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Acute and Chronic Renal Failure Treatment

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ABSTRACT
Acute kidney injury (AKI) is a common condition in multiple clinical settings. Patients with AKI are at an increased risk of death, over both the short and long term, and of accelerated renal impairment. As the condition has become more recognized and definitions more unified, there has been a rapid increase in studies examining AKI across many different clinical settings. This review focuses on the classification, diagnostic methods and clinical management that are available, or promising, for patients with AKI. Furthermore, preventive measures with fluids, acetylcysteine, remote ischemic preconditioning, as when dialysis should be initiated in AKI patients are discussed.
Keywords: Kidneys,chronic renal failure,kidneydisease,prevention herbal plants,environmental agents.

I. INTRODUCTION

Kidney disease, or renal disease, technically referred to as nephropathy, is damage to or disease of a kidney. Nephritis is an inflammatory kidney disease and has several types according to the location of the inflammation. Inflammation can be diagnosed by blood tests. Nephrosis is non-inflammatory kidney disease. Nephritis and nephrosis can give rise to nephritic syndrome and nephrotic syndrome respectively. Kidney disease usually causes a loss of kidney function to some degree and can result in kidney failure, the complete loss of kidney function. Kidney failure is known as the end-stage of kidney disease, where dialysis or a kidney transplant is the only treatment option

The concept of Acute Renal Failure (ARF)1 has undergone significant re-examination in recent years. Acute Kidney Injury (AKI) is the term that has recently replaced the term ARF.AKI is defined as an abrupt (within hours) decrease in kidney function, which encompasses both injury (structural damage) and impairment (loss of function).It is a syndrome that rarely has a sole and distinct pathophysiology. Furthermore the syndrome is quite common among patients without critical illness and it is essential that health care professionals, particularly those without specialisation in renal disorders, detect it easily.

Chronic kidney disease is defined as prolonged kidney abnormalities (functional and/or structural in nature) that last for more than three months.Acute kidney disease is now termed acute kidney injury and is marked by the sudden reduction in kidney function over seven days. About one in eight Americans (as of 2007) have chronic kidney disease,a rate that is increasing over time.Chronic kidney disease, also called chronicle kidney failure, involves a gradual loss kidney function. Your kidneys filter wastes and excess fluids from your blood, which are then removed in your urine. Advanced chronic kidney disease can cause dangerous levels of fluid, electrolytes and wastes to build up in your body. About one in eight Americans (as of 2007) have chronic kidney disease,a rate that is increasing over time.Chronic kidney disease, also called chronic kidney failure, involves a gradual loss kidney function. Your kidneys filter wastes and excess fluids from your blood, which are then removed in your urine. Advanced chronic kidney disease can cause dangerous levels of fluid, electrolytes and wastes to build up in your body.

ADVANTAGES
- Restoration of normal renal function
- Freedom from dialysis
- Return to normal life
- Reverses pathophysiological changes related to RF
- Less expensive than dialysis after 1st year

DISADVANTAGES
- Life long medications
- Multiple side effects from medication
- Increased risk infection
- Major surgery

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CAUSES

Results from acute damage to renal structure Possible causes .Acute glomerulonephritis pyelonephritis. May also result from acute tubular necrosis ANT. Damage of kidney structure from exposure to toxins solvents drug and heavy metals ATN is the mostCommon cause of acute renal failure Results from conditions block of urine outflow Possible causes obstruction of urine outflow by calculi tumors prostatic hypertrophy.

HISTORY:

The first description of ARF, then termed ischuriarenalis, was by William Heberden in 1802. At the beginning of the twentieth century, ARF, then named Acute Bright's disease, was described in William Osler's Textbook for Medicine (1909), to be "as a consequence of toxic agents, pregnancy, burns, trauma or operations on the kidneys". During the First World War the syndrome was named war nephritis, and was reported in several publications. The syndrome was forgotten until the Second World War, when Bywaters and Beall published their classical paper on crush syndrome. Acute tubular necrosis (ATN) was the term that was used to describe this clinical entity, because of histological evidence for patchy necrosis of renal tubules at autopsy. For many years in clinical practice, the terms ATN and ARF were used interchangeably. However, it is Homer W. Smith who is credited for the introduction of the term acute renal failure, in a chapter on Acute renal failure related to traumatic injuries in his 1951 textbook The kidney-structure and Function in Health and Disease.CKD diagnosed in the general population (community CKD) has a significantly different natural history and the course of progression compared to the CKD in patients referred to the nephrology practices (referred

CKD).Community CKD is seen mainly in the older population. These individuals have had a lifelong exposure to cardiovascular risk factors, hypertension, and diabetes which can also affect the kidneys. The average rate of decline in GFR in this population is around 0.75 to 1 ml/min/year after the age of 40 to 50 years.In a large study of community based only 1% and 20% of patients with CKD stages G3 and G4 required renal replacement therapy.

