A STUDY ON INCIDENCE, CLINICAL PROFILE AND OUTCOME OF ACUTE KIDNEY INJURY IN CHILDREN ADMITTED TO PAEDIATRIC INTENSIVE CARE UNIT (PICU) OF A TERTIARY CARE CENTRE

DISSERTATION SUBMITTED FOR THE DEGREE OF

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CERTIFICATE

This is to certify that the dissertation entitled "**A STUDY ON INCIDENCE, CLINICAL PROFILE AND OUTCOME OF ACUTE KIDNEY INJURY IN CHILDREN ADMITTED TO PAEDIATRIC INTENSIVE CARE UNIT (PICU) OF A TERTIARY CARE CENTRE**" is the bonafide work of **Dr. V. JAKANATTANE** in partial fulfilment of the university regulations of the Tamil Nadu Dr. M.G.R Medical University, Chennai, for M.D Degree Branch VII – PAEDIATRIC MEDICINE examination to be held in April 2017.

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BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled "**A STUDY ON INCIDENCE, CLINICAL PROFILE AND OUTCOME OF ACUTE KIDNEY INJURY IN CHILDREN ADMITTED TO PAEDIATRIC INTENSIVE CARE UNIT (PICU) OF A TERTIARY CARE CENTRE**" submitted by **Dr. V. JAKANATTANE** to the faculty of Pediatrics, The Tamil Nadu Dr. M.G.R Medical University, Chennai in partial fulfillment of the requirement for the award of M.D Degree Branch VII (PAEDIATRIC MEDICINE) is a bonafide research work carried out by him under our direct supervision and guidance.

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DECLARATION

I, **Dr. V. JAKANATTANE**, solemnly declare that the dissertation titled "**A STUDY ON INCIDENCE, CLINICAL PROFILE AND OUTCOME OF ACUTE KIDNEY INJURY IN CHILDREN ADMITTED TO PAEDIATRIC INTENSIVE CARE UNIT (PICU) OF A TERTIARY CARE CENTRE**" has been conducted by me at Institute of Child Health and Research Centre, Madurai under the guidance and supervision of **Prof. Dr. M. NAGENDRAN M.D.,DCH.**.

This is submitted in part of fulfillment of the regulations for the award of M.D Degree Branch VII (Paediatric Medicine) for the April 2017 examination to be held under The Tamil Nadu Dr. M.G.R Medical University, Chennai. This has not been submitted previously by me for any Degree or Diploma from any other University.

Place : Madurai **Dr. V. JAKANATTANE**

Date :

CONTENTS

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Who every good have killed, may yet destruction flee;

Who ‘benefit’ has killed, that man shall ne’er escape free!

- **THIRUKURAL**

(He who has killed every virtue yet escape; there is no escape for him who has killed a benefit)

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INTRODUCTION

INTRODUCTION

Acute Kidney Injury (AKI), erstwhile known as Acute Renal Failure (ARF) is a clinical syndrome appertaining to a reversible accumulation of urea, creatinine and nitrogenous waste products and disturbances in maintenance of fluid and electrolyte homeostasis[(1)].

Acute Kidney Injury substituted the term Acute Renal Failure in view of the following reasons. The term *failure* reflects only part of the spectrum of damage to the kidney that occurs clinically. In most cases of damage, the reduction in kidney function is submissive. Moreover, the term *renal* is less recognized by the general population making communication with patients and family more challenging, hence "kidney" has replaced "renal".[(2)]

Acute kidney injury (AKI) is a common co-morbidity in critically ill children and is associated with an increased risk of morbidity and mortality[(3)].The etiology of acute kidney injury (AKI) is complex and multifactorial; some factors, such as age and sex are non-modifiable while others, including exposure to medications, are controllable and present the opportunity to decrease the risk of AKI. The reported incidence of AKI admitted to intensive care unit (ICU) varies widely in critically ill children from 10% - 80%[(4)]. The wide variations in the reported incidence of AKI are due to presence of more than 30 definitions for AKI

in previous literary texts. Therefore, it necessitated the need to establish a precise definition for AKI.

A uniform definition for acute kidney injury has existed only since 2004, when the Acute Dialysis Quality Initiative (ADQI) proposed the Risk, Injury, Failure, Loss, End-stage kidney disease (RIFLE) criteria[5] for AKI in adults. Later in 2007, a modified paediatric RIFLE (p-RIFLE)[6] emerged. Since then two modifications of the RIFLE: Acute Kidney Injury Network (AKIN) (2007)[9], and Kidney Disease: Improving Global Outcomes (KDIGO) (2012)[11] have emerged. All of the three modern definitions are based on changes in serum or plasma creatinine (Cr) and urine output (UO).

Clinical symptoms may be subtle in the early stages of AKI. As the kidney injury progresses and affects the glomerular filtration rate (GFR) Creatinine starts to rise. Oliguria or anuria may develop early, but sometimes the UO remains intact for quite long. Later in the course of AKI the severely diminished GFR manifests as electrolyte and acid-base disturbances, most often as elevated potassium and acidosis[82].

The pathogenesis of AKI is still poorly understood. Several different pathways have been proposed and studied. The arising consensus suggests that AKI is a syndrome with several different predisposing factors and mechanisms of pathophysiology. A growing amount of data supports the idea that risk for AKI

increases with a growing “burden of illness” whether chronic or acute[11]. AKI has significant consequences. It is associated with morbidity and permanent loss of kidney function. All severity stages of AKI are associated with significantly higher short-term[104] and long-term mortality[105].The burden of AKI based on the previous studies were reported to be around 5 % among hospitalized children and 30 % in critically ill children[12,13]. The etiology of Acute Kidney Injury is complex and multifactorial. Demographic factors and economical variations show differences among patterns of AKI in various parts of the world. Many paediatric studies on the incidence of AKI are confined to developed countries and often based on retrospective data[14-16]. Few studies have been conducted to determine the incidence and clinicoetiological profile of AKI in children from developing countries in the recent years[17,18]. Therefore, extrapolation of results from studies from the developed countries to children in developing countries may not be valid.

In India, few studies have been conducted prospectively to find out the incidence and clinical profile of AKI[19,20]. The results of those studies gives incidence of AKI in a range of 25% - 40% in critically ill children[19,20]. In a study by Sriram Krishnamurthy et al[19], conducted at JIPMER, the incidence of AKI was 5.2% among hospitalized children and 25.2% in critically ill children. Similar study done by Mehta et al at AIIMS[20], estimated the incidence of AKI to be 9%

among hospitalized children and 36.1% among critically ill children. Both these studies used AKIN staging for classifying AKI.

Knowledge on the burden of AKI (ie) incidence, clinical profile and outcome of AKI is vital for initiation of preventive and therapeutic strategies. In view of the limited data available on the incidence and clinical profile of pediatric AKI from Indian children, and the regional variations in the clinical profile of AKI, the present prospective study was conducted. There are studies comparing RIFLE and AKIN criteria, have shown little difference between them[21-23]. But these studies are limited to comparison of criteria's in adults and not in paediatric population. Bagshaw[22] et al conducted the first study in 2008 to compare the RIFLE and AKIN criteria and relate them to AKI in an ICU setting. According to this study AKIN criteria, which were derived from the renowned RIFLE criteria, was not significant in bringing substantial benefits to improve sensitivity and predictive ability. Lopes[21] et al compared AKIN and RIFLE staging system and found that AKIN classification had superior sensitivity to AKI but was inferior for outcome prediction in critically ill patients Hence this study compares the efficacy of pRIFLE and AKIN criteria in studying the incidence and outcome of AKI in PICU patients.

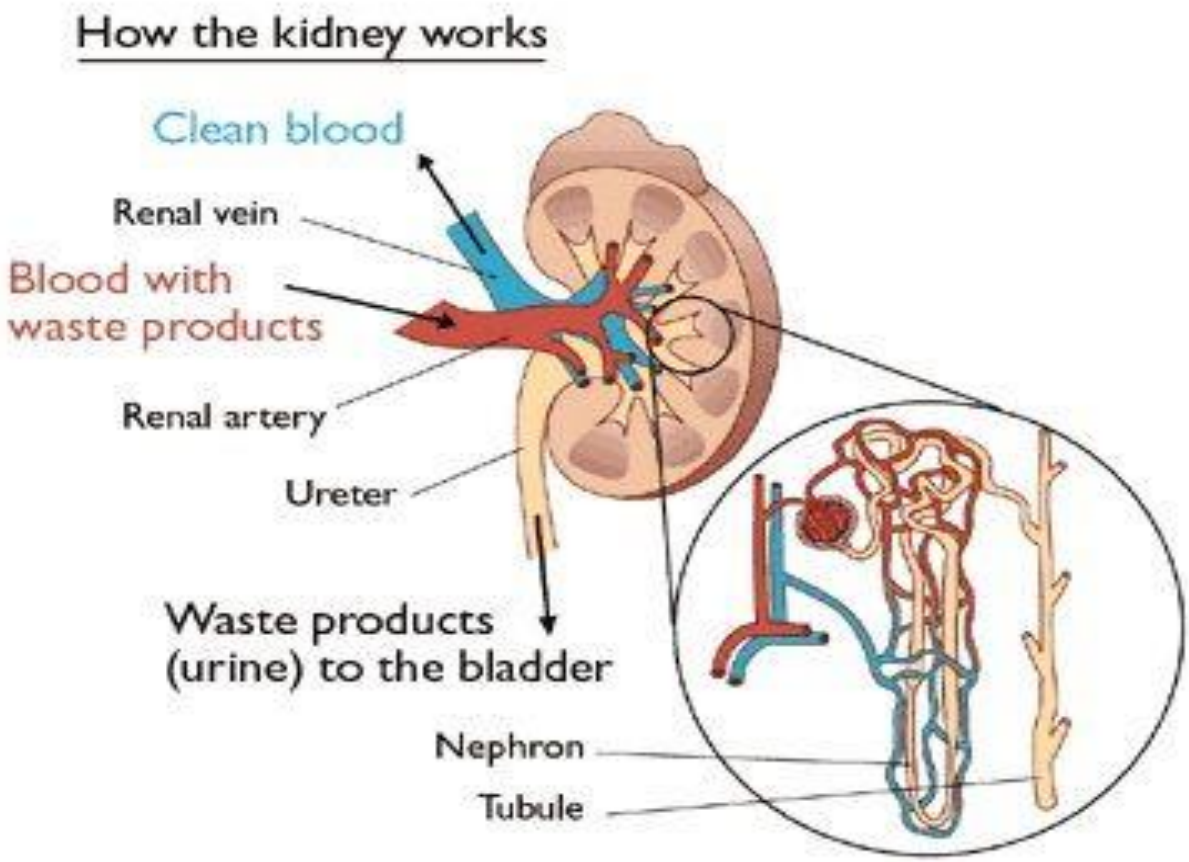
REVIEW OF LITERATURE

Kidneys are vital organ which perform the essential function of removing waste products from the blood, the regulation of sodium, potassium and other electrolytes, the regulation of fluid balances and blood pressure, the maintenance of acid-base balance, and the production of various hormones[24].

The functional unit of the kidney is the nephron, composed of a filtering unit called the glomerulus and its associated renal tubule. Each kidney is comprised of roughly one million nephrons. Arterial blood enters the kidney through the renal artery. Blood entering the glomerulus is filtered across the fenestrated glomerular capillary wall, producing an ultrafiltrate that crossed into Bowman's space and then enters the tubular lumen proper. As this ultrafiltrate traverses the length of the tubule, its composition is modified by reabsorption and secretion of specific components by the tubular epithelial cells. The end result of this process is the formation of urine, which is transported to the bladder via the ureters, and the concomitant return of cleaned blood to the circulation through the renal vein[24].

ACUTE KIDNEY INJURY

Acute kidney injury is any insult to the kidney, resulting in abrupt loss of function leading to disruption of fluid and electrolyte homeostasis. The visible and measurable symptoms of AKI include oliguria or anuria and accumulation of



products normally excreted by the kidneys such as creatinine, urea, and potassium, which as the situation progresses leads to acidosis[25].

AKI is a common comorbid condition in critically ill children[3] and adults and independently predicts mortality in these patient populations[6,13]. Any degree of kidney injury has significant implications on patient health; even mild, reversible AKI has important clinical consequences including increased mortality[26,27]. Unfortunately, mild injury to the renal system begins long before the loss of kidney function can be measured with standard tests[5,11].

The etiology of AKI has changed over the past decade from primary renal disease to complications of other systemic illness[28,29]. In critically ill patients,

AKI is commonly multifactorial[4,6,15,16,29,30], resulting from conditions including hypotension, sepsis, congenital heart disease, ischemic injury, nephrotoxins or malignancy. Regardless of etiology, the manifestations and clinical consequences of AKI are similar. Some of the major causes of Acute Kidney Injury is given in Table 2.

