develop more effective management strategies, ultimately striving for better outcomes and reduced mortality rates in patients suffering from this devastating condition.

Keywords: Acute Renal Failure, Renal Replacement Therapy, Haemodialysis, Electrolytes, Vancomycin, Kidney Disease Improving Global Outcomes.

1. Background

Acute renal failure (ARF) is a complicated illness that can appear clinically in a variety of ways, from a slight increase in serum creatinine to anuric renal failure. It frequently goes unrecognized and has serious repercussions. Recent epidemiological studies show the wide range of etiology and risk factors, describe the increased mortality linked to this disease (especially when dialysis is required), and imply a connection to the progression of chronic kidney disease (CKD) and dependence on dialysis[1,2]. There is mounting evidence

that even small variations in serum creatinine are linked to higher inpatient mortality. Over the past few decades, ARF has been the subject of intensive clinical and basic research efforts[3-6]. An important public health issue with high morbidity, mortality, and healthcare expenditures is acute renal failure (ARF). No therapeutic approach, besides dialysis, consistently increases survival, reduces damage, or hastens recovery[7]. Acute kidney injury (AKI) which is only present for seven days, is identified by increased serum creatinine levels, a sign of renal excretory function, and decreased urinary output (oliguria), a quantitative marker of urine production[8]. A common illness called acute kidney damage (AKI) is independently linked to higher mortality. A definition needs to be standard to improve the effectiveness of clinical care and research. The definition of AKI has significantly altered since 2004 when the Risk, Injury, Failure, Loss, and End-Stage Renal Disease (RIFLE), AKI Network (AKIN), and Kidney Disease Improving Global Outcomes (KDIGO) categories were developed[9].

2. Epidemiology

A common and significant diagnostic and therapeutic problem for clinicians is acute renal damage. The incidence varies between definitions and populations, ranging from more than 5000 cases per million per year for acute renal injury that does not require dialysis to 295 cases per million per year for disease that does[10,11]. The disease occurs 19% of the time in hospital inpatients, and it is most prevalent in critically sick patients, in whom acute renal injury is more common than 40% at the time of intensive-care unit admission if sepsis is present[12]. On the day following admission to an intensive care unit, the prevalence is greater than 36%, and during admission to an intensive care unit, the prevalence is larger than 60%8. Data from 3,585,911 people, the majority of whom resided north of the equator (84% HIC), were collected as part of a meta-analysis of 154 studies that classified AKI according to the 2012 Kidney Disease Improving Global Outcomes (KDIGO) classification[13,14]. This analysis revealed that 8.3% of ambulatory patients and 20.0-31.7% of patients receiving varying levels of in-hospital care had community-acquired AKI. Others report substantially lower incidences, which could be related to local conditions and AKI classifications. The average pooled death rate was 23%, but those requiring KRT had a rate of 49.4%15,[16].

3. Clinical Presentation

Depending on the environment, the appearance may be subtle. Patients who are in the hospital may acquire ARF following a catastrophic occurrence; outpatients frequently are not in acute distress. (a) In an outpatient context, symptoms could include weight gain, a change in urination patterns, or flank pain. ARF signs are often identified by clinicians prior to inpatients. (b) Edema, colourful or frothy urine, and orthostatic hypotension in patients with volume depletion are symptoms.

Table 1: RIFLE criteria for acute kidney injury Adapted from Bellomo and colleague

	GFR/Scr criteria	Urine output criteria
Risk	Scr ↑ 50% or GFR ↓ 25%	<0.5 ml/kg/hour × 6 hours
Injury	Scr ↑ 100% or GFR ↓ 50%	<0.5 ml/kg/hour × 12 hours
Failure	Scr ↑ 200% or GFR ↓ 75%	>0.3 ml/kg/hour × 24 hours
Loss	Persistent ARF (>4 weeks)	
ESRD	ESRD (>3 months)	

RIFLE criteria for acute kidney injury Adapted from Bellomo and colleagues. As GFR or UO deteriorate, the patient moves from risk (class R) to failure (class F). Class R has a high sensitivity and class F a high specificity for acute kidney injury. RIFLE=risk, injury, failure, loss, end stage. GFR=glomerular filtration rate. Scr=serum creatinine concentration. UO=urine output. ESKD=end-stage kidney disease[23].

