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Acute Kidney Injury: The Modern Therapeutic Approach

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Abstract

Organ transplant has increased in the last few years, and preoperative evaluation is a key factor for a favorable outcome. The kidney is one of the organs that are currently most transplanted. Recognizing causes of kidney injury is important for health care costs and morbidity of the patient. It is important to determine if the patient is a candidate for a kidney transplant or a combined liver-kidney transplant, depending on the underlying cause of the disease. Work up studies, and risk factors have to be taken into account; like hypertension, ascites, use of nephrotoxic drugs, and diuretics. This preoperative evaluation reviews the key factors that prevent the most important causes in renal injury and highlights the importance of single or combined transplant surgery.

Keywords: Acute kidney injury; Liver transplant candidate evaluation; Prevention

Introduction

Acute Kidney Injury (AKI) is diagnosed by clinical data, clinical history or low renal output. It can be defined as an acute progressive loss of renal function with a rapid onset over a period of hours to days, sometimes accompanied by oliguria (urine volume excretion less than 400 mL/day in adults or less than 20 mL/hour). Many causes may lead to AKI, including low blood volume, certain drugs and more. It can be categorized in prerenal, intrinsic and postrenal. Prerenal is usually due to low blood volume which leads to renal ischemia. Intrinsic is usually due to glomerulonephritis, acute tubular necrosis and interstitial nephritis. Postrenal is secondary to an obstructive cause of the urinary tract. Management is on identification of the underlying cause and renal replacement therapy can be offered to depend on the clinical evolution of the patient. The Acute Dialysis Quality Initiative group developed the Risk, Injury, Failure, Loss and End –stage kidney disease (RIFLE) system for classification and diagnosis of acute kidney injury. Loss and End stage injury refers to the time of evolution since the patient has had acute kidney injury; the Loss criteria greater than 4 weeks and End Stage Renal Disease greater than three months.

Pathophysiology

It involves a complex set of factors leading to decrease kidney function. Vascular constriction and congestion, apoptosis, leukocytes and immunomodulators play an important role in the development of AKI. Therapy involves a multidisciplinary approach and many modalities cannot achieve a therapeutic improvement [1].

Kidney function loss is secondary to ischemia or toxins, because of the high cellular demand for oxygen and ATP, which leads to tubular epithelial cell death. Apoptosis can be either intrinsic or extrinsic; the latter involves the binding of ligands to receptors via caspases. Intrinsic apoptosis involves mitochondrial outer membrane permeability and the secondary release of cytochrome c via B-cell lymphoma 2 family and tumor suppressor protein p53 [1,2].

Renal hypoperfusion can be caused by a number of factors, which include a low cardiac output, low blood volume, dehydration, etc. Hypoperfusion carries low oxygen to the kidney, which in return responds with a series of autoregulation modalities via the afferent and efferent arterioles. These arterioles are mainly controlled by prostaglandins, catecholamines, nitric oxide, renin-angiotensin-aldosterone and adenosine, among other factors, are essential for the maintenance of glomerular filtration rate [2].

Decrease in renal blood flow leads to the reduction of glomerular filtration rate, whatever the cause of AKI. Ischemia is usually the cause of intrinsic injury; postrenal injury is secondary to an increased pressure in the renal tubules. Tubule cellular death, which is initiated as the epithelium flattening, loss of the brush border and loss of tight junction all lead to the reduction in GFR and loss of the autoregulatory response. In ATN, the isosthenuria is due to the failure to maintain a medullary solute gradient. Eventually, the remaining nephrons undergo hypertrophy to maintain hyperfiltration, which in time leads to glomerular sclerosis until complete renal failure ensues [3].

Preoperative Evaluation of Kidney Function

Until recently, the Acute Kidney Injury Network includes a modification of a difference greater or equal to 0.3 mg/dl for 48 hours with respect to the initial creatinine value; or an increment of 1.5 times the baseline value over the last 7 days; or a urine volume less than 0.5 ml/kg/hr for 6 hours. These criteria help to identify an acute kidney injury before it develops, and an underlying cause to be investigated. On the basis of these criteria, the next 48 hours since the diagnosis of acute kidney injury, a new creatinine value should be obtained. Defined in terms of time, an AKI has an elevated creatinine value for less than three months; while chronic kidney disease has a GFR less than 60 ml/min for 3 months or more (Table 1). Patients with a gradual increase in creatinine level should have a detailed clinical history emphasizing

Stage	Glomerular filtration rate(eGFR
1	≥ 90
2	60 to 89
3a	45 to 59
3b	30 to 44
4	15 to 29
5	<15 or on dialysis

eGFR (ml/min/1.73m^2) is an approximate of renal function, based on serum Creatinine but taking into account age, race, and sex.