AKI is traditionally classified by the location of the pathophysiology relative to the kidney; 'prerenal' diseases alter the perfusion of the kidney, affecting oxygen delivery to the organ, 'intrinsic' diseases cause damage within the kidneys themselves, and obstructions of the urinary tract are considered 'post-renal' injury[1,3,13,24]. Each of the conditions related to the development of AKI causes renal injury through different mechanisms.

In prerenal AKI, regardless of cause, the result is a decrease in the perfusion of the kidney. If renal hypoperfusion is restored quickly (e.g. a rapid replenishment of fluid volume to the kidney), kidney function may be quickly restored. Otherwise, the process of acute tubular necrosis (ATN) begins. ATN is most commonly caused by a lack of oxygen (ischemia) to renal tissue; other causes of ATN are toxic or vascular insults to the kidney or through inflammatory mechanisms[24].

Table 1 : Some major causes of Acute Kidney Injury

Classification	**Etiology**
Pre-renal	Decreased intravascular volume • Dehydration • Hemorrhage • Sepsis Hypoalbuminemia Cardiac Failure
Intrinsic	Glomerulonephritis • Postinfectious/post-streptococcal • Lupus erythematosus • Henoch Schonlein Purpura Hemolytic uremic syndrome Acute tubular necrosis/ Cortical necrosis Renal vein/artery thrombosis Rhabdomyolysis Acute interstitial nephritis Tumor infiltration Tumor lysis syndrome
Post-renal	Posterior urethral valve Ureteropelvic junction obstruction Ureterovesicular junction obstruction Ureterocele, Tumor Urolithiasis Hemorrhagic cystitis Neurogenic bladder

(Nelson Textbook of Pediatrics, 20th edition page 2540)

DEFINITION OF ACUTE KIDNEY INJURY

The Acute Dialysis Quality Initiative convened an international consensus panel in 2002 and proposed the RIFLE criteria for use in critically ill adults in 2004[5].

The RIFLE classification[5] is based on Serum Creatinine (SCr) and Urine Output (UO) determinants, and considers three severity classes of AKI (Risk, Injury and Failure), according to the variations in SCr and/or UO, and two outcome classes (Loss of kidney function and End-stage kidney disease). The patient should be classified using the criteria (SCr and/or UO) which leads to the worst classification (maximum RIFLE), for example, if a patient was in the Risk class according to the UO but in the Injury class according to SCr variation, then the worst criteria (SCr) should be used for classifying the severity of AKI in this patient. RIFLE classification has been shown in Table 2.

A modification of the RIFLE (known as the pRIFLE) has been suggested for use in paediatric populations in 2007[6]. The changes are minor and include a focus on the estimated creatinine clearance, calculated using the Schwartz formula[7], as the measure of GFR. Serum creatinine in children is dependent on body mass, which is directly related to height and age of a child .Schwartz formula is therefore appropriate for use in children.(eCCl= K X length in cm/plasma creatinine in mg/dL)[7]. This formula has been validated as a good means to estimate creatinine

clearance in paediatric patients, in whom its measurement by 24 hour urine collection is challenging[8]. When a measure of the patient's baseline GFR is not available or is unknown, the panel suggested assuming a GFR of 75ml/min/1.73m2, the lower limit of normal[5]. In addition, the threshold for the 'Failure' category was modified from a serum creatinine ≥ 4 mg/100ml to an estimated creatinine clearance (eCCl) < 35 ml/min/1.73m^2, and the time interval for the urine output criteria was increased from 6 hours to 8 hours. The pRIFLE classification has been shown in Table 2.

Acute kidney Injury Network (AKIN) proposed a new classification of Acute Kidney Injury which came into practice in March 2007[9]. It is regarded as the later version of the RIFLE classification with some modifications. The diagnosis of AKI is only considered after achieving an adequate status of hydration and after excluding urinary obstruction. The AKIN classification only relies on SCr and not on GFR changes; baseline SCr is not necessary in the AKIN classification, and it requires at least two values of SCr obtained within a period of 48 hours. These modifications were based on the cumulative evidence that even small increases in SCr are associated with a poor outcome[10]. AKIN staging has been shown in Table 2.

Table 2 : Current Criteria used for diagnosis of Acute Kidney injury

Classification	Stage	Creatinine criteria	Urine output criteria
RIFLE (Bellomo et al., 2004)	Risk	Increased creatinine x1.5 or GFR decrease >25%	<0.5 ml/kg/h x 6h
	Injury	Increased creatinine x2 or GFR decrease >50%	<0.5 ml/kg/h x 12h
	Failure	Increased creatinine x3 or GFR decrease >75% or creatinine ≥4 mg/100ml (acute rise of ≥0.5 mg/100ml dl)	<0.3 ml/kg/h x 24h or anuria x 12h
	Loss	Persistent ARF = complete loss of renal function > 4 weeks (defined as the need for renal replacement therapy (RRT) for >4 weeks)	
	End-stage	End-stage renal disease (defined as the need for dialysis for >3 months)	
Pediatric RIFLE (pRIFLE) (Akcan-Arikan et al., 2007)	Risk	eCCl decrease by 25%	<0.5 ml/kg/h x 8h
	Injury	eCCl decrease by 50%	<0.5 ml/kg/h x 16h
	Failure	eCCl decrease by 75% or eCCl <35 ml/min/1.73m^2	<0.3 ml/kg/h x 24h or anuria x 12h
	Loss	Persistent failure >4 weeks	
	End-stage	End-stage renal disease (persistent failure >3 months)	
AKIN (R. L. Mehta et al., 2007)	1	Increased creatinine x 1.5-2 or creatinine increase ≥0.3 mg/dl	<0.5 ml/kg/h x 6h
	2	Increased creatinine x 2-3	<0.5 ml/kg/h x 12h
	3	Increased creatinine x ≥3 or creatinine ≥4.0 mg/dl with an acute increase of 0.5 mg/dl	<0.3 ml/kg/h x 24h or anuria x 12h
KDIGO (Kidney Disease: Improving Global Outcomes (KDIGO) Acute Kidney Injury Working Group, 2012)	1	Increased creatinine x1.5-1.9 or ≥0.3 mg/dl increase	<0.5 ml/kg/h x 6-12h
	2	Increased creatinine x2.0-2.9	<0.5 ml/kg/h x ≥12h
	3	Increased creatinine x3 or creatinine ≥4.0 mg/dl or initiation of RRT or eGFR <35 ml/min per 1.73m^2 (<18 years)	<0.3 ml/kg/h x ≥24h or anuria x ≥12h

AKIN: Acute Kidney Injury Network; GFR: glomerular filtration rate; eCCl: estimated creatinine clearance; eGFR: estimated glomerular filtration rate; KDIGO: Kidney Disease: Improving Global Outcomes; RRT: renal replacement therapy

The latest classification was proposed by the Kidney Disease: Improving Global Outcomes (KDIGO) Acute Kidney Injury Work Group, was based on the previous two classifications, and had the aim of unifying the definition of AKI[11]. According to this definition, AKI was diagnosed as an increase in Serum creatinine by at least 26.4μmol/L within 48 hours or an increase in Serum creatinine to 1.5 times baseline, which is known or presumed to have occurred within 7 days before, or a urine volume of less than 0.5 mL/kg per hour for 6 hours. For KDIGO criteria, the 26.4 μmol/L increase needs to be within 48 hours but a 1.5-fold increase can occur within 7 days to diagnose AKI; and the 1-week or 48-hour timeframe is for diagnosis of AKI, not for staging[11]. KDIGO classification has been shown in Table 2.

INCIDENCE OF ACUTE KIDNEY INJURY

The prevalence of AKI in children is on the increase due to advancement of diagnostic and therapeutic options as well as advances in paediatric and neonatal critical care[18]. The incidence of AKI among children admitted to intensive care units has been estimated to range from 10% - 35%[4,31,32]. In more severely critically ill children, the incidence of AKI has been reported to occur in 90% of patients with traumatic injuries or those needing vasopressor support and requiring mechanical ventilation[33]. The incidence of AKI ranges from 30% to 50% in children undergoing cardiac surgery[34-37]. In Indian studies, the incidence of AKI

has been estimated to be in the range of 25% - 40%[19,20,38]. The wide variability in AKI incidence may be due to differences in the definition of AKI used; however, it is reasonable to postulate that specific populations of critically ill children have higher rates of AKI due to underlying differences in risk.

OUTCOME OF ACUTE KIDNEY INJURY

AKI has been associated with increased mortality in adult patients as shown by a meta-analysis of 24 studies reported a pooled estimated mortality rate of 31.2% in patients with AKI compared with 6.9% in patients without AKI[39]. In children, the short-term outcomes of critically ill children with AKI have been well documented. High mortality rates ranging from 30% to 40% have been consistently reported in critically ill children with AKI[4,15,16,31,32,33].

When compared with other patients, mortality rates are significantly higher among children who develop AKI. In a study of critically ill neonates and children who received extracorporeal membrane oxygenation (ECMO), the adjusted odds of death in patients with AKI was three times higher (95% confidence interval (CI): 2.6-4.0) than in those without AKI[28]. In addition, a study of 430 infants who had cardiac surgery for congenital defects found that more severe AKI was associated with increased hospital mortality[35]. When compared with patients without AKI, the odds of mortality in those who developed AKI ranged from 5.1 (95% CI: 1.7 – 15.2) for patients with moderate AKI to 9.5 (95% CI: 2.9 – 30.7) for patients with

severe AKI. Acute kidney injury is also related to measures of morbidity in children[24]. In a multicentre, retrospective analysis of paediatric ICU admissions, AKI was shown to be independently associated with mechanical ventilation and increased ICU length of stay[31]. Extended use of mechanical ventilation and increased hospital stay have been shown to be independently associated with AKI in children undergoing cardiac surgery[34,35,37]. While short-term outcomes of AKI in children have been well documented, little information exists on long-term survival in paediatric patients who develop AKI. Askenazi et al (2006) followed a cohort of 174 children who developed acute renal failure during their hospitalization and survived to hospital discharge. They found a survival rate of 80% three to five years posthospital discharge; most deaths (65%) occurred within one year of the initial hospitalization[40]. It was previously thought that patients who survived an episode of AKI would recover full kidney function; a recent meta-analysis found that adults with AKI were nine times more likely to develop chronic kidney disease and three times more likely to develop end-stage renal disease compared to patients without AKI[41].

In a retrospective study of paediatric inpatients who developed AKI during their hospitalization, two-thirds had recovered full renal function and 15% had improved renal function by hospital discharge, though the definition of ‘recovered’ and ‘improved’ renal function was not clearly stated by the authors[29]. In the same

patient cohort, 14% developed chronic renal failure and 5% who were discharged needed ongoing renal replacement therapy (RRT)[29]. Among a cohort of paediatric AKI patients who survived three to five years post-hospital discharge, 29 patients consented to additional renal assessments. Residual renal injury was defined as the presence of any one of: microalbuminuria, hypertension, hematuria, or an estimated glomerular filtration rate outside a normal range, where normal was defined as 90-150 ml/min/1.73 m^2. Fifty-nine percent of patients had signs of renal dysfunction at the time of follow-up[40].

MANAGEMENT OF ACUTE KIDNEY INJURY

Treatment of AKI in critically ill children focuses on avoiding or minimizing further renal injury and preventing life-threatening imbalances of fluids or electrolytes[42]. Patients with severe AKI or those with severe fluid overload or electrolyte imbalance may require renal replacement therapy[11]. While modern RRT has largely eliminated the traditional, life-threatening complications of AKI such as hyperkalemia, arrhythmia, and uremic coma, children with AKI treated with RRT still have a high mortality rate, ranging from 30% to 50%[30,43]. This high mortality rate has remained stable over the past two decades[30,43] and is likely due to a number of factors. For example, interactions between the kidney and other major organs, known as ‘crosstalk’, result in cycle of AKI and multi-organ failure, which leads to death[44-46]. In addition, AKI also impairs the immune system,

increasing a patient's susceptibility to infection[47]. Finally, AKI often results in fluid overload, which has been shown to be an independent risk factor for mortality in AKI[48].

RISK FACTORS FOR ACUTE KIDNEY INJURY

In the ICU, AKI is usually multifactorial with several different insults affecting the kidneys in an additive way. The combined risk for each patient comprises both acute exposures and insults causing AKI, and chronic conditions and patient related factors that define how susceptible each patient is to develop AKI[11].

Table 3: Risk factors for Acute Kidney Injury[103]

Susceptibles	Very premature neonates Cardiac failure Organ transplantation (stem cell)
Exposures	Volume depletion Cardiopulmonary bypass Nephrotoxin exposure Mechanical ventilation Sepsis Vasopressors

PATHOPHYSIOLOGY OF ACUTE KIDNEY INJURY

AKI is complex, multi-etiological syndrome with several different pathophysiological mechanisms. For many years, vasomotor disturbances and ischaemic injury were the main focus of attention in the study of aetiology of AKI[25,82]. Since then, growing knowledge on the mechanisms of AKI have shown that though important, ischaemic-reperfusion injury is only one of the mechanisms causing AKI[49].