4. Etiology

There are numerous typical reasons for AKI in seriously unwell patients. AKI is primarily caused by sepsis in about 50% of patients[17-19]. According to a number of studies, sepsis-induced AKI increases both the short- and long-term risk of dying[20,21]. In a 2010 study, we investigated the risk of acute kidney injury (AKI) in 1,836 hospitalized patients with community-acquired pneumonia, a typical infectious reason for hospitalization in affluent nations[21]. According to RIFLE criteria, we discovered that 34% of all hospitalized patients with pneumonia and over 25% of those with non-severe pneumonia experienced AKI[22].

5. Pathophysiology

Vasculitis and glomerulonephritis are two inflammatory diseases that affect the kidney parenchyma. Their etiology is complicated and involves nearly every element of the innate inflammatory system as well as antibody-mediated and immune-cell mediated pathways. Because acute kidney injury related to prerenal causes is the most prevalent type in industrialized nations, hospital inpatients, and particularly critically ill patients, it is the emphasis of this seminar[24-28]. Animal studies have contributed significantly to our understanding of the pathogenesis of prerenal acute kidney damage. The several pathways that are probably involved as well as the causes of organ harm are shown in studies of models of acute ischaemia brought on by acute blockage of the renal artery. Leucocytes enter the kidney, endothelium is damaged and expresses adhesion molecules, cytokines are released, toll-like receptors are triggered, intrarenal vasoconstrictor pathways are engaged, and apoptosis is induced. The coagulation system is also locally activated. In tubular cells, polarity loss or inversion and loss of adherence to the basement membrane are also associated alterations. Organ cross-talk, or renal impairment leading to organ injury elsewhere, appears to be possible through unknown mechanisms, highlighting the intricacy of the biological response to AKI. Unfortunately, this ischemic model does not have much clinical application to conditions like sepsis. The most frequent cause of acute kidney injury in hospital patients and patients in the intensive care unit is sepsis. Since 80% renal-artery blockage for two hours does not result in sustained renal failure, the model is thus not very useful for periods of diminished perfusion, as can occur after major surgery[29-36]. As a result, many of the guiding principles that clinicians employ to inform their comprehension of acute renal injury are questionably applicable to patients in contemporary hospitals or critical care units. Sepsis, major surgery (particularly open-heart surgery) and severe decompensated heart failure are the most frequent causes of acute renal injury in these patients. In none of these cases is the renal artery blocked. More pertinent models are required. Considering the ambiguities surrounding animal models of acute kidney injury, it becomes sense to continue pathogenetic research in humans. Such investigations are challenging, nevertheless, because kidney biopsy samples taken to look into acute tubular necrosis are unnecessary in the absence of viable therapeutic therapies. Thus, histological evaluation is only employed for quick post-mortem evaluation, adding significant confounders such selection bias, premortem hypoxia, and premortem ischaemia. Despite the emergence of promising new methods, assessing renal perfusion—or blood flow—remains challenging and limited to invasive procedures. As these data reveal renal blood flow in individuals with known acute kidney injury, caution should be used when interpreting them because organ oedema, tubular damage and increased tubular luminal pressure could all be present and be the reason for the measured alterations[37]. Reported reductions in renal blood flow might be a symptom of acute kidney injury rather than its root cause. When injury is anticipated and the time of such injury is known, such as during cardiac surgery and renal transplantation, certain natural models of human acute kidney injury exist. Cardiac surgery does not allow for tissue evaluation and has not yet produced any insights into etiology. Renal transplantation has undergone extensive research

and enables tissue evaluation. It is a rare cause of acute kidney injury, nevertheless, and is exacerbated by the use of nephrotoxic medicines. Furthermore, we think it is challenging to extrapolate findings from a non-perfused, cold-solution-preserved organ outside the body to typical clinical drivers of acute kidney injury such sepsis, haemorrhage or major surgery[23,38]. The key potential pathways implicated in pathogenesis of AKI due to ischaemia or sepsis mention in figure 1.