Table 1: Chronic Kidney Disease Stages.

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an underlying cause. It is important to evaluate the use of nephrotoxic drugs; NSAIDs, COX-2 inhibitors, aminoglycosides, vancomycin, ACE inhibitors, angiotensin receptor blockers, diuretics, recreational drugs, herbal remedies and if low fluid intake has taken place. Physical exam should be directed to sign of cardiac heart failure, peripheral edema, jugular distension and dehydration. At the present moment, many hospitals perform combined organ transplantation. It is important to calculate the renal function during the preoperative evaluation with the use of the MDRD or Crockoft Gault formula, whether acute or chronic renal injury is present. The use of these criteria provides a better evaluation for renal function than just the creatinine. Recognizing the causes of renal failure is important for lowering health care costs and the morbidity of the patient. Patients with renal failure have a higher rate of hypervolemia, electrolyte disturbance, acid-base imbalance, infections and poor healing process [4-6]. About 30% of patients that recover from acute kidney disease have an increased risk for developing chronic kidney disease or heart disease [7-9].

Patients with stage 4-5 should be considered for transplant kidney surgery (Table 2). The majority of patients with normal renal function receive solid organ transplantation alone. Close attention should be placed in hepatic cirrhosis, chronic heart failure and malnutrition because the creatinine can have a lower value secondary to the loss of muscle mass. Initial evaluation in a transplant patient should have a detailed clinical history, family history, a careful physical exam, urine analysis and renal ultrasound. The average kidney should measure between 10 to 12 cm in diameter. Loss of Kidney cortex is considered a feature of chronic kidney disease [10]

Elevated creatinine levels should prompt for previous creatinine work up history for the last 3 to 6 months, for comparative initial renal function and the lapsed time of renal deterioration. According to the obtained results, we must consider if the patient has an acute renal disease that is resolving, of primary setting, getting worse or has a pre-existing CKD [10-12].

Cockroft - Gault formula

a) Male

$$Cl\ Cr = ((140 - \text{age in years}) \times \text{weight (kg)} / \text{Blood Cr} \times 72$$

b) Female

$$Cl\ Cr = (\text{male Cl Cr}) \times 0.85\text{: female}$$

Causes and Clinical Presentation

Prerenal AKI is usually due to low blood volume in 40-70% of the cases. Damage to renal blood vessels, glomeruli, tubules and interstitium, cause intrinsic AKI in 10-50% of the cases. An exception is made when blood vessels are involved since it produces a combined prerenal and intrinsic AKI.

Main prerenal causes	EXAMPLES
Hypovolemia	Severe bleeding Diarrhea, vomit, burns, diuretics
Hypotension	Septic,cardiogenic or anaphylactic shock
Low renal blood supply	ACE inhibitors/angiotensin II receptor blockers, NSAIDS, COX2 selective inhibitors, hepatorenal syndrome, renal artery occlusion
Severe oedema	Cardiac insufficiency, liver cirrhosis, nephrotic syndrome

Table 2: Transplant kidney surgery.

Prerenal AKI

Secondary to any cause that produces a low blood flow within the kidney. Autoregulation depends mainly on the combination of preglomerular vessel dilation (due to prostaglandins and nitric oxide) and postglomerular vessel vasoconstriction (angiotensin II). About 20% of the causes of AKI are due to prerenal etiology.

Intrinsic AKI

Approximately 45 % of cases of AKI in the hospital are secondary to ATN. In Table 3, we describe the principal etiologies.

Postrenal AKI

It occurs in about 10% of cases. It has a high sensitivity to be detected in renal ultrasonography. Is mainly due to obstructive causes. The main causes in Table 4.