The kidneys maintain their perfusion pressure and glomerular filtration rate in different haemodynamic situations very efficiently by autoregulation with the afferent and efferent arterioles in each glomerulus reacting to vasoconstrictive and vasodilatory factors[50]. In the autoregulation range, the afferent arteriole reacts to decreased perfusion pressure with vasodilatation. In situations where the autoregulation is disturbed, such as extreme global hypotension, vascular thrombosis, vascular clamping, or oxygen depletion the response is vasoconstriction and reduction of GFR[25]. However, significant periods of isolated warm ischemia are tolerated by the kidneys without sustained injury[51]. Reperfusion following ischemia is also damaging to the tissues and this type of damage is often called ischaemic-reperfusion injury. In situations where autoregulation fails, depletion of adenosine triphosphate (ATP) follows initiation of the complex mechanisms leading from ischemia to injury.

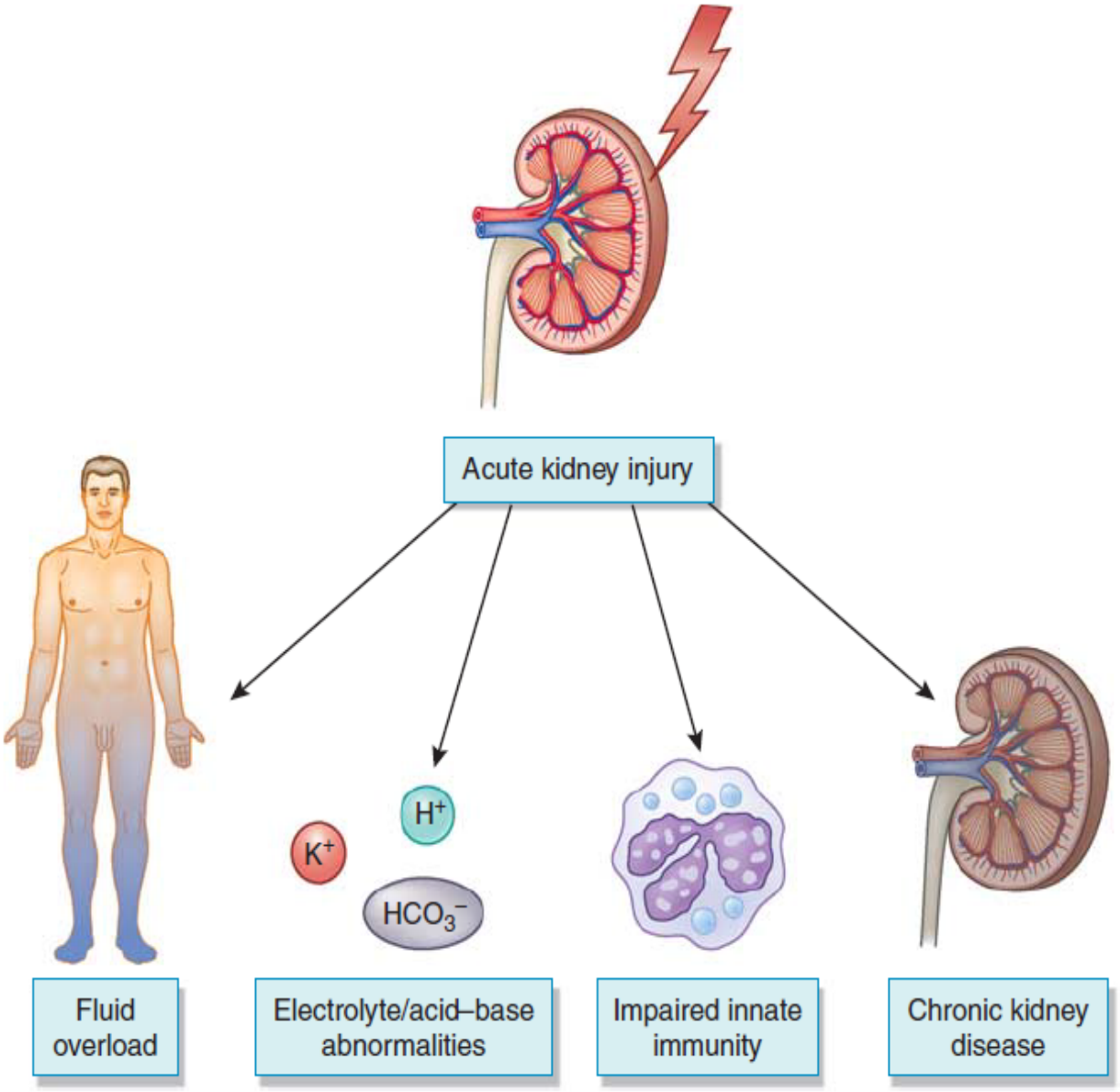


FIGURE: Effects of Acute Kidney Injury

Damage to the endothelium and release of nitric oxide (NO) seems to play a role in local imbalance of vasoactive substances[52]. These reactions are accompanied by metabolic changes[25,53], activation of the coagulation system[54], and an inflammatory reaction[55]. The damaged vascular endothelium leads to increased permeability[56], and further increased leukocyte infiltration[57]. The damaged cells in the kidneys lose their cytoskeletal structure[58] and release more proinflammatory and chemotactic substances that further enhance the reaction[59]. Obstruction of the tubules by cell casts and back leak of glomerular filtrate to capillaries may contribute to the injury[60,61]. Reperfusion injury further damages the cells via oxidative processes[53]. Most tubular cells, however, usually remain viable[62-64]. Both necrosis[64] and apoptotic processes[65] have been seen in the damaged kidney cells.

SEPTIC ACUTE KIDNEY INJURY

Sepsis is the most common predisposing factor for AKI in the critically ill[66]. Despite early assumptions[67], septic AKI is far more complex than just ischaemic-reperfusion injury resulting from poor haemodynamics or low RBF[49]. It seems that septic AKI is multifactorial, and the mechanism of development may vary significantly between patients[68,69]. It is poorly understood why only a minority of sepsis patients have a classical tubular necrosis when assessed

histopathologically[70], and actually most renal tubular cells remain intact in septic AKI[64]. Most of the data on septic AKI have been derived from animal studies[71].

Animal models have suggested considerable variability in RBF in relation to systemic haemodynamic changes in sepsis[72]. In a recent study systemic haemodynamics and RBF were measured noninvasively from septic patients showing constantly reduced RBF in comparison to cardiac output (CO)[73]. Also, in previous studies RBF and GFR have been poorly correlated[70,74]. Thus, the loss of GFR in septic AKI can occur in the presence of a normal or even hyperdynamic RBF, and because of disturbed autoregulation uncoupling of systemic haemodynamics and RBF occurs[71].

In sepsis the excessive systemic inflammatory reaction most likely plays a key role in the development of kidney injury and multiple organ failure[75]. The release of various inflammatory mediators, from pathogens and from immune cells, induces direct toxicity to tubular cells and triggers a complex cascade of inflammation[73,76].

At the cellular level, immunomodulators such as tumour necrosis factor α, Interleukin 6, and leukotrienes[77] are suggested to cause apoptosis or even necrosis in tubular cells. In addition, the inflammatory stimulus induces the release of nitric

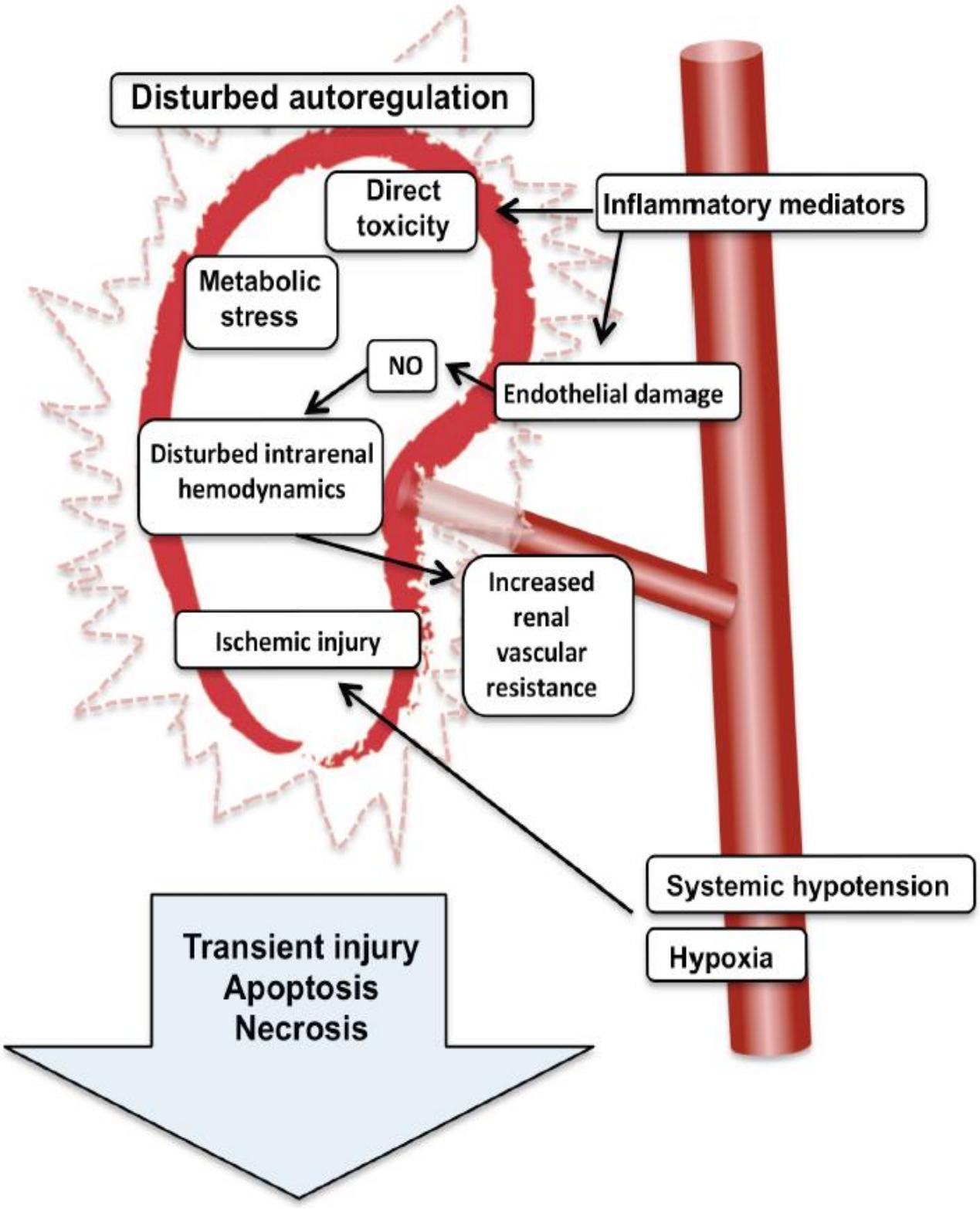


Figure 2 : Pathogenesis of Septic Acute Kidney Injury[82]

oxide (NO) in response to endothelial damage causing disturbances in intrarenal hemodynamics[78] and shunting in the periglomerular system. It has been suggested that excess dilatation of the efferent arteriole compared to the afferent arteriole[79,80] would lead to "local hypotension" in the glomeruli and loss of GFR. In response, the renin-angiotensin-aldosterone (RAA) system[81] is activated leading to increased renal vascular resistance[72], further decreasing RBF. Oxidant stress, mitochondrial dysfunction, and microcirculatory abnormalities have also been proposed as contributors to septic kidney injury, but the role of these mechanisms remains unclear[69].

MEASURING KIDNEY FUNCTION

1. GLOMERULAR FILTRATION RATE

As the main function of the renal system is the removal of waste products from the body, the glomerular filtration rate (GFR), defined as the volume of plasma cleared of a substance per unit of time, is widely accepted as the most useful overall index of kidney function[83]. Ideal substance to measure GFR is one that is excreted only by the kidneys and not reabsorbed. Inulin, a substance not naturally available in the human body meets these criteria. Radioactive markers can also be used to measure GFR[84], however these measures are not practical for routine clinical use. Creatinine, an endogenous substance that is closest to an ideal[85] and creatinine clearance, the amount of creatinine cleared from the blood during a

given time period, can be used to estimate GFR. However, measuring creatinine clearance requires a 24 hour urine collection which can be both difficult and inconvenient for patients[83]. Creatinine clearance also tends to overestimate the true GFR[86]. As such, a number of formulae have been derived to estimate GFR based solely on serum creatinine levels, known as estimated creatinine clearance. In adults, the Modification of Diet in Renal Disease (MDRD) formula is the most widely used[87]. The MDRD estimates GFR using creatinine, age, sex and race (African-American versus other races). The formula is not accurate for use in children, the elderly, those with unusual muscle mass and weight (e.g. morbidly obese patients)[24]. In children, the Schwartz formula is widely used[7]. The Schwartz formula estimates a child's GFR based on the child's height and serum creatinine. In AKI studies CrCl/GFR equations are used to estimate a baseline creatinine for patients lacking it by back calculating with the assumption of a normal GFR of 75 ml/min / 1.73 m^2[87].