6. Risk Factor of AKI

A. Non-modifiable: Black race, old age, and male sex, Chronic renal disease that was present before Proteinuria and/or a high albumin-to-creatinine ratio, Diabetes mellitus, Hypertension, Chronic liver conditions and/or side effects of portal hypertension, Cardiomyopathy and/or a reduced ejection fraction, coronary artery disease or a recent myocardial infarction, Long-term obstructive pulmonary disease, vascular disease of the periphery, a malignancy.

B. Potentially modifiable: A condition anaemia, Critical ailment, Sepsis, Major noncardiac surgery, trauma, and cardiac surgery, Media exposure that uses radiocontrast, Fluid overflow, Fluid resuscitation utilizing artificial colloids such as hydroxyethyl starch or chloride-rich solutions like 0.9% saline Drug toxicity, drug interactions, or nephrotoxic drugs, High-risk or life-threatening procedures[39].

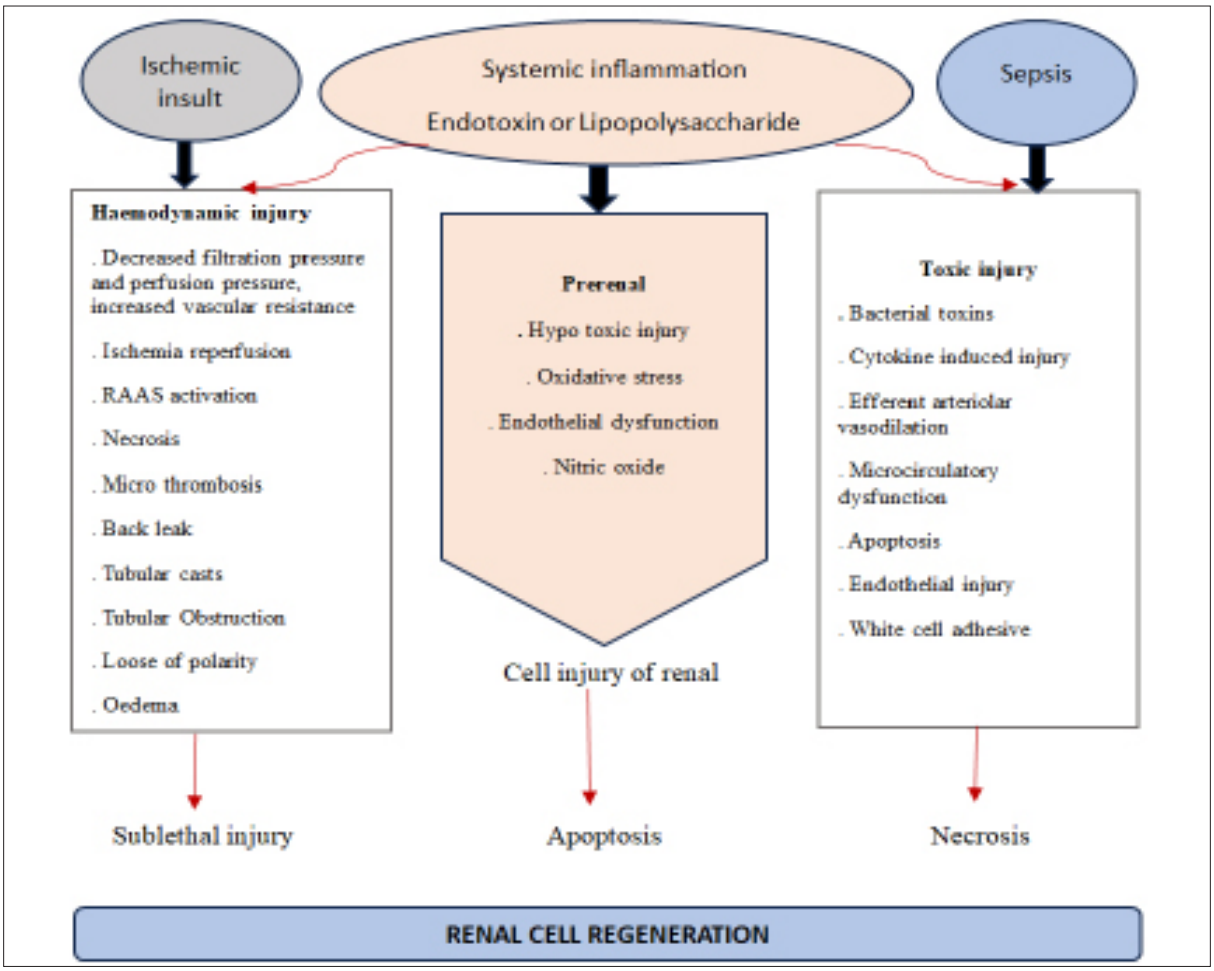

Figure 1: Key potential pathways implicated in pathogenesis of AKI due to ischaemia or sepsis[23]

7. Diagnosis

Since acute kidney injury does not have any distinctive clinical findings and is asymptomatic up to extreme levels of function loss, diagnosis is usually made in conjunction with another acute illness. Oliguria is a helpful indicator, although it is neither sensitive nor specific[40]. The majority of the time, laboratory tests are used to determine the presence of acute kidney injury in high-risk situations (such as sepsis, major surgery, bleeding, or volume losses). The typical diagnostic analytes are amounts of urea and creatinine. When a patient has elevated blood creatinine levels, it's crucial to determine whether they have acute kidney damage, chronic kidney disease, or a short-term acute sickness on top of a long-term chronic condition. Usually, the clinical setting offers hints. The existence of chronic kidney disease is suggested by abnormal serum creatinine levels prior to presentation, pertinent risk factors (such as hypertension or diabetes), a slow course of the presenting illness, high serum concentrations of creatine or phosphate, or both, and normocytic anaemia. Small kidneys may be visible on a renal ultrasonography, which could indicate a persistent illness. The existence of obstruction is rare when acute kidney injury has a sudden and obvious etiology, such as pneumonia with septic shock, heart surgery, trauma with haemorrhagic shock, or diarrhoea. It can be easy to assume that the trigger is the presence of noticeably elevated intra-abdominal pressure because of the clinical situation and elevated bladder pressure. It is crucial to consider the possibility of