Acute Tubular Necrosis	
Ischemic Origin	Prerenal Cardiovascular Surgery
Toxics	Antimicrobials Iodine Contrasts Chemotherapeutics (cisplatin) Opioids Poisons Heavy Metals (mercury, copper, lead)
Intratubular Deposit	
	Acute Uric Nephropathy
	Severe Hypercalcemia Multiple Myeloma Sulfamides Acyclovir
Organic Pigments	Mioglobin-rhabdomyolysis Hemoglobinuria
Glomerulonephritis	**Acute poststreptococcal** **Systemic Diseases (vasculitis, Systemic erythematosus lupus, etc)** **Membranoprolipherative**
Intersticial Nephritis	Antimicrobials NSAIDs Diuretics
Cortical Necrosis	Abruptio placentae Disseminated intravascular coagulapathy
Vascular Disease	Thrombosis or bilateral renal artery embolism Uremic hemolytic síndrome/PTT Antiphospholipid sydrome Malignant hypertension

Table 3: Principal etiologies.

Postrenal AKI	
Acquired uropathy	Benign prostatic hypertrophy Lithiasis
Congenital anomalies	Urethral valves Vesical diverticulum Neurogenic bladder Spina bifida
Retroperitoneal fibrosis	Radiation Methisergide
Infectious	Tuberculosis Aspergillosis Schistosomiasis Actinomycosis Candidiasis
Intratubular Obstruction	Acyclovir Sulfamides

Table 4: High sensitivity to be detected in renal ultrasonography.

Diagnostic Approach

If a patient is considered for transplant surgery has an acute renal injury, most of the time the transplanting organ can initially recover renal function (i.e. hepatorenal syndrome) because hypoperfusion was the cause of renal dysfunction. There is also an emerging evidence that cystatin "c" is a more sensitive indicator of kidney function than the serum creatinine concentration in patients with liver or heart disease [13-15].

As mentioned previously, past workup studies indicate acute or chronic renal function loss. Initial symptoms include anorexia, nausea, vomits, muscle cramps, polyuria, nocturia, etc. These are secondary to uremic syndrome, however, prolong symptom history is more indicative of CKD. Anemia, metabolic calcium-phosphorus imbalance, metabolic acidosis and hyperkalemia are suggestive of CKD with similar past workup studies. Renal ultrasound: It can be used to see the anatomy, size, cortical/medullary relationship and obstructive site can be detected. Small size kidney detection on ultrasound, mainly renal cortex thinning and loss of the cortex/medullary relationship, are suggestive of CKD. Normal size kidneys are usually seen in AKI, except in diabetic nephropathy or amyloidosis (normal or increased size).

Once obstructive injury is excluded, it is important to determine if AKI is prerenal due to hypovolemia (vomits, diarrhea, hemorrhage, diuretics), body volume redistribution (ascities, intestinal edema), systemic circulation (cardiac insufficiency, cirrhosis) or nephrotic síndrome with edema.

Urinalysis: It's important to do a urinary sediment since there can be hematuria and proteinuria that leads to the cause and outcome of the renal disease. Proteinuria can be appreciated with the urinary protein/creatinine ratio or the measurement of proteins in a 24 hour urine collection (mostly seen in malnutrition or patients with an increased lean muscle). Urinary sodium levels are important for guiding the differential diagnosis. Previous use of diuretics or CKD can have urinary sodium levels greater than 20 mEq/lt and fractional sodium excretion greater than 1. The reason that it is important to use the fractional excretion of urea (Table 5).

Percutaneous renal biopsy should be considered in renal transplant candidates that have an undetermined cause of renal failure, hematuria or proteinuria. The most frequent complication in renal biopsy is hematuria in 1% of the cases (in patients with normal bleeding times and without thrombocytopenia). Transjugular renal biopsy should be performed in patients considered for a liver transplant and have abnormal bleeding times due to a high rate of complications (50%). This type of biopsy should also be considered for patients on mechanical ventilation [16-18]. Renal biopsy can show intrinsic renal pathology; ATN due to hypoperfusion; glomerulosclerosis, atrophy, fibrosis [19,20] which can aid in evaluating for simple or combined transplant surgery. If hematuria and cirrhosis are presented, one should suspect a glomerulonephritis secondary to hepatitis B or C. If we have a patient with ethylic cirrhosis and hematuria; it can be secondary to IgA nephropathy.

	AKI prerenal	ATN
Osm_u (mOsm/kg)	>400	<350
Na_u (mmol/l)	<20	>40
RFI	<1	>2
FeNa	<1	>2
U_u/U_{pl}	>10	210
Cr_u/Cr_{pl}	>20	<15

Osm_u: Urinary osmolarity. RFI: Renal Failure Index. FeNa: fractional excretion of sodium. U_u: urinary urea concentration and in plasma U_{pl}. Cr_U: Urinary creatinine and plasma creatinine Cr_{pl}.