In clinical practice, a rapid decline in GFR, indicative of kidney injury, is assessed by an increase in serum creatinine and/or oliguria. Creatinine is insensitive to changes in the GFR; the concentration of Creatinine starts to rise when half of the kidney function has already been lost[88]. Changes in Creatinine are therefore slow after an injury to the kidneys. The correlation of GFR and urine output is not linear. Urine output might be normal in AKI because of tubular injury

and impaired concentration ability[11,89]. Low urine output can be a result of urinary track obstruction. In addition, diuretics or other medications may alter the diuresis. In very obese patients, the straightforward utilizations of urine output per weight (ml/kg/h) leads to overestimation of AKI[11,89]. However current classifications for AKI are based on changes in serum creatinine and/or urine output[5,6,9,11]. Other diagnostic tools, including clinical history, physical examination, renal ultrasound, fractional excretion of sodium (FeNa), fractional excretion of urea, blood urea nitrogen (BUN), and urine microscopy, may point towards the etiology of the renal injury.

BIOMARKERS IN ACUTE KIDNEY INJURY

Due to known limitations in the current gold standard for AKI (creatinine and diuresis), new biomarkers to recognize AKI more sensitively, specifically, and earlier are needed. These markers identify early stress responses of the kidney and appear in the urine or plasma before changes in serum creatinine are evident[90]. Properties of an ideal biomarker would be[91,92]

- must be generated by damaged, but not healthy cells.
- concentration in the body must be proportional to the extent of the damage
- should be expressed early after damage.
- concentration should decrease rapidly after the acute injury to enable therapeutic monitoring.
- should be easily, rapidly, and reliably measurable.

The most widely studied and validated early biomarker of AKI in children is neutrophil gelatinase-associated lipocalin (NGAL). Levels of NGAL in the urine and plasma of children undergoing cardiopulmonary bypass were significantly elevated within 2-6 hours after bypass in children who subsequently developed AKI[93-96]. Increased NGAL measurements have also been shown to be associated with hospital length of stay and the duration and severity of AKI[93-96].

Studies in the paediatric ICU and emergency department settings have shown that increases in NGAL predicted AKI roughly one to two days before corresponding increases in serum creatinine were evident[97-99]. Two recent meta-analyses of NGAL studies in critically ill adults and children have confirmed the clinical utility of this marker as an early indicator of AKI and showed significant associations between increasing NGAL levels and clinical outcomes including mortality, length of stay and the initiation of renal replacement therapy[100].

Serum cystatin C levels have also been shown to be highly correlated to AKI and can be used as a marker of disease progression after kidney transplantation[101]. In addition, kidney injury molecule-1 (KIM-1), interleukin-18 (IL-18), and liver fatty acid binding-protein have been shown to be associated with renal ischemia[102]. The use of biomarkers is a rapidly developing field; however, to date, biomarkers have not been incorporated into clinical practice and thus are not currently a practical method to assess and diagnose acute kidney injury.

Finally, similar to the idea of cardiac angina, the concept of 'renal angina' has recently been introduced to identify patients who may benefit the most from early treatment to manage or prevent the development of AKI[106]. There are few signs and symptoms in the early stages of AKI when interventions are likely to be the most effective and the definition places children at moderate, high or very high risk of developing AKI. As the risk for AKI increases, less laboratory evidence of AKI (i.e. specified changes of serum creatinine) is needed to meet the threshold for a diagnosis of renal angina.

Recently, the idea of renal angina has been further developed into an index for use in critically ill children.

Risk	Description	Risk
Moderate	ICU admission	1
High	Transplantation	3
Very High	Mechanical Ventilation & Inotrope use	5

X

Injury (eCrCl)	Injury (Fluid Overload)	Injury Score
No change	<5%	1
0 - 25% ↓eCrCl	≥5 %	2
25-50% ↓eCrCl	≥10%	4
>50% ↓eCrCl	≥15%	8

Renal angina index ranges from 1 – 40 and a Index ≥ 8 is considered Angina (+).

For the purpose of this dissertation both p-RIFLE and AKIN staging were used. Similar to the RIFLE, the AKIN definition also defines levels of severity, defined as Stages 1, 2 and 3, which correspond to RIFLE severity levels of 'Risk', 'Injury' and 'Failure', respectively. Also similar to the RIFLE, the AKIN is based on both changes in creatinine and urine output; however, it also adds a measure of time. The time constraint of 48 hours for diagnosis was selected based on evidence that small increases in creatinine within 24 to 48 hours were associated with a threefold increase in 30-day mortality[110]. Studies have shown that the RIFLE and AKIN criteria show concordance in critically ill patients[111].

AIMS AND OBJECTIVES

1. To determine the Incidence of Acute Kidney Injury in critically ill children admitted to Paediatric Intensive Care Unit (PICU) of a Tertiary care centre.
2. To determine the Clinical profile and outcome of children with Acute Kidney Injury in critically ill children.
3. To determine the predictors of mortality.
4. To compare Acute Kidney Injury Network (AKIN) Staging and p-RIFLE classification in Paediatric age group.

MATERIALS AND METHODS

The design is a prospective observational study of critically ill children admitted to Paediatric Intensive care Unit (PICU) at Institute of Child Health and Research Centre, Govt. Rajaji Hospital, Madurai.

- All children within the age group of 1 month to 12 years with length of stay for atleast 48 hours in PICU over a period of 1 year (July 2015 – June 2016) were included in the study after getting consent from parents.
- PICU admission was based on one or more of the following criteria:
    - Impaired level of consciousness (Glasgow coma scale < 8)
    - Signs suggestive of severe increase in intracranial pressure (e.g., hypertension, bradycardia, papilledema)
    - Hypoventilation or respiratory failure (oxygen saturation < 90% or arterial oxygen (PaO_2) <60 mmHg with supplemental oxygen or arterial CO_2 ($PaCO_2$) >60 mmHg)
    - Uncontrollable or poorly controlled seizures
    - Hypotension requiring inotropic support
    - Requirement of renal replacement therapy (RRT)
    - Fulminant hepatic failure.

- EXCLUSION CRITERIA
    - Patients with known chronic kidney disease
    - Bilirubin level >5 mg/dl
- Institutional ethical committee approval obtained

SAMPLE SIZE ESTIMATION

Sample size was calculated using the formula $4pq/d^2$

P – incidence of Acute Kidney Injury

Q – (1-P)

D – absolute precision

The incidence of Acute Kidney injury in critically ill children was estimated to be around 30% based on current literature and assuming an variation of 5% (absolute precision d = 0.05), the sample size was estimated to be around 335.

The study subjects were enrolled consecutively until the sample size was achieved. A detailed clinical history and a thorough physical examination was conducted as soon as the patient was stabilized and weight, height, temperature, blood pressure, pulse, respiratory rates, capillary refill, oxygen saturation, presence of dehydration, presence of anemia, presence of edema were noted. Systemic examination also was done.

Height was measured for those children who were 2 years and above, and were able to stand, using a stadiometer. This was made locally with a tape attached to a board, which had a fixed headpiece and a mobile footpiece. Those younger than 2 years, or those too sick to stand had their length taken using a stadiometer placed flat on a table. This procedure required 2 people; one ensures whether the heels and knees touch the board with feet together while the other ensures that the head and back are in position against the board with eyes looking straight ahead. The footpiece or headpiece was then moved to touch the child, and the reading taken for length and height respectively, and recorded to the nearest centimeter.

Blood pressure (BP) measurement was done using sphygmomanometer, which has three velcro paediatric cuff sizes to fit infants, toddlers and older children. The proper cuff width was selected based on the width of the BP cuff which is almost half the circumference of the arm and also long enough to encircle the arm. With the child in sitting position, the first Korotkoff sound and the fifth Korotkoff sounds were recorded as the systolic and the diastolic BP measures respectively. The measurement was repeated and an average of the two was recorded.

The diagnosis of Acute Kidney Injury was based on the AKIN staging; pRIFLE classification was also used to diagnose AKI for the purpose of comparing AKIN staging and p-RIFLE classification. Serum creatinine or urine output was used to diagnose and stage AKI, using a criterion that led to a higher stage classification.

Data collected includes demographic information, admission diagnoses/final diagnosis and co-morbidities, serum creatinine at the time of admission, other hematological and metabolic parameters. A total of 4 ml of intravenous blood was withdrawn (2 ml for complete blood count and 2 ml for renal and liver function tests) and centrifuged. Serum creatinine estimation was performed by modified Jaffe method[107] using the autoanalyzer. This measured value was considered as "initial" serum creatinine. Estimation of serum creatinine was repeated daily for 3 consecutive days and daily thereafter until discharge from hospital. Urine output was measured 6th hourly in PICU.

An absolute increase in serum creatinine of ≥0.3 mg/dL or an increase in serum creatinine of more than or equal to 1.5-fold from the initial serum creatinine was considered as AKI. Similarly, a decrease in serum creatinine of more than or equal to 0.3 mg/dL or a decrease in serum creatinine of ≥1.5-fold from the initial serum creatinine was also considered as AKI. If there was a progressive rise in serum creatinine values, re-classification and progression to maximum AKI stage during the hospital stay was recorded. Indications for RRT were as per standard hospital protocols.

Abdominal ultrasound was done for children fulfilling the criteria for acute kidney injury once they were resuscitated, to determine the presence of kidneys, kidney size, a description of the renal parenchyma, and evaluation for the presence

of urinary tract obstruction. Ultrasound abdomen was done by qualified radiologist.

Estimated creatinine clearance (eCCl) was calculated as percent change of daily creatinine from baseline creatinine (using Schwartz formula), Baseline creatinine used is lowest consistent serum creatinine 90 days or more prior to admission. For patients without a prior baseline, an assumed creatinine clearance of 75 ml/min/1.73 m^2 is used[5,87].

Normal GFR for age= K X Length (cm) / Serum creatinine (mg/dL)

(where the constant K=0.45 for infants, and 0.55 for children and adolescents) [87]

Short term outcomes (complete renal recovery, partial renal recovery and death) were recorded. Complete renal recovery was defined as normal serum creatinine for age (0.2-0.4 mg/dL for infants, 0.3-0.7 mg/dL for 1-12 years, 0.5-1 mg/dL for >12 years) and normal blood pressure at discharge. Partial renal recovery was defined as elevated serum creatinine for age or persistent hypertension at discharge.

- ADQI[5] – definition for functional AKI recovery
- Complete renal recovery – return of creatinine value to less than the threshold for RIFLE-R or within 50% of baseline

- Partial renal recovery – patients are off RRT, but fail to return to within 50% of baseline serum creatinine
- Non recovery – patients who require persistent RRT

But there are certain limitations for the definition as it,

- Depends on the presence of baseline creatinine.
- Lacks clarity about the role of urine output in the recovery process.

Pediatric risk of mortality III score was used for assessment of severity of illness. Patients were followed-up until 3 months after discharge.

Shock was defined as the presence of at least two of the following: Tachycardia (heart rate > 2 SD for age), feeble pulses, cool peripheries and hypotension (blood pressure <−2SD for age and sex) or capillary filling time > 3 s, temperature instability[108]. Blood pressure >95th percentile for age, height and gender was labeled as hypertension. Sepsis was defined according to the International pediatric sepsis consensus conference definition[109].