blockage as a cause of acute kidney damage or acute-on-chronic renal disease in cases where the presentation is less clear. In any disease, renal ultrasonography may be beneficial. Even though acute tubular necrosis is typically thought to be the primary cause of the majority of cases of intrinsic acute kidney injury linked to prerenal triggers, the syndrome can also appear in some persons after an inflammatory parenchymal disease. Glomerulonephritis, interstitial nephritis, and vasculitis are the most prevalent of these disorders. One of these diagnoses may be indicated by certain clinical features, such as the occurrence of macroscopic haematuria in glomerulonephritis, systemic symptoms in vasculitis, or the recent initiation of a medicine known to cause interstitial nephritis. Malignant hypertension, pyelonephritis, bilateral cortical necrosis, amyloidosis, malignant illness, and nephrotoxins are additional prevalent causes of parenchymal acute kidney injury. Patients frequently exhibit acute kidney injury without blockage or a distinct prerenal etiology. Urinary microscopy in these individuals frequently reveals glomerular pathological alterations, including haematuria, proteinuria, fragmented red cells, red-cell casts, white-cell casts, or granular casts, individually or in combination. Testing for eosinophils in urine samples should be done when interstitial nephritis is suspected. The test's sensitivity, though, is low. Particularly in sepsis, urine biochemical analysis is of little value[41-43]. In systematic reviews of research in animals or people, measurement of variables like the fractional excretion of sodium or urea has not consistently been demonstrated to have a clear link with histopathological findings. Little is known about the relationship between biochemical tests and damage biomarkers, clinical course, or prognosis in critically ill patients[44]. Albuminuria is a potential disease biomarker and a substantial risk factor for the development of acute renal injury. It is unknown how urine microscopy, a putative proxy indicator of tubular injury, and histology relate to one another. However, the urine microscopy score (As per the measurement of tubular cells and casts) corresponds with hospital mortality, biomarkers of injury, and increasing acute kidney injury. Any urine abnormalities may have therapeutic implications, although they are unknown. Blood testing can identify signs of an unexplained immune state, and specific autoantibody tests can reveal patterns indicative of particular forms of vasculitis. If clinically necessary, a kidney biopsy could reveal diagnostic alterations[23,45-47].