Table 5: Urinary index in prerenal AKI and ATN.

Treatment and Prevention

Treatment depends on vital urgency, underlying pathology and the need for hemodialysis. Major complications are volume overload hyperkalemia, hypermagnesemia, hyperuricemia, metabolic acidosis, calcium/phosphorus imbalance (hypocalcemia, hyperphosphatemia).

Common AKI complications include

- Hyperkalemia (serum potassium >5.5 meq/L) or a rapidly increasing serum potassium
- Uremic Signs, such as pericarditis, or an otherwise unexplained decline in mental status
- Severe metabolic acidosis (pH less than 7.1)
- Fluid overload or pulmonary edema

Patients that require hemodialysis have a medical treatment initially, since it cannot be inmediately performed.

Other Factors to take into Account

Arterial hypotension

Antidiuretic hormone is stimulated due to hypotension. As a result, the distal tubules and the collecting ducts become permeable to water [10]. If hypotension persists, it can produce severe oliguria or even anuria. Not identifying this problem in time can lead to ischemia and posterior necrosis. This in turn produces a lesion in the proximal tubules of the outer stripe of the medulla, and the proximal convoluted tubules which can end into an acute tubular necrosis [21]. In these patients, if hypotension is not due to hemorrhagic shock, multiple studies suggest initial management with isotonic crystalloids for intravascular expansion. A recent Cochrane study demonstrated that colloids are not superior to crystalloids and does not reduce de risk of death in burned, trauma or surgical patients [22]. Acute kidney injury has been described, which could be to the increase in the oncotic pressure [23].1500 to 3000 ml is administered IV energetically depending on the age and comorbidities. If hypotension persists after intravascular volume is replenished, treatment should be with vasopressors [24]

The majority of studies recommend the use of norepinephrine, dopamine or vasopressin. Dopamine has the inconvenient of causing a higher probability of arrhythmia in comparison with norepinephrine. A subgroup analysis showed that dopamine was associated with an increased rate of death at 28 days among patients with cardiogenic shock, but not in patients with septic or hypovolemic shock [25]. Vasopressin is principally used when shock is refractory to norepinephrine [26].

Patients with arterial hypotension have a worse perfusion and an increase in acid lactic level [27].A stability protocol should be taken into account during the first 6 hour. It is important to maintain a mean arterial blood pressure ≥ 65 mmHg, a central venous pressure between 8 and 12 mmHg, lactate level balance, central venous oxygen saturation >70% and urine output of ≥ 0.5 ml/kg/h [27].

Diuretic use in the prevention of acute kidney injury?

Prophylactic use of furosemide has been demonstrated of not being nephroprotective in cardiac surgery candidate [28,29]. It's also ineffective and it can even worsen the renal function in the treatment

of acute renal injury [30,31]. Doses higher than 1 gm/day has been associated to a higher risk of toxicity; contrary to when administered in a continuous infusion rate of 0.5 mg/kg/hr [32]. The use of loop diuretics is not recommended for the prevention for AKI, exception is management of hypervolemia [10].

Is the use of dopamine a myth or reality in the prevention and treatment of AKI?

Multiple studies have demonstrated that the use of dopamine in patients with AKI does not produce the same vasodilation effect as in normal subjects. They have also shown that there is no beneficial use for prevention and treatment of AKI [33,34]. Lauschke et al. [34] demonstrated through Doppler ultrasound that dopamine increases the renal vascular resistance in patients with acute kidney injury. There is an increased risk of adverse effects such as tachyarrhythmias, myocardial and intestinal ischemia and T-cell function suppression [35]. Recent KDIGO guides do not recommend using low dose dopamine for the prevention or treatment of kidney injury.

Are there other medications for the prevention of AKI?

Fenoldopam mesylate is a dopamine type-1 receptor agonist with similar effects as dopamine in a low dose regimen [36]. There are some studies that favor and others that are against the protective renal effects. Some preliminary studies have shown an antiinflammatory effect [37,38] nonetheless, fenoldopam has a potent antihypertensive effect. This is the only approved indication, which can lead to a considerable high risk of hypotension, [39,40] suggesting not to use it [10].

Prevention of contrast-induced nephropathy?

