International Journal of Pharmaceutical Research and Applications
Volume 6, Issue 6 Nov-Dec 2021, pp: 665-669 www.ijprajournal.com ISSN: 2249-7781

Acute Kidney Injury: Epidemiology, Pathophysiology, Aetiology, Treatment.

Kumar S Giri*[1] Aditya G Tamnar[2] Vaibhav V Kakade[3]
Department of Pharmacology, HSBPVT's, College of Pharmacy, Kashti, Ahmednagar, Maharashtra

Submitted: 01-12-2021 Revised: 11-12-2021 Accepted: 14-12-2021

ABSTRACT:
Acute kidney injury is a clinical syndrome. Recent updates in clinica research will Be of his syndrome and in the elucidation of thats pathogenesis. With this knowledge we will able to conduct more accurate epidemiologic studies the effort to gain a better understanding of the impact of its syndrome. We will also study the aetiology of the syndrome .AKI is a syndrome that rarely has the sole and distinct pathophysiology. Recent evidence,ofthe basic science and clinical research, is beginning to change our views for AKI from a single organ failure syndrome to a syndrome where the kidney plays aactive role in the progress of multi-organ dysfunction. In this review we provide the most recent updates in the epidemiology ,aetiology , pathophysiology, sign and symptoms, tests and finally treatment of AKI.

KEYWORDS: Elucidation, pathogenesis, syndrome.

I. INTRODUCTION:

Acute Kidney Injury (AKI) is a term that has recently replaced the term ARF. AKI is defined as a abrupt (within hours) decrease in kidney function, which encompasses both injury (structural damage) and its impairment (loss of function). It is the syndrome that rarely has sole of distinct pathophysiology. Many patients with AKI having a mixeaetiology wherecomplicate recognition and treatment. Furthermore a syndrome is common among patients without critical illness and it is essential that health care professionals, particularly those without specialisation A renal disorders, detect it easily.

The concept of Acute Renal Failure (ARF)1 has a undergone significant re-examination in recent years. Traditionally, emphasis was given the most severe acute reduction in kidney function, as manifested The severe azotaemia and often by oliguria or anuria. However, the recent evidence suggests that even relatively mild injuryof kidney function manifested by small changes in a serum creatinine (sCr) and/or urine output (UO), is a predictor of the serious clinical consequences.

Classification of the AKI includes pre-renal AKI, acute post-renal obstructive nephropathy and its intrinsic acute kidney diseases.Of these, only 'intrinsic' AKI represents the true kidney disease, while pre-renal and post-renal AKI are the consequence of extra-renal diseases to leading to the decreased glomerular filtration rate (GFR). If these pre- and the post-renal conditions persist, they will be eventually evolve to renal cellular damage and hence the intrinsic renal disease.

EPIDEMIOLOGY:

The lack of standard definition of the syndrome had the great impact in the reported incidence and clinical significance of AKI and its true form of impact is not well known. The incidence varies, depending on the It's tused, patient population and geographical area studied.
Large differences are observed in world that causes of AKI between developing and developed countries.

In the urban areas the developing countries and developed countries the , main causes of AKI in hospital acquired (renal ischaemia, sepsis and nephrotoxic drugs) while in the rural areas it is more commonly the consequence of community acquired disease (diarrhoea, dehydration, infectious diseases, animal venoms etc.). In other countries AKI especially in developing countries is also a major problem that relates with the true knowledge of its impact in many parts of the world.

On the other hand community AKI is usually uncommon although a recent study estimated its incidence at 4.3% among all hospital admissions.However even this incidence remains an underestimate of the true impact of the community acquired AKI due to non-referral patients to hospitals.
In the recent large scale epidemiologic study, the incidence of AKI in the hospitalised children in the

International Journal of Pharmaceutical Research and Applications
Volume 6, Issue 6 Nov-Dec 2021, pp: 665-669 www.ijprajournal.com ISSN: 2249-7781

US was found to occur in 3.9 per 1000 admissions.The majority of AKI cases in children are secondary of the volume responsive mechanisms (e.g. diarrhoea and the renal hypoperfusion after surgery) and secondary to sepsis.

Multiple studies have shown that AKI is the elderly (usually defined as older than 65 years) is increasingly common and there is age-dependent relationship between AKI and older age.This has been a attributed in the part of to anatomic and the physiologic changes in the ageing kidney and in part to various comorbidities - i.e. hypertension, cardiovascular disease, chronic kidney disease (CKD) - that may require procedures and the medications that act as kidney stressors and alter renal haemodynamics or nephrotoxic.