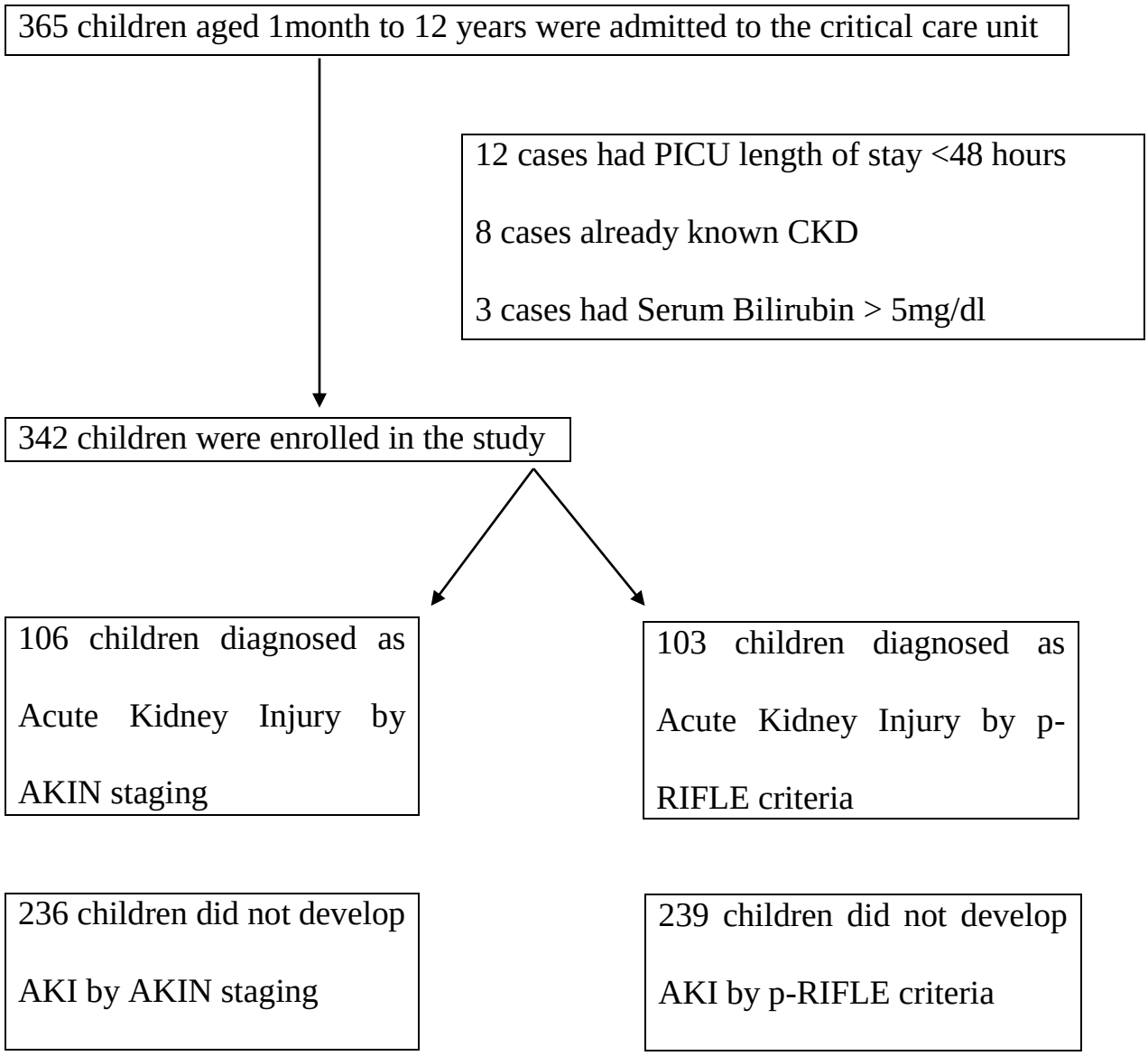
Other variables recorded include the use of mechanical ventilator, inotropes, length of stay in the Intensive Care Unit, use of Renal Replacement Therapy (RRT).

STATISTICAL ANALYSIS

The data collected regarding all the selected cases were entered in Microsoft excel sheet 2010. Results were analyzed using the SPSS version 19 (IBM corporation, New York, U.S.A). Continuous data were reported as mean ± SD (if normally distributed) and median (range) (if non-normally distributed). Categorical variables were expressed as proportions. The incidence of AKI was defined as its occurrence as a proportion of total admissions. Continuous variables with normal distribution were compared using Student *t*-test while those not normally distributed were analyzed using Mann Whitney U test. Categorical data were analyzed using Pearson Chi-square test or Fischer exact test. P value was calculated using chi square test. Multivariate binary logistic regression models were used for multivariate analysis of statistically significant variables in univariate analysis ($P < 0.05$), to determine predictors of fatality in AKI.

OBSERVATION AND RESULTS

Our study enrolled 342 children in the time period of 12 months and observed for the development of Acute Kidney Injury.



On the whole, 342 critically ill children admitted to PICU were screened for AKI. 106 children developed AKI giving an incidence of 31% (by AKIN staging). 103 children developed AKI using p-RIFLE classification giving an incidence of 30.1%. Both AKIN staging and p-RIFLE classification were statistically significant in detecting the number of AKI cases.

Of the children enrolled in our study, 198 (57.9%) were male and 144 (42.1%) were female. Of the 106 children who developed AKI, 58 (54.7%) were male and 48 (45.3%) were female. The median age of 342 children was 30 months (IQR = 2 – 144 months), with majority of the participants under 60 months of age (68.4%, 234/342). The median age in children with AKI was 36 months (IQR = 2 – 144 months, with majority of children under 60 months of age (67%, 71/106).

The severity of Acute Kidney Injury was given by the staging of Acute Kidney Injury. According to AKIN staging, stage 1 included 43 (40.6%) cases, stage 2 included 28 (26.4%) cases, stage 3 included 35 (33%) cases. According to p-RIFLE classification, 35 (34%) children were included in Risk category, 31 (30.1%) were included in the Injury category and 37 (35.9%) were included in the Failure category. 3 cases of Risk category progressed to Injury category and 3 cases to Failure category while 1 case from Injury category progressed to Failure category. The mean level of maximum creatinine value in AKI children was estimated to be 2.1±1.7 mg/dl.

TABLE 4: SEX DISTRIBUTION

SEX	AKI	NON-AKI	TOTAL
MALE	58 (54.7%)	140 (59.3%)	198 (57.9%)
FEMALE	48 (45.3%)	96 (40.7%)	144 (42.1%)
TOTAL	106 (100%)	236 (100%)	342 (100%)

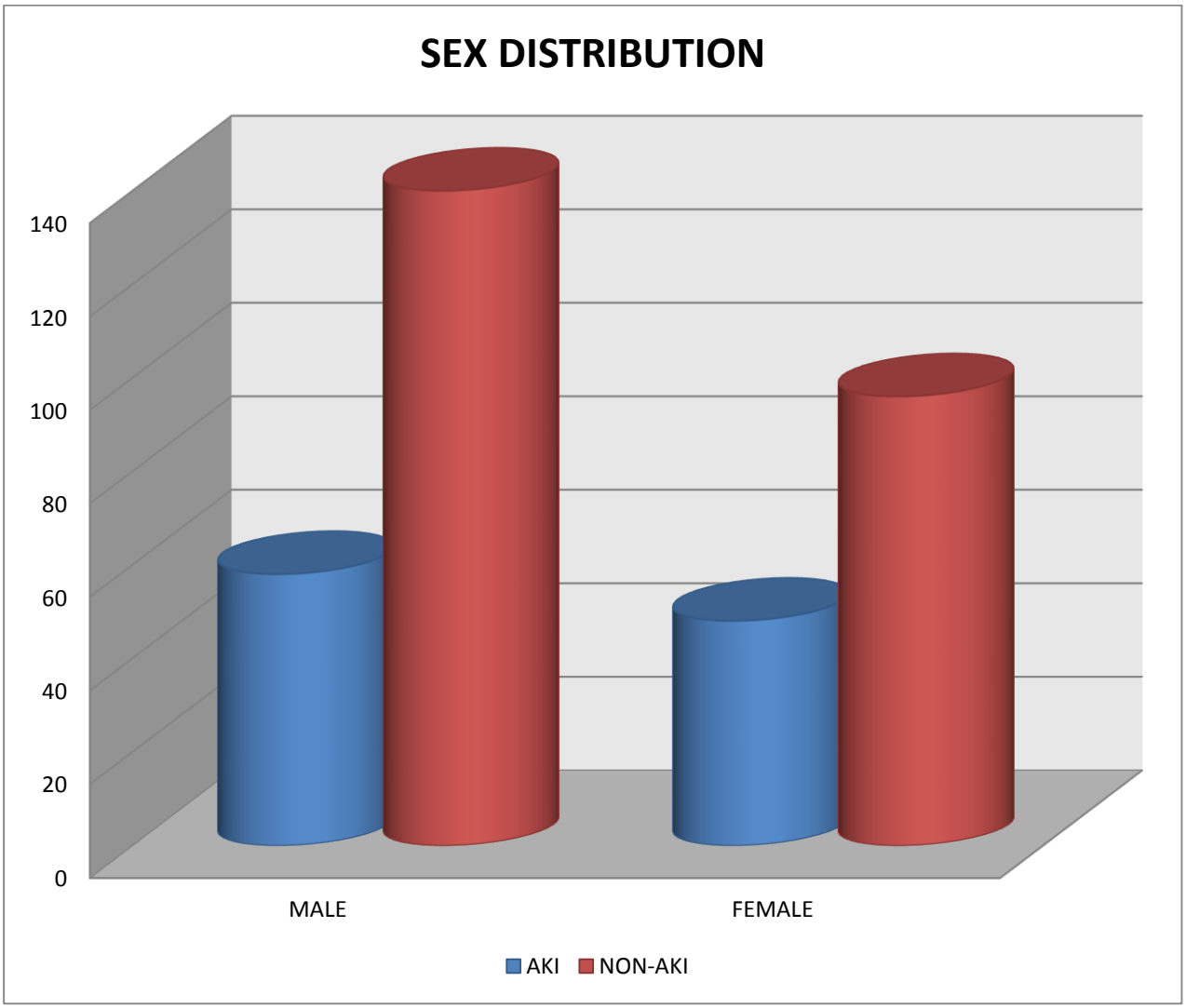


TABLE 5a: CASE DISTRIBUTION

By AKIN staging:

STAGING	CASES
STAGE 1	43 (40.6%)
STAGE 2	28 (26.4%)
STAGE 3	35 (33%)
TOTAL	106 (100%) (P Value <0.0001)