Nephropathy is believed to be caused by vasoconstriction and direct epithelial toxicity through free radicals. It is produced in the first 24 to 48 hours. Maximum creatinine level is reached in the 4^{th} to 5^{th} day and back to normal value on the 10^{th} day. The main risk factors are diabetes, creatinine level ≥ 1.5 mg/dl or GFR<60 ml/min/1.73m^3, contrast solutions with high osmolarity, multiple myeloma, congestive heart failure, NSAID use, and hypotension.

To prevent nephropathy isotonic sodium bicarbonate is recommended over the use of isotonic saline solution if there is not any contraindication, and it can be used in hypocalcemia and metabolic alkalosis. The recommended dose is 3 ml/kg one hour before the contrast medium (iso-osmolar or hypo-osmolar), then to an infusion rate of 1 ml/kg/hour the next six hours of administering the contrast. 24 hours before and after the administration of the contrast medium, 600-1200 mg of oral N-acetylcisteine can be administered every 12 hours, because of its antioxidizing effect. Although the medication is still debateable, it is still recommended since it produces very few side effects.

Main risk factors for developing AKI in surgery?

In a 2005-6 prospective study performed in the United States, 152244 surgeries were performed, found that the main risk factors for developing AKI in general surgery was: Age greater or equal to 56 years old, presence of ascites, hypertension, male gender, diabetes mellitus, abdominal surgery, decompensated heart failure [37].

Liver Failure

MELD (Model for End-Stage Disease) including creatinine is a recently acquired scale for patients with kidney injury that has increased candidates with renal dysfunction [41]. Up to 6% of patients with a need for a liver-kidney transplant in 2007. It is important to distinguish the cause of renal dysfunction since a hepatorenal syndrome will resolve kidney failure with liver transplant alone. Patients with hepatorenal syndrome that need a continuous renal replacement therapy can develop acute tubular necrosis with a partial renal function recovery. If the estimated time of dialysis is between 6 and 8 weeks, the patient can be included in the combined liver and kidney list [42-44]. According to the definition by the NKF-KDOQI, a patient with an AKI more than 8 to 12 weeks in duration has a CKD and should be considered for combined liver and kidney transplant. Patients with CKD and GFR less than 30 ml/min and liver failure should also be considered for combined transplant.

In patients with moderate CKD, renal biopsy can aid in establishing the grade of chronicity depending on the tubular atrophy and glomerulosclerosis. Fibrosis greater than 30% can also be included in the combined transplant waiting list. In 2008, the consensus on simultaneous liver-kidney transplantation conference included the following criteria to receive liver and kidney transplantation:

- CKD and cirrhosis symptomatic portal hypertension or hepatic vein wedge pressure gradient>10 mmHg.
- Liver failure with CKD and GFR less than 30 ml/min
- Hepatorenal syndrome with a creatinine greater than 2 mg/dl or liver failure and AKI with dialysis greater or equal to 8 weeks

How to prevent hepatorenal syndrome?

It is defined as a potentially reversible cause. It can be seen in patients with hepatic cirrhosis, ascites, renal dysfunction and endogenous vasoactive activity increase. It is mainly produced because of splenic vasodilation, systemic circulation reduction and increase in the renin-angiotensin-aldosterone system which in turn reduces glomerular filtration via glomerular vasoconstriction.

types are described: Hepatorenal syndrome type 1: Fast progressive loss of renal function, defined as twice the initial creatinine value or creatinine >2.5 mg/dl in less than 2 weeks. It is usually accompanied of significant jaundice, coagulopathy and liver failure.

Hepatorenal syndrome type 2:

-Moderate renal failure with a creatinine between 1.5 mg/dl and 2.5 mg/dl. It is usually due to a gradual deterioration of weeks to months, in patients with ascites refractory to diuretic treatment. These patients have lower mortality rate than patients with type 1.

The main precipitating factors of the hepatorenal syndrome are hemodynamic alterations. The arterial hypotension is secondary to polyuria due to the use of diuretics, paracentesis ≥ 5. lts without volume replacement in patients with refractory ascites and in with gastrointestinal bleeding. Care must be taken in sepsis, especially of abdominal origin (spontaneous peritonitis).

Conclusions

Renal failure prevention is of high importance since it decreases the morbimortality rate of the patient. Careful awareness should be placed when nephrotoxic medications are selected. A detailed clinical history is important in order to establish risk factors and prevent possible complications. In the present time, many hospitals perform liver transplant surgery or combined liver-kidney transplant surgery. It is important to do the necessary studies to evaluate if the patient is a candidate for a single transplant or a combined transplant surgery.

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