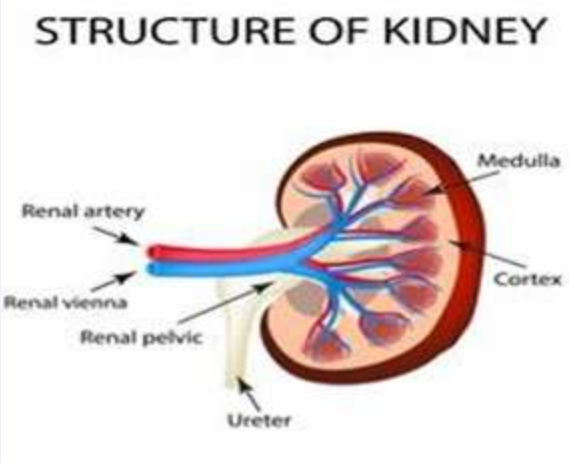

Figure : structure of kidney

ACUTE RENAL FAILURE

Classification of AKI includes pre-renal AKI, acute post-renal obstructive nephropathy and intrinsic acute kidney diseases. Of these, only 'intrinsic' AKI represents true kidney disease, while pre-renal and post-renal AKI are the consequence of extra-renal diseases leading to the decreased glomerular filtration rate (GFR). If these pre- and/or post-renal conditions persist, they will eventually evolve to renal cellular damage and hence intrinsic renal disease. The current diagnostic approach of AKI is based on an acute decrease of GFR, as reflected by anacute rise in Cr levels and/or a decline in UO over a given time interval. Recently several biomarkers have been proposed for the diagnosis of AKI and these are in various stages of development and validation. Nevertheless, it is not clear, if a single or multiple biomarker approach is necessary to diagnose the complicated and multifactorial aspects of AKI.

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Who get acute renal failure

Acute renal failure is increasingly common, particularly in elderly people, although reported incidences vary according to the definition used and the population studied. In 1993 a community based study found an incidence of severe acute renal failure (serum creatinine > 500 µmol/l) of 172 per million adults per year, of whom 72% were over 70.Age related incidence rose from 17 per million per year in adults under 50 to 949 per million per year in the 80-89 age group. More recent prospective studies report an overall incidence of acute renal failure of almost 500 per million per year and an incidence of acute renal failure needing dialysis of more than 200 per million per year. This is double the UK incidence of end stage renal disease needing dialysis and places high demands on healthcare resources.Acute renal failure accounts for 1% of hospital admissions and complicates more than 7% of inpatient episodes,mostly in patients with underlying chronic kidney disease. When the condition is severe enough to need dialysis in-hospital mortality is around 50%, and it may exceed 75% in the context of sepsis or in critically ill patients

CHRONIC RENAL FAILURE

Chronic kidney disease (CKD) is a type of kidney disease in which there is gradual loss of kidney function over a period of months to years. Initially there are generally no symptoms; later, symptoms may include leg swelling, feeling tired, vomiting, loss of appetite, and confusion. Complications include an increased risk of heart disease, high blood pressure, bone disease, and anaemia.

Causes of chronic kidney disease include diabetes, high blood pressure, glomerulonephritis, and polycystic kidney disease. Risk factors include a family history of chronic kidney disease. Diagnosis is by blood tests to measure the estimated glomerular filtration rate (eGFR), and a urine test to measure albumin(38).Ultrasound or kidney biopsy may be performed to determine the underlying cause. Several severity-based staging systems are in useInitial treatments may include medications to lower blood pressure, blood sugar, andcholesterol(40).Angiotensin converting enzyme inhibitors (ACEIs) or angiotensin II receptor antagonists (ARBs) are generally first-line agents for blood pressure control, as they slow progression of the kidney disease and the risk of heart disease. Loop diuretics may be used to control edema and, if needed, to further lower blood pressure(41).NSAIDs should be avoided.Other recommended measures include staying active, and certain dietary changes such as a low-salt diet and the right amount of protein. Treatments for anemia and bone disease may also required(42). Severe disease requires hemodialysis, peritoneal dialysis, or a kidney transplant for survival.Chronic kidney disease affected 753 million people globally in 2016: 417 million females and 336 million males. In 2015 it caused 1.2 million deaths, up from 409,000 in 1990.The causes that contribute to the greatest number of deaths are high blood pressure at 550,000, followed by diabetes at 418,000, and glomerulonephritis at 238,000.