Several studies have also shown that AKI is the associated with short and long term adverse outcomes. These have been reviewed recently in updates.

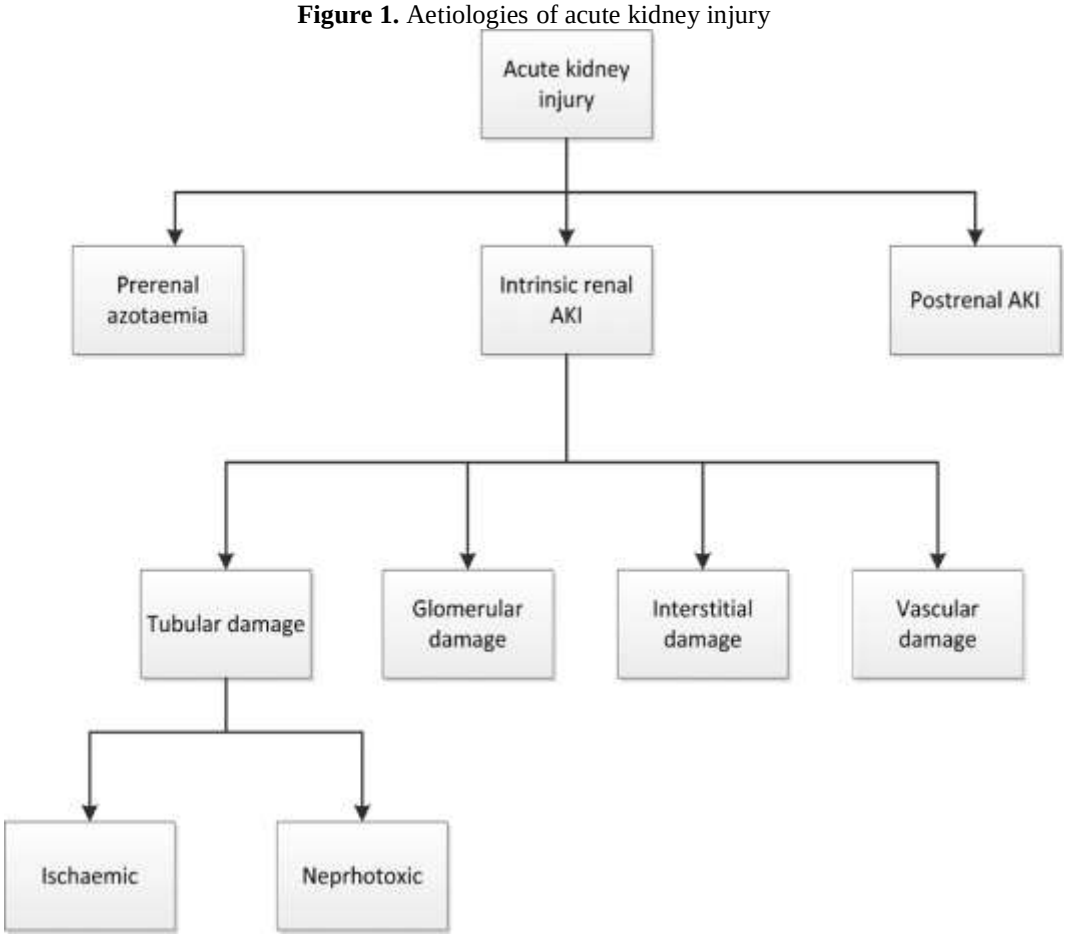
AETIOLOGY:

There are numerous Of the causes of AKI, mainly related to the focal mismatch between oxygen and nutrient delivery (because of impaired microcirculation) to the nephrons andincreased energy demands (due to the cellular stress).For many years that diagnosis and management of AKI was based on the concept of classification to the three main categories: pre-renal, intrinsic and post-renal.