8. Management of AKI

8.1 General management

The concepts of managing acute kidney injury that has already occurred include treating or eliminating the cause and maintaining homoeostasis while healing takes place. By performing actions ranging in difficulty from fluid restriction to extracorporeal renal replacement therapy, problems can sometimes be avoided. The majority of specialists suggest beginning nutritional support as soon as feasible, recommending that it be delivered as for other hospital inpatients or those in intensive care units and contain enough calories and protein. There is no evidence that some renal dietary therapies are effective or required. It is important to provide the required daily intake of vitamins and trace minerals. Parenteral feeding is preferable over the enteral method[48]. If an acidosis is present, a bicarbonate infusion, nebulized salbutamol, or a combination of all three should be administered to patients with hyperkalaemia (potassium concentrations >6 mmol/L). 10 mL of 10% calcium gluconate solution should also be administered intravenously if the serum potassium level is greater than 7 mmol/L or if electrocardiographic hyperkalaemia symptoms are present. While renal replacement therapy is set up, these treatments are intended to provide temporary relief. Although chemical imbalance is virtually always present, unless it is severe, it rarely needs therapy. Correcting anaemia might be necessary. To account for the decreased clearance brought on by the loss of renal function, drug therapy needs to be changed. Prophylaxis for stress ulcers is advised. The prevention of infection should receive careful consideration. Using loop diuretics in people with polyuria can occasionally avoid fluid excess. There are no specific recommendations for managing fluids, and fluid restriction may be required in some circumstances. However, we believe that the best technique to prevent fluid overload in critically ill patients who have had their fluid levels resuscitated and have clearly defined oliguria or anuria is to immediately begin renal replacement treatment. We recommend utilizing this strategy because there is already some fluid overload and nutritional demands often call for at least 1 L of fluid per day in addition to pharmacological needs of 500 mL per day. These fluid sources cannot be compensated for by insensitive losses. In patients with acute renal impairment, fluid excess is now commonly accepted to increase the risk of death. 10–20% of the permitted limit can be used to cause adverse clinical consequences. It is thought that significant azotaemia, indicated by urea concentrations >30 mmol/L or creatinine concentrations >300 mol/L, is a sign of an unfavourable toxic condition. However, there are no recommendations that specify the level of acute azotaemia that is tolerable. Unless recovery is imminent or has already begun, or unless a return to normal urea and creatinine concentrations is anticipated within the next 24 to 48 hours, we believe that renal replacement therapy should be utilized to treat this level of azotaemia. Randomized controlled trials have not yet been able to define the ideal time for intervention with artificial renal assistance[23].

8.2 Fluid management

Renal function is compromised by both hypovolemia (low renal perfusion) and hypervolemia (high kidney perfusion). As renal and cardiac dysfunction exacerbate one another to form cardiorenal syndromes, impaired cardiac function contributes to both issues. Compared to healthy organs, an injured kidney or heart is more likely to experience clinical signs of hypovolaemia or hypervolaemia. Therapeutic measures must be escalated as symptoms get worse. Hypotension often indicates kidney hypoperfusion despite clinically apparent hypervolaemia when fluid redistributes to the venous system, tissue interstitial, or third compartments, such as in hepatorenal syndrome, congestive heart failure, or capillary leakage during sepsis[49]. In patients with renal and/or heart failure, both organs' potential to continue working under hypovolemia or hypervolemia is greatly diminished. In an otherwise euvolemic patient with AKI, a single bolus of buffered crystalloid fluid can detect prerenal AKI

and subclinical hypovolemia. Because doing so could worsen oedema or congestion and lower tissue oxygenation, balanced crystalloids shouldn't be administered for a lengthy period of time. Loop natriuretic should be given to individuals with hypervolemia who have AKI instead of fluids. In critically ill patients with inhibited vasomotor response, vasopressors are frequently needed to boost cardiac output[15,50]. The fluid management of AKI diagrammatically represent in figure 2.