Kidney injury biomarkers:

By the time KDIGO SCr criteria for AKI are met, the decline in glomerular filtration rate (GFR) and likely structural damage that preceded that decline have been present for several hours. It has been hypothesized that delayed detection of AKI is one of the reasons why intervention trials aimed at treating AKI have failed. Therefore, a lot of effort has been put into finding biomarkers that could detect kidney injury earlier, before functional biomarkers (SCr and serum cystatin C) have changed, and which would be related to the clinical course of AKI, predict the need of dialysis, or other complications. These biomarkers provide information on tubular injury, which commonly precedes functional decline. In Table 2, the most well-studied biomarkers are summarized. Of these, liver-type fatty acid-binding protein (L-FABP) is approved for use in Japan, neutrophil gelatinase-associated lipocalin (NGAL) may be used in some localities in Europe and the combination of tissue inhibitor of metalloproteinase-2 (TIMP-2) and insulin-like growth factor-binding protein 7 (IGFBP-7) is approved for use in the USA.

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Table 2. Biomarkers of acute kidney injury

Type of biomarker	Biomarker	Description	Kinetics
Tubular injury	Kidney injury molecule 1 [10]	Tested in urine. Upregulated after injury to proximal tubuli. Activates immune cells leading to clearance and remodeling of injured cells.	Detected 12–24 h after injury, and will peak at 48–72 h post-injury
	IL-18 [11]	Tested in urine and serum. Upregulated after ischemic injury to proximal tubuli. Has pro-inflammatory characteristics.	Detected within the first 6 h after injury, and will peak at 12–18 h post-injury
	NGAL [12]	Tested in urine and serum. Is released both from distal and proximal tubuli from damaged cells and actives protective enzymes, and prevents production of radicals. NGAL is also released from liver and neutrophils in sepsis.	Detected within 3 h of injury, and will peak at 6 h post-injury
	L-FABP [13]	Tested in urine. Protein that is expressed in proximal tubuli after ischemic injury.	Detected within 1 h after injury, and will peak within 6 h post-injury
	TIMP-2 and IGFBP-7 [14]	Tested in urine. Both these biomarkers induce G1 cell cycle arrest that prevents proliferation of endothelial cells.	Detected within 12 h of injury
Glomerular filtration	Cystatin C [15]	Tested in serum. Protein, which is produced at a constant rate and filtered freely, reabsorbed and metabolized in the proximal tubuli.	Detected 12–24 h after injury, and will peak within 48 h post-injury

Dialysis :

The current recommendation on when to start RRT involves life-threatening changes in fluids, electrolytes, the acid–base balance or uremic complications [1]. However, controversy exists over the benefit of initiating dialysis at an early stage, when life-threatening complications have not yet developed, versus later stage [98]. The accumulated data from clinical trials with varying quality and observational studies have not concluded an optimal timing for start of RRT. A recent RCT [The Artificial Kidney Initiation in Kidney Injury (AKIKI) trial] found that early versus delayed RRT in the intensive care unit offered no benefit in terms of outcome [99]. While the most recent randomized trial [the Early vs Late Initiation of Renal Replacement Therapy in Critically Ill Patients With Acute Kidney Injury (ELAIN) trial] showed that early initiation of continuous RRT reduced mortality, hospital length of stay and duration of RRT compared with those with late initiation [100]. Interestingly, the late initiation group in the ELAIN trial resembled the early initiation group in the AKIKI trial, and may explain the contradictory results.