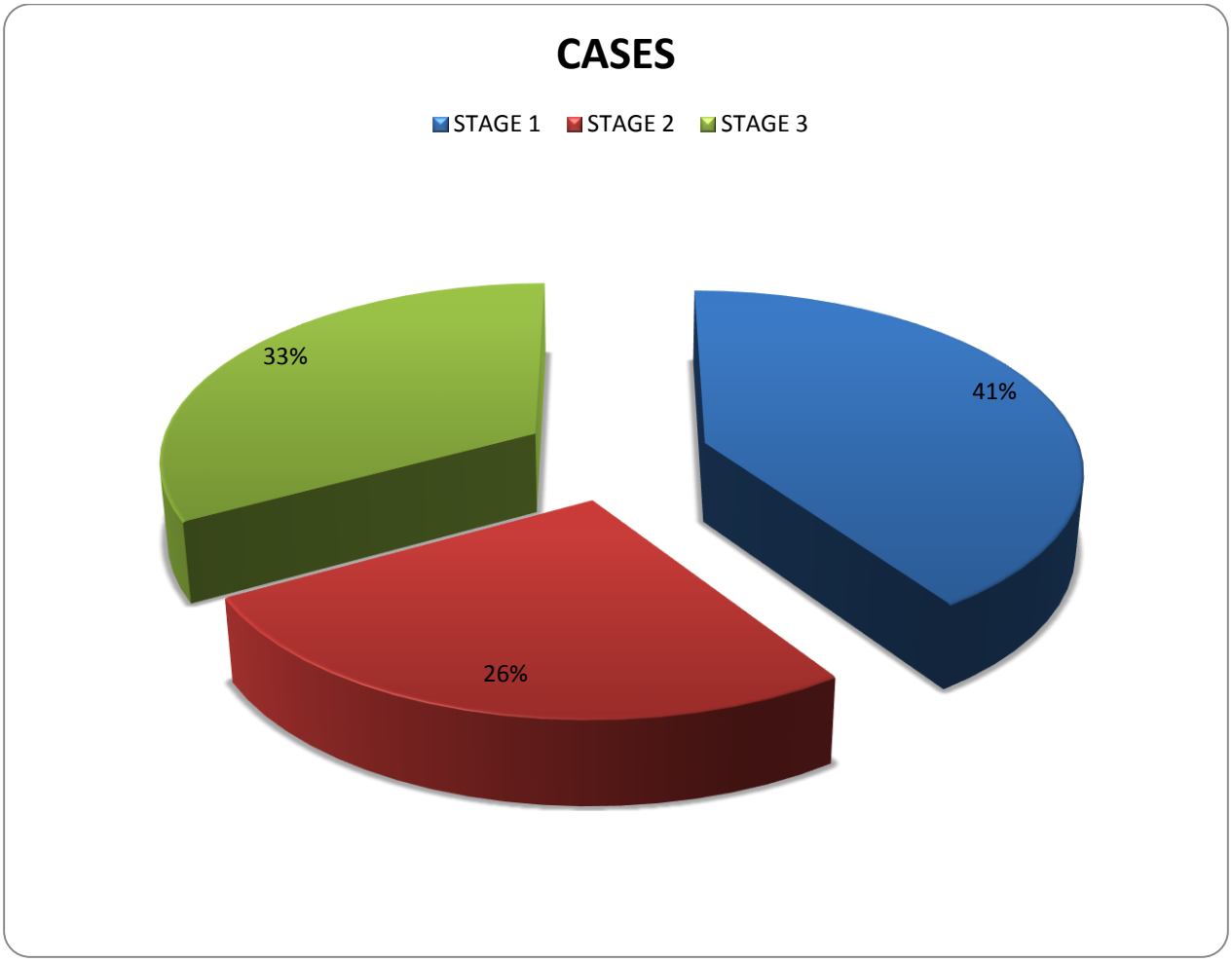
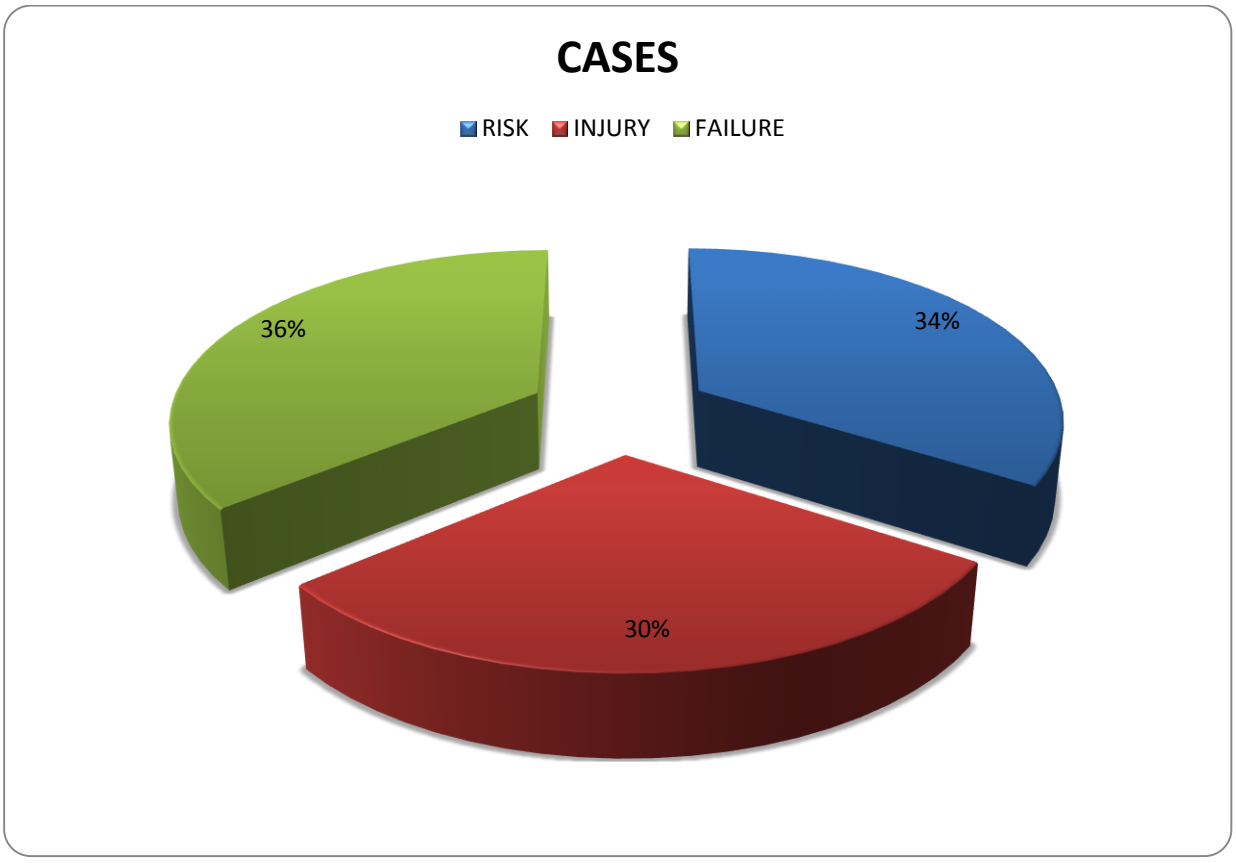


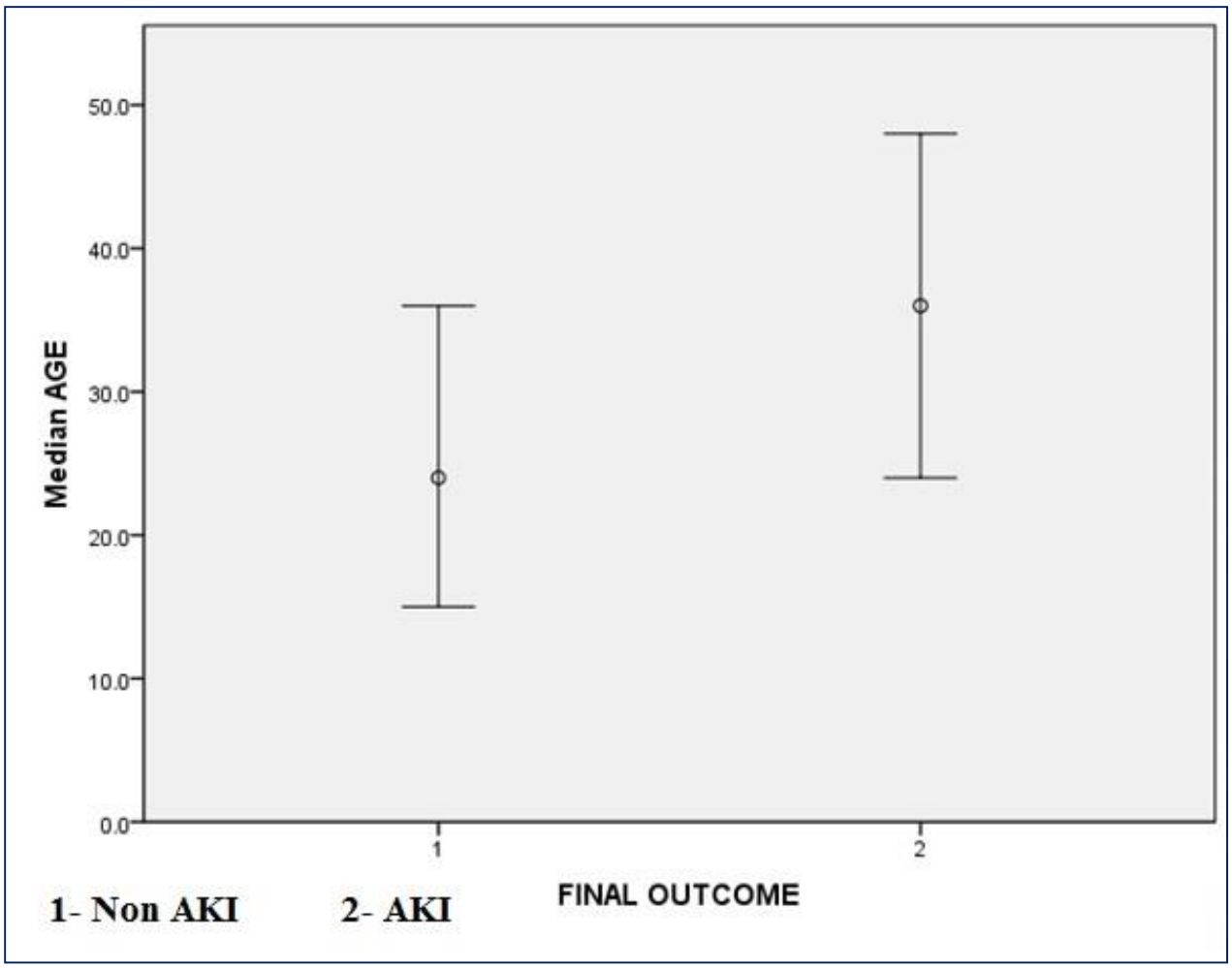
TABLE 5b: CASE DISTRIBUTION BY p-RIFLE CRITERIA

RIFLE CLASSIFICATION	CASES
RISK	35 (34%)
INJURY	31 (30.1%)
FAILURE	37 (35.9%)
TOTAL	103 (100%) (P Value <0.0001)



AGE DISTRIBUTION

Median age of the entire study population was 30 months (range 2 - 144 months). Median age of children with AKI was 36 months (range 2 - 144 months). Median age of children who succumbed with AKI was 24 months (range 2 – 144) and the median age of children who survived was 42 months (range 2- 144).



DURATION OF STAY

Mean duration of stay among the children who developed AKI (n=106) was 9.4 ± 4.5 days whereas the Mean duration of stay among the children who did not develop AKI was 5.6 ± 3.2 days. Mean duration of stay who survived with AKI was 11.1 ± 4.1 days while the mean duration of those who died was 7.1 ± 4.0 days.

SEVERITY OF ILLNESS SCORE

Severity of illness was assessed by PRISM III scores. The mean PRISM III score among the children who developed AKI was 26.4 ± 8.3 when compared with the mean PRISM III score among non-AKI children was 10.2 ± 6.5.

The mean level of maximum creatinine value during the hospital stay was 1.02 mg/dl (SD 1.2). The mean level of maximum creatinine value in children with AKI (n=106) during the hospital stay was 2.1 mg/dl (SD 1.7).

ETIOLOGICAL FACTORS OF ACUTE KIDNEY INJURY

The etiological factors of Acute Kidney Injury are listed in Table 6. Infections constitute 56.6% cases (60/106) of AKI. Sepsis was made as diagnosis in 18 cases of which 15 were culture positive and 3 were culture negative. Organisms isolated includes Coagulase Negative Staphylococcus (6 children), Staphylococcus aureus (3 children), Non fermentative gram negative bacillus (3 children), Klebsiella pneumonia (2 children), E. coli (1 child). Other common etiologies were Meningoencephalitis, Urinary Tract Infections (UTI), Congenital

heart diseases, Snake envenomation, Scorpion sting, Acute Glomerulonephritis, HUS d+, Nephrotic syndrome and surgical causes. Of the 7 cases of UTI, organisms isolated include profuse growth of E.coli (4 cases), profuse growth of Coagulase Negative Staphylococcus (2 cases), Non fermentative gram negative bacillus (1 case).

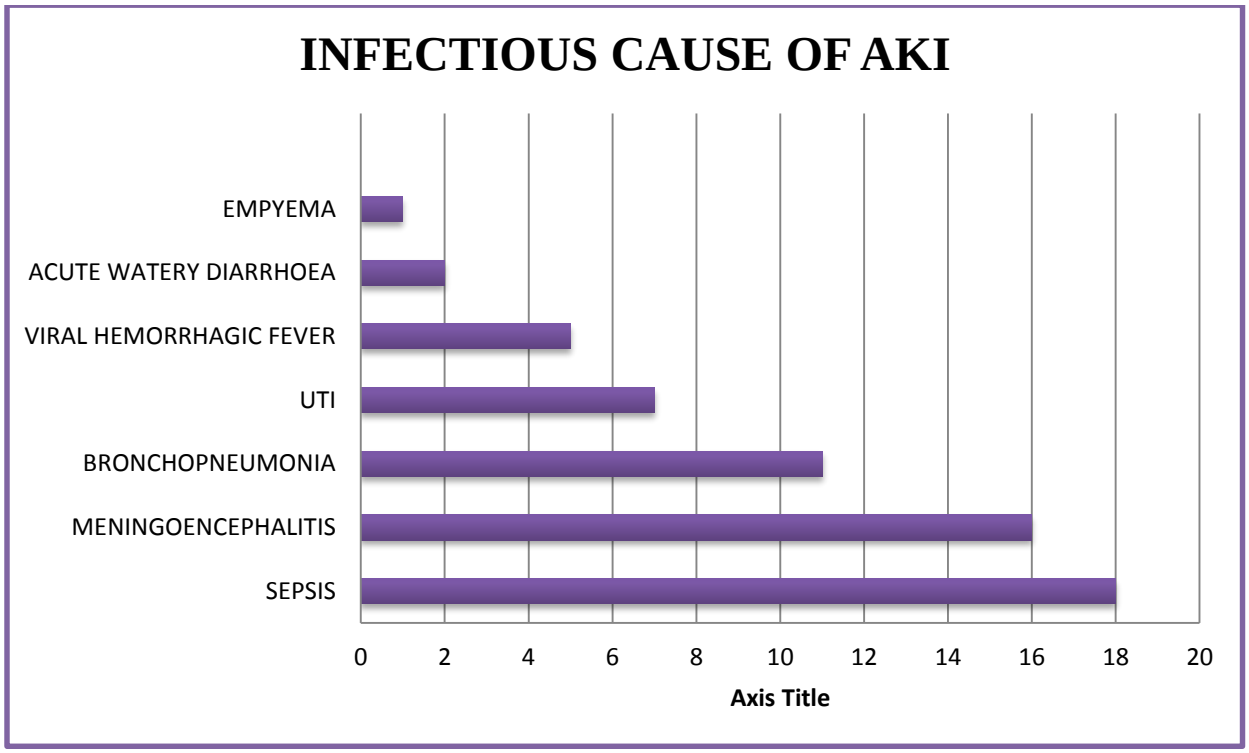
MORTALITY

Mortality rate in children with AKI (as described by AKIN stage) was found to be 42.5% in our study. 45 out of 106 expired during the study. All these 45 cases were identified as AKI by p-RIFLE criteria also and mortality rate according to p-RIFLE classification was 43.7% (45/103). Among the AKIN stage I cases, 15/43 (34.9%) died, in stage II cases, 11/28 (39.3%) died and in stage III cases, 19/35 (54.3%) died (differences were not statistically significant). Among p-RIFLE class, in Risk class 13/35 (37.1%) died, in Injury class 12/31 (38.7%) died and in Failure class 20/37 (54%) died.