8.3 Nephrotoxic drugs and agents

All potentially nephrotoxic medications that might stopped should be, as the risk for AKI rises with using drugs that are nephrotoxic. Only take essential drugs as directed for the specified length of time[51,52]. If at all possible, close observation of drug concentrations is also required (for vancomycin, for instance). Utilization of arterial radiocontrast agents should be restricted to circumstances wherein the therapeutic benefit justifies the danger, and they should be administered at the lowest volume possible. For instance, unless absolutely essential, the ventriculography portion of cardiac catheterization should not be performed[53,54]. Finally, fluids with non-physiologic sodium and chloride ratios may exacerbate AKI. Most patients prefer balanced electrolyte solutions, such as lactated Ringer's solution[15].

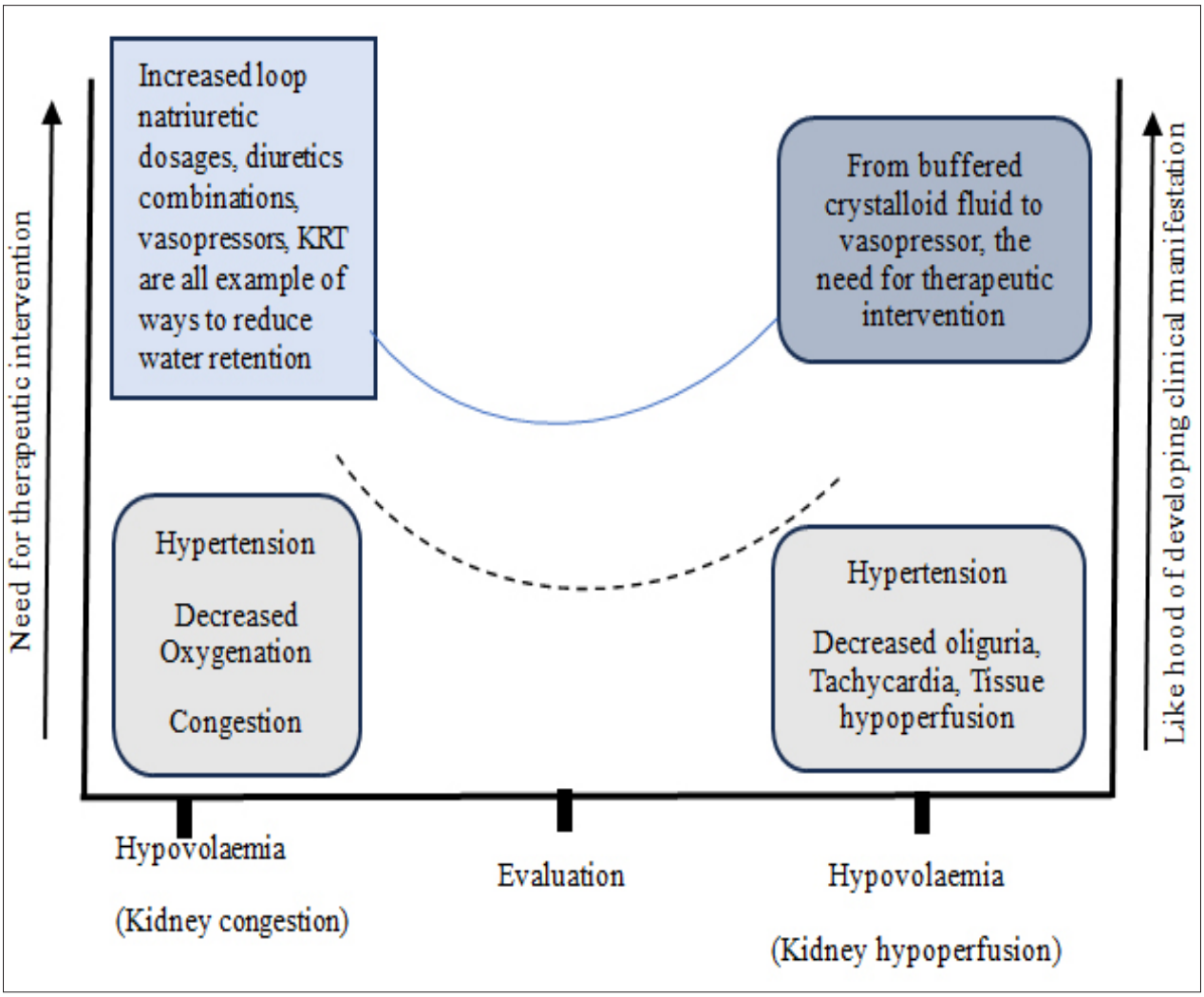

Figure 2: Fluid management in AKI15

8.4 Haemodynamic management

According to the type of circulatory shock the patient suffers, the management of blood pressure and cardiac function in view of the situation that lead to AKI (for instance, septic shock or cardiac surgery) is complicated and includes context-specific considerations. To control hemodynamic in patients with AKI, however, there are certain broad principles that can be applied. Organs, including the kidneys, are adequately perfused under typical conditions at a mean arterial pressure (MAP) of 65mmHg[50,55-59]. Studies examining whether ICU patients should have higher MAP objectives have found mixed results[57]. Patients with severe (and occasionally poorly controlled) hypertension may benefit from a higher MAP when in shock, even if there is no set target that can be advised. According to certain studies, the patient's normal blood pressure can be changed to identify the MAP objectives for customized blood pressure management[60]. Similar to this, people with elevated venous pressure (due, for instance, to right-sided heart failure) might not be able to maintain a sufficient perfusion pressure for the kidney at a MAP of 65mmHg. In addition, because it can impact both arterial flow and venous pressure, intra-abdominal hypertension is particularly hazardous for kidney perfusion[61]. The outcome is, clinicians must personalize patient care, occasionally attempting a higher MAP in certain circumstances. It is essential to maintain a healthy volume status while also using vasopressors to treat vasomotor paralysis. Studies utilizing functional hemodynamic monitoring to direct