- **Pre-renal AKI**, renal hypoperfusion leads to an decreased GFR (without damage to the renal parenchyma), as an adaptive response to the various extra-renal insults.It is known as the maintaining a normal GFR is dependent on the adequate renal perfusion of kidney. The kidneys receive up to 25% of cardiac output and thus any failure of the systematic circulating blood volume or isolated failure of the intra-renal circulation can have been a profound impact on renal perfusion.
- **Post-renal AKI** occurs after the acute obstruction of the urinary flow, which Is intra-tubular pressure and thus decreases GFR.63 In addition, acute urinary tract obstruction can lead to the impaired renal blood flow and inflammatory processes that contribute to diminished GFR.Post-renal AKI can develop if obstruction is located at any level of the urinary collection system the (from the renal tubule to urethra). In case the obstruction is above the bladder it must involve both kidneys and (or one kidney in the case of a patient with a single functioning kidney) to produce significant an renal failure.However, a patient with pre-existing renal insufficiency may Have AKI with obstruction of only one of kidney. Urinary obstruction may present in as anuria or intermittent urine flow (such as polyuria alternating with oliguria) but may also present as annocturia or nonoliguric AKI . Timely reversion of pre-renal or the post-renal causes usually results in prompt recovery of function, but late correction can lead to The damage.
- **Intrinsic renal aetiologies of AKI** can be challenging to the evaluate because of the wide variety of injuries that can be occur to the kidney. Generally, four structures of its kidney are involved including **tubules, glomeruli, the interstitium**, and **intra-renal blood vessels**.

International Journal of Pharmaceutical Research and Applications
Volume 6, Issue 6 Nov-Dec 2021, pp: 665-669 www.ijprajournal.com ISSN: 2249-7781

Figure 1. Aetiologies of acute kidney injury



PATHOPHYSIOLOGY:

Essentially AKI is the term used to describe the clinical syndrome that occurs when renal function is acutely decreased to the point that the body accumulates waste products and becomes unable to its maintain electrolyte, acid-base and water balance.

The pathophysiology of AKI is and complex. The most common cause of AKI is ischaemia, which can occur for the number of reasons. Physiological adaptations, in response to itse reduction in blood flow can compensate to a certain degree, the resulting cellular injury leads to organ dysfunction. The kidney is the highly susceptible to injury related to ischaemia, resulting in vasoconstriction, endothelial injury, and activation of inflammatory processes. ischaemia compromising blood flow to the critical nephron structures present therein. Following the reduction in effective kidney perfusion, the epithelial cells are the unable to maintain adequate intracellular ATP for essential processes. This ATP-depletion leads to cell injury and if it is an severe enough can lead to cell death by necrosis or apoptosis.

The nephron's natural function is the filter, concentrate and reabsorb many substances from the tubular lumen, and the concentration of these substances may reach toxic levels for the surrounding epithel cells. AKI is also very common in setting of sepsis. In sepsis the circulation anhyperdynamic and blood flow is altered, The pathophysiology of septic-AKI is very Complex involves inflammation, oxidative stress microvascular dysfunction and amplification of injury via secretion of cytokines by the tubular cells.The traditional classification of AKI into pre-renal, intrinsic-renal and post-renal has recently been challenged since histological diagnosis is performed very rarely and the distinction.However, these latter models are a quite extreme and are not representative of the clinical manifestations of the AKI in humans, where renal blood flow never fully stops (except in certain surgical procedures i.e. a abdominal aortic aneurysm repair) but less severe forms of low blood flow followed by the

International Journal of Pharmaceutical Research and Applications
Volume 6, Issue 6 Nov-Dec 2021, pp: 665-669 www.ijprajournal.com ISSN: 2249-7781

reperfusion generally occur.The animals used in the studies are usually young and healthy but most patients developing AKI are old and with a significant comorbidities (diabetes, CKD, hypertension). Moreover in experimental animals AKI is mono-causal while in humans is often of multiple co-existing aetiologies syndrome . A further analysis of pathophysiologic mechanisms is beyond the scope of this review paper.

Table 1. Causes of acute kidney injury

Caregory	Abnormality	Possible causes
Pre-renal	Hypovolaemia	Haemorrhage. Volume depletion. Renal fluid loss. Third space.
	Impaired cardiac function	Congestive heart failure. Acute myocardial infarction. Massive pulmonary embolism .
	Systemic vasodilatation	Anti-hypertensive medications. Gram negative bacteraemia. Cirrhosis. Anaphylaxis .
	Increased vascular resistance	Anaesthesia. Surgery. Hepatorenal syndrome. NSAID medications. Drugs that cause renal vasoconstriction .
Intrinsic	Tubular	Renal ischaemia. Endogenous toxins.
	Glomerular	Acute post-infectious glomerulonephritis. Lupus nephritis. IgA glomerulonephritis. Infective endocarditis. Goodpasture syndrome . Wegener disease.
	Interstitium	Infections. Medications.
	Vascular	Large vessels. Small vessels.
Post-renal	Extrarenal obstructions	Prostate hypertrophy. Improperly placed catheter. Bladder, prostate or cervical cancer. Retroperitoneal fibrosis.
	Intrarenal obstructions	Nephrolithiasis. Blood clots. Papillary necrosis.