Mortality were highest in the Bronchopneumonia and Meningoencephalitis group. 81.8% (9/11 cases) died among Bronchopneumonia and 68.6% (11/16 cases) died among Meningoencephalitis, 55.6% (10/18 cases) died among sepsis cases. No mortality among scorpion sting, nephrotic syndrome cases. The mortality in children < 10 months of age was found to be high as compared with age group of >10 months and this difference was statistically significant (p value 0.0406).

TABLE 6: ETIOLOGICAL FACTORS OF AKI CASES

ETIOLOGY	**N (%)**
Infections	**60 (56.6%)**
Cardiac causes (Congenital heart disease and Congestive Cardiac Failure)	9 (8.5%)
Snake envenomation	7 (6.6%)
Status Epilepticus (Seizure disorder, Febrile Seizures, Toxin induced)	5 (4.7%)
Surgical causes (PUJ obstruction, Hydroureteronephrosis, Hypoplastic kidney, Ewings sarcoma)	5 (4.7%)
Acute Glomerulonephritis	4 (3.8%)
Scorpion sting	4 (3.8%)
HUS d+	3 (2.8%)
Nephrotic syndrome	3 (2.8%)
Poisoning (Organophosphorus, Abrus precatorius, Native Medication)	3 (2.8%)
Diabetic Ketoacidosis	2 (1.9%)
Acute severe asthma	1 (0.9%)
AMONG INFECTIONS (N=60)	
Sepsis • Culture positive • Culture negative	18 (30%) 15 (83.3%) 3 (16.7%)
Meningoencephalitis	16 (26.7%)
Bronchopneumonia	11 (18.3%)
Urinary Tract Infection	7 (11.7%)
Viral hemorrhagic fever	5 (8.3%)
Acute watery diarrhea	2 (3.3%)
Empyema thorax	1 (1.7%)


INFECTIOUS CAUSE OF AKI
EMPYEMA
ACUTE WATERY DIARRHOEA
VIRAL HEMORRHAGIC FEVER
UTI
BRONCHOPNEUMONIA
MENINGOENCEPHALITIS
SEPSIS
0
2
4
6
8
10
12
14
16
18
20
Axis Title

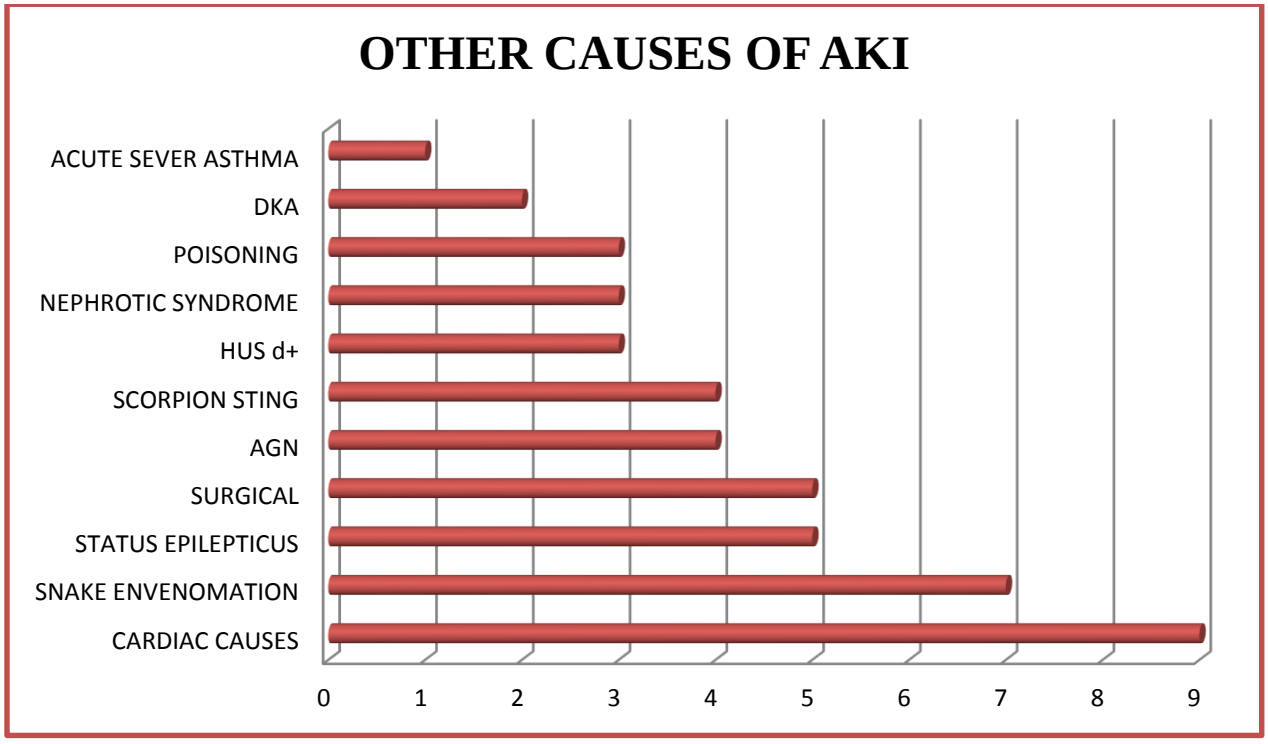

OTHER CAUSES OF AKI
ACUTE SEVER ASTHMA
DKA
POISONING
NEPHROTIC SYNDROME
HUS d+
SCORPION STING
AGN
SURGICAL
STATUS EPILEPTICUS
SNAKE ENVENOMATION
CARDIAC CAUSES
0
1
2
3
4
5
6
7
8
9

TABLE 7a: MORTALITY

AKIN STAGE	SURVIVORS	DEATH	TOTAL
STAGE 1	28 (65.1%)	15 (34.9%)	43 (100%)
STAGE 2	17 (60.7%)	11 (39.3%)	28 (100%)
STAGE3	16 (45.7%)	19 (54.3%)	35 (100%)
TOTAL	61 (57.5%)	45 (42.5%)	106 (100%)

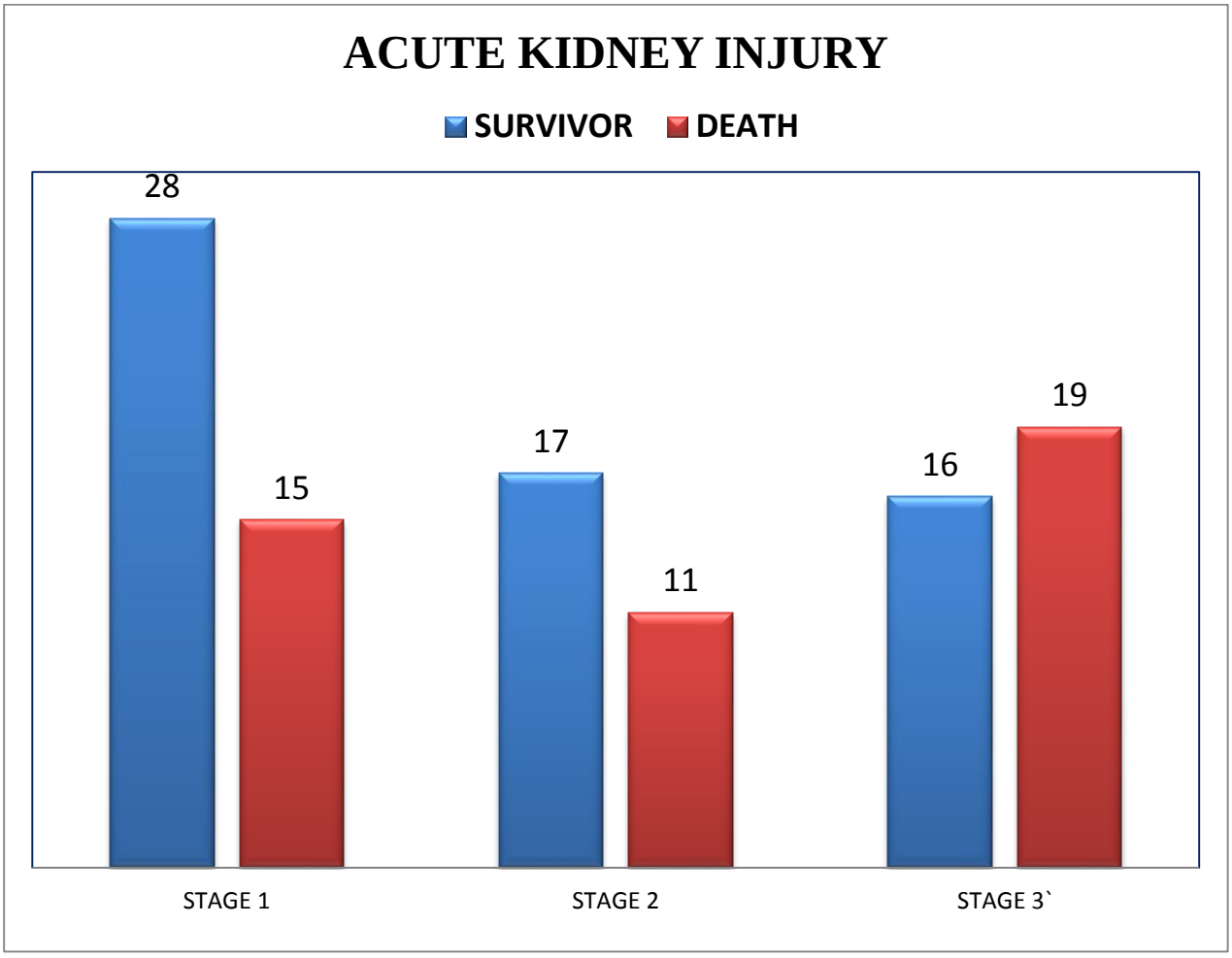
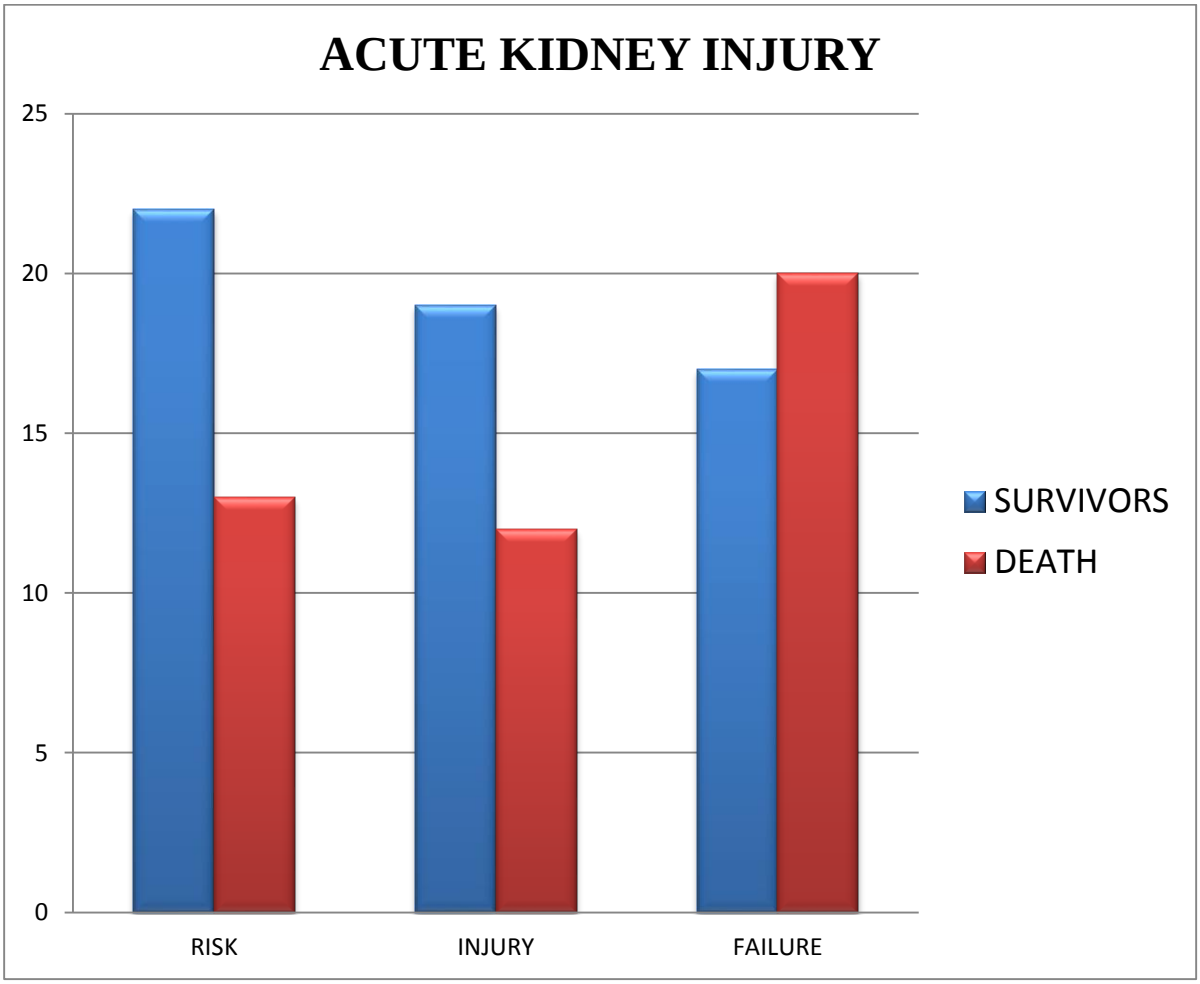