hemodynamic management in cardiac surgery and sepsis have showed promise[62]. The first-line vasopressor for vasodilatory shock is noradrenaline[63]. None of the drugs are "kidney-friendly" more than the others; alternative medications are frequently reserved for cases of refractory shock or for certain illnesses. In patients with severe septic shock, corticosteroids are frequently recommended as a supplementary therapy, and angiotensin II may be helpful in some circumstances of angiotensin II insufficiency[15,64].

8.5 RRT and extracorporeal support

AKI can become so severe in some patients that Renal Replacement Therapy (RRT) is necessary[65-67]. Such an activity cannot be justified by any one set of norms. Physicians must consider all pertinent factors, including the general history of the patient's illness, the existence of comorbid conditions, potassium levels, fluid status, acid-base status, creatinine and urea levels, and urine output[68].

It's still debatable when RRT should begin. From 2016 to 2017, three randomised trials attempted to address this issue. 620 ICU patients with KDIGO AKI stage 3 were divided into two groups: those who received immediate RRT or those who received delayed RRT (in which RRT was initiated in reaction to severe hyperkalaemia, severe metabolic acidosis, pulmonary oedema, 72 hours of oliguria, or a blood urea concentration of greater than 37 mmol/L). Although the usage of RRT, the frequency of catheter-related bloodstream infections, and the amount of time it took for diuresis to occur were all reduced by such a delayed interventional strategy, mortality was unaffected[69].

This experiment received criticism for comparing late with extremely late RRT and for using intermittent haemodialysis (instead of continuous RRT) in a significant part of patients, despite the fact that the majority of patients were receiving vasopressor medication. In the second study, 231 patients with stage 2 KDIGO AKI were given the choice of undergoing RRT immediately after stage 3 or not at all. Early RRT was found to increase the likelihood of renal recovery and reduce mortality from 547% to 393% (all by continuous RRT)[70,71]. This study has received criticism for its lack of multicentricity, inclusion of a cohort of patients who underwent just surgery, and predominance of post-cardiac surgery AKI. In the third experiment, 488 patients in French ICUs with septic shock and stage 3 KDIGO AKI were given the option of receiving RRT within 12 hours or a 48-hour delay if renal recovery had not yet taken place[72].