SIGN & SYMPTOMS :

Signs and symptoms of acute kidney injury differ depending on the causes and may include:

- Too little urine leaving the body.
- Swelling in legs & ankles and around the eyes.
- Fatigue or tiredness.
- Shortness of breath.
- Confusion.
- Nausea.
- Seizures or come in severe cases.
- Chest pain or pressure.

In some cases, AKI causes no symptoms and is only found through tests done by doctor.

International Journal of Pharmaceutical Research and Applications
Volume 6, Issue 6 Nov-Dec 2021, pp: 665-669 www.ijprajournal.com ISSN: 2249-7781

TESTS TO FIND AKI :

Depending on the causes of acute kidney injury, your doctor will run different tests if he suspects that you may have AKI. It is important that AKI is found as soon as possible because it can lead to chronic kidney insury or , even kidney failure. It may lead to heart disease or deth.

The following tests may be done:

- **Measuring urine output:** your doctor will track hou much urine you pass each day to find the causes of your AKI.
- **Urine test:** doctor will look at your urine to find signs of kidney failure.
- **Blood tests:** blood tests will help to findout levels of the creatinine, urea nitrogen phosphorus and potassium should be done by addition to blood tests for protein in order to look at kidney function.
- **Imaging tests:** imaging tests, such as the ultrasound, may help your doctor see your kidneys and look for anything is abnormal.
- **Kidney biopsy:** in some situations, your doctor will do the procedure where a tiny piece of your kidney is removed with the special needle, and looked at under an microscope.

TREATMENT:

Treatment for AKI usually requires you to stay in the hospital for while. Most people with acute kidney injury are already in hospital for another Diseases or reason. How long you will stay inta hospital depends on the cause of your AKI and how quickly your kidneys recover. In more Of cases, dialysis may be needed to help replace kidney function until To kidneys recover. The main goal of your doctor is to treat what is causing your acute kidney injury. Your doctor will work to treat all of your symptoms and complications until your kidneys recover. After having AKI, your chances are higher for a other health problems (such as kidney disease or stroke, heart disease) or having AKI again in the future. The chances for developing kidney disease and kidney failure increase every time of AKI occurs. To protect yourself, you should follow up with your healthcare provider or doctor to keep track of your kidney function and recovery. having kidney damage and to save kidney the function are to prevent acute kidney injury or to find and treat as early as possible.

II. CONCLUSION :

AKI is an important clinical syndrome associated with the poor clinical outcomes for hospitalised patients. Considerable advances have been made in the refining the definition of this syndrome and in the elucidation of its underlying pathophysiologic mechanisms and we studied the aetiology and also the signs and symptoms and treatment of AKI. These innovative will aid in the design of epidemiologic studies and randomised trials of the preventive and therapeutic interventions.

REFERENCES :

[1]. the review article on kidney injury, by Konstantinos Markis and Loukiaspanou.
[2]. Kidney injury: diagnosis and prevention and treatment in Google www.ncbi.nlm.nih.gov
[3]. Management of acute kidney injury : core curriculum 2018 in Google www.ajkd.org
[4]. Acute kidney injury –the lancet in Google www.thelancet.com
[5]. Kidney injury and failure in Google www.kidneyfund.org
[6]. Eknoyan G. Emergence of the concept of acute renal failure. Am J Nephrol 2002;22:225-30.
[7]. Uchino S, Bellomo R, Goldsmith D, Bates S, Ronco C. An assessment of the RIFLE criteria for acute renal failure in hospitalized patients.
[8]. Levy EM, Viscoli CM, Horwitz RI. The effect of acute renal failure on mortality. A cohort analysis. JAMA 1996;275:1489-94.
[9]. Hoste EA, Clermont G, Kersten A, Venkataraman R, Angus DC, De Bacquer D, et al. RIFLE criteria for acute kidney injury are associated with hospital mortality in critically ill patients: a cohort analysis.