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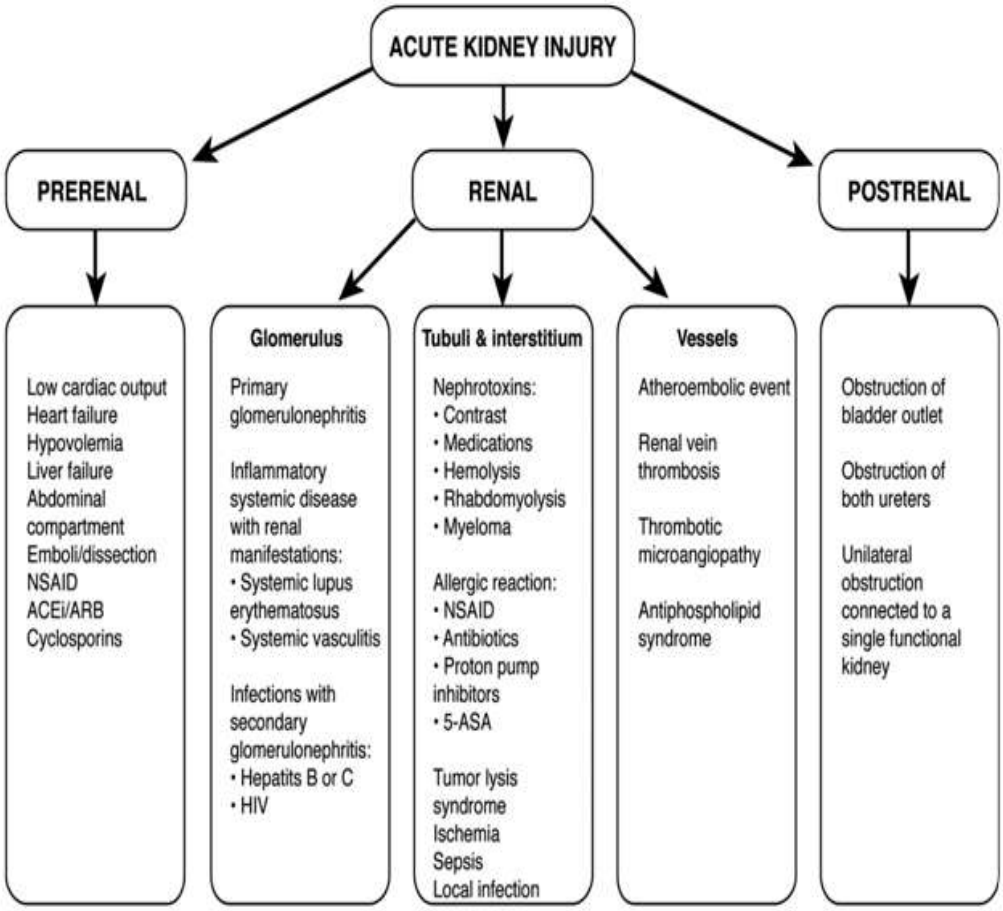

Treatment of acute renal failure

Treat underlying cause

- ✓ Blood pressure
- ✓ Infections
- ✓ Stop inciting medication s
- ✓ Nephrostomy tube ureteral stent s if obstruction
- ✓ Treat scleroderma renal crisis with ACE inhibitorRenal replacement therapy. Dialysis and renal transplant

Treatment

Depending on the cause, some types of kidney disease can be treated. Often, though, chronic kidney disease has no cure.Treatment usually consists of measures to help control signs and symptoms, reduce complications, and slow progression of the disease. If your kidneys become severely damaged, you might need treatment for end-stage kidney disease.

Treating the cause

Your doctor will work to slow or control the cause of your kidney disease. Treatment options vary depending on the cause. But kidney damage can continue to worsen even when an underlying condition, such as diabetes mellitus or high blood pressure, has been controlled

Treatment for end-stage kidney disease

If your kidneys can't keep up with waste and fluid clearance on their own and you develop complete or near-complete kidney failure, you have end-stage kidney disease. At that point, you need dialysis or a kidney transplant.

II. CONCLUSION

Chronic renal failure represents a critical period in the evolution of chronic ree and is associated with complications and comorbidities that begin early in the course of the disease. These conditions are initially subclinical but progress relentlessly and may eventually become symptomatic and irreversible.Acute renal failure is a serious medical condition that could complicate the course of many of your patients. The mortality rate from acute tubular necrosis is around 50% and

hasn't changed much over the past 3 decades, despite significant advances in supportive care.

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