According to the study, there were no notable

variations in mortality or other patient-centered outcomes. Although intermittent haemodialysis is a rare practice in the majority of ICUs in Australia, New Zealand, North America, and the UK, a third of the patients had treatment while they were in shock. This practice was criticized. In addition, 17% of patients required emergency RRT, and 9% of patients died during the 48-hour delay. A much larger trial including 3000 patients was started a large portion of patients this ongoing dispute, and it is now almost finished[73].

As soon as the choice is made to begin acute RRT, three types of RRT are available: peritoneal dialysis, intermittent RRT, which can be either intermittent haemodialysis or slow protracted dialysis, and continuous RRT[74,75]. Peritoneal dialysis is routinely utilized in many resource-limited countries but is infrequently employed in high-resource countries because to its clearance limitations, difficulties with fluid removal, and complications. In these countries, there has been an increase in interest in using it as a logistically efficient dialysis treatment. Numerous studies in this field have indicated acceptable performance and outcomes[76,77].

Whether to utilize intermittent or continuous RRT is a hotly debated topic. To address this issue, no well powered randomised controlled trials have been conducted. However, the small to medium studies conducted do not imply a difference in patient survival. Therefore, based on the patient's survival, continuous, slow prolonged dialysis, or intermittent RRT all seem to be viable choices[78]. However, a body of empirical research, including meta-analyses imply that, in comparison to continuous RRT, the utilization of intermittent haemodialysis could lead to renal recovery takes longer[79].

Following a landmark study on RRT intensity, the ATN project and the RENAL study, two sizable multicentre randomised controlled studies[80], have established the benchmark for solute removal intensity at an administered dose of continuous RRT equivalent to 20–25 mL/kg per hour of effluent formation[81]. Importantly, continuous RRT was administered to every patient with AKI receiving vasopressor support in both investigations, suggesting that continuous RRT is the norm for patients with haemodynamic instability[82].

Despite the fact that these two critical trials established the benchmark for solute clearance, volume control has not yet been the subject of a sizable multicentre randomised controlled study. Therefore, despite concerns regarding the effects of a positive fluid balance on renal and patient outcomes, individual clinical judgment continues to be the major factor in volume control[83,84]. It is unclear when RRT should be halted once it has been initiated. This problem has not been examined in any randomised controlled studies. A spontaneous urine production of more than 500 mL per day, according to observational studies, appears to have enough discriminating to be utilized for the purpose of considering a trial of continuous RRT cessation[85].

The following actions that should be taken into consideration and carried out from the initial patient observation to the onset of AKI and the recommendation of RRT are schematically summarized in the appendix. The appropriate prescription in terms of modality and operational parameters should be set once the aims for RRT have been identified, ensuring ongoing monitoring and data-driven feedback on therapeutic change. Technology can assist in this process by helping to prescribe, deliver, and monitor the treatment as well as by modifying the various steps based on dynamic factors that are unique to the patient[8,86-91].

8.6 Post care AKI

In comparison to other individuals who did not acquire AKI, people with AKI typically experience worse medium- to long-term results[92,93]. By closely monitoring patients who experience one or more bouts of AKI while receiving hospital or ICU treatment, this observation raises the possibility of improving patient care[94]. These patients seem particularly vulnerable and might need particular medical procedures[95]. It goes without saying

that providing longer-term care will cost more and might be more difficult in some regions. In order to target the individuals most likely to benefit from this therapy, risk prediction models that can recognize patients at high risk for CKD after AKI may be highly useful[8,96].

9. Conclusion

The description and categorization of AKI, our knowledge of the pathophysiological mechanisms, and our interactions with other fields and organ systems are all significantly evolving. Epidemiology describes a rising occurrence that is partially brought about by more thorough clinical assessment and identification. A notable progress in the subject is represented by new biomarkers and sophisticated diagnostic methods, which enable prompt and successful preventative and protective actions to be taken. The RIFLE classification, which has received widespread validation, offers a reliable evaluation of the risk of AKI and consequences. Enhanced extracorporeal organ support technology, Haemodynamic management, renal replacement therapy, Nephrotoxic drugs, Fluid management, more individualized pharmacological therapy, and standardized and protocolized management of physiological endpoints have all contributed to improvements in the management of patients with AKI that have occurred simultaneously with improvements in hospital and intensive care quality.

10. Conflict of Interest: None

11. References

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