TABLE 7b: MORTALITY

RIFLE CLASS	SURVIVORS	DEATH	TOTAL
RISK	22 (62.9%)	13 (37.1%)	35 (100%)
INJURY	19 (61.3%)	12 (38.7%)	31 (100%)
FAILURE	17 (46%)	20 (54%)	37 (100%)
TOTAL	58 (56.3%)	45 (43.7%)	103 (100%)

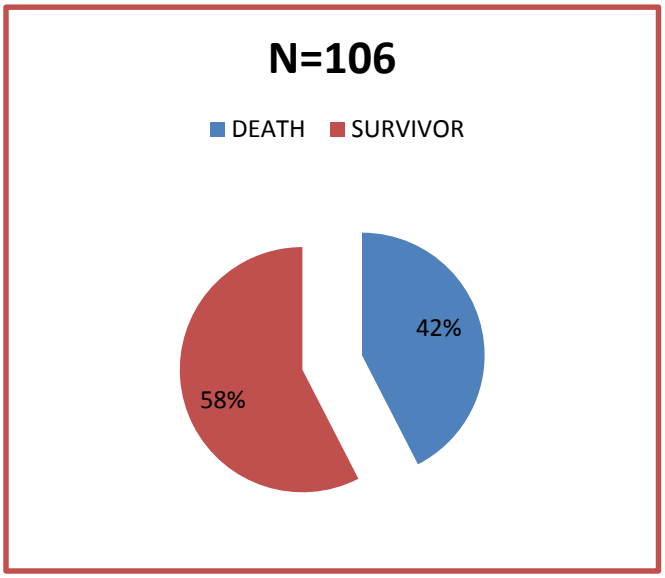


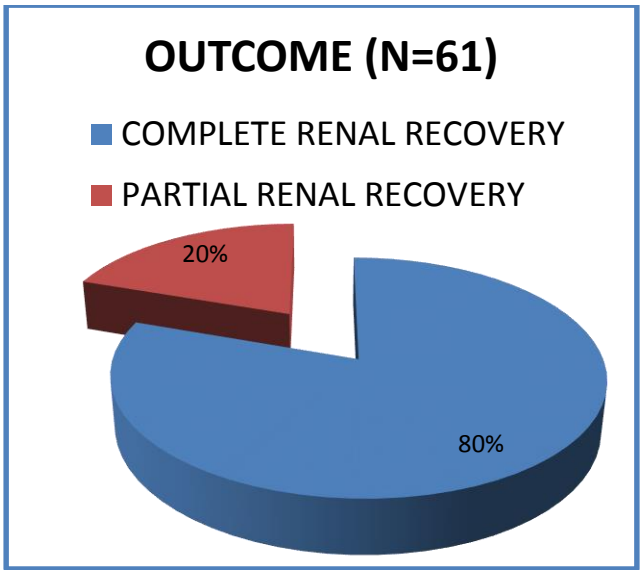
SHORT TERM OUTCOME

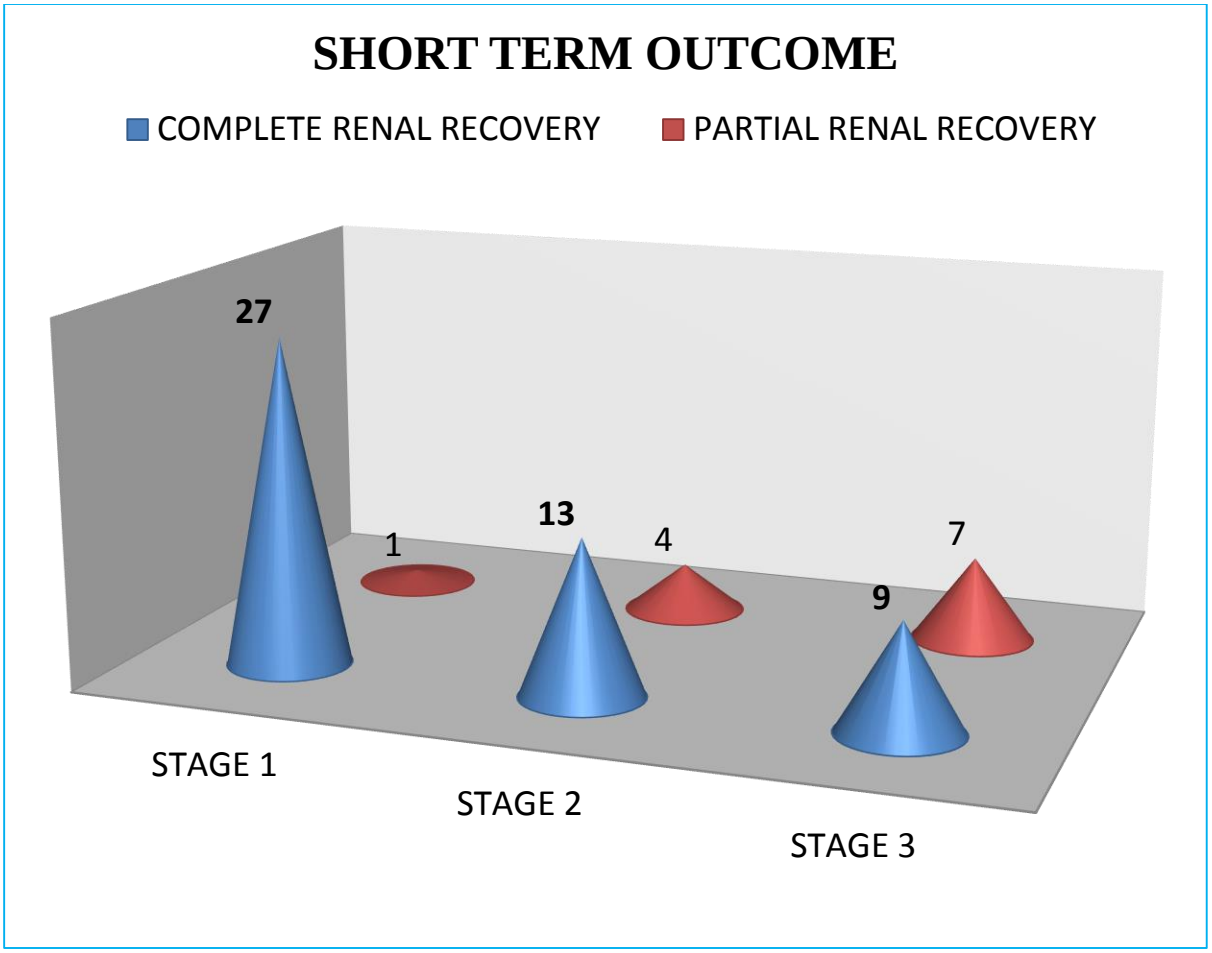
A total of 49 children of the survivors with AKI (80.3%) had complete renal recovery while 12 children (19.7%) of the survivors had partial renal recovery. In AKI stage 1, out of the survivors, 27 (96.4%) had complete renal recovery while 1 (3.6%) had partial renal recovery at discharge. In AKI stage 2, 13 (76.5%) had complete renal recovery while 4 (23.5%) had partial renal recovery at discharge. In AKI stage 3, 9 (56.3%) had complete renal recovery, while 7 (43.7%) had partial renal recovery at discharge.

TABLE 8: SHORT TERM OUTCOME

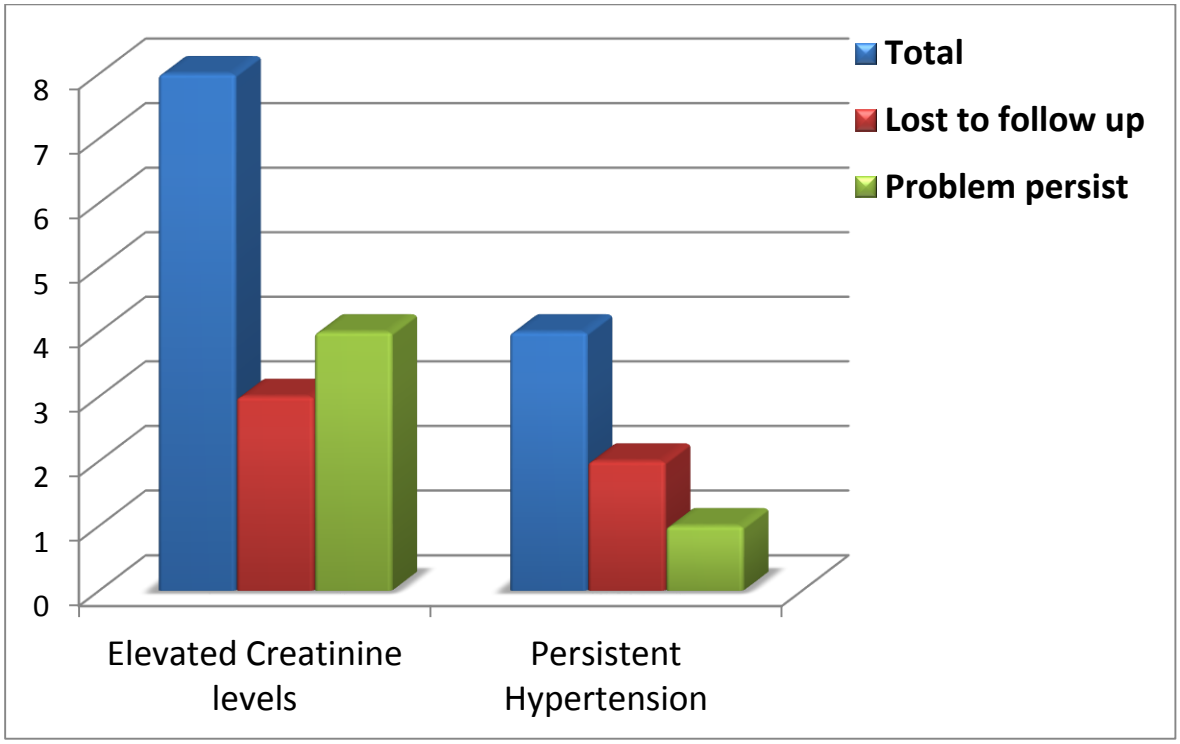
	STAGE 1	**STAGE 2**	**STAGE 3**	**TOTAL**
COMPLETE RENAL RECOVERY	27 (96.4%)	13 (76.5%)	9 (56.3%)	49 (80.3%)
PARTIAL RENAL RECOVERY	1 (3.6%)	4 (23.5%)	7 (43.7%)	12 (19.7%)
TOTAL	28 (45.9%)	17 (27.9%)	16 (26.2%)	61 (100%)


N=106
DEATH
SURVIVOR
42%
58%


OUTCOME (N=61)
COMPLETE RENAL RECOVERY
PARTIAL RENAL RECOVERY
20%
80%


SHORT TERM OUTCOME
COMPLETE RENAL RECOVERY
PARTIAL RENAL RECOVERY
27
1
13
4
9
7
STAGE 1
STAGE 2
STAGE 3

- 12 children who had partial renal recovery at the time of discharge were followed up over a period.
- Among the 12, 4 had hypertension and 8 had elevated creatinine levels.
- 4 children with elevated creatinine level were referred to higher centre for hemodialysis. And 3 of these cases were lost to follow up. 1 patient died after 3 days.
- 4 patients still have elevated creatinine values above 50% baseline. (Followed up for 1 month after discharge).
- 1 child with hypertension became normotensive – complete recovery over a mean period of 2 months, 1 child had hypertension at 2 months of follow up. (the other 2 children were lost to follow up).



RENAL REPLACEMENT THERAPY

A total of 28 children (26.4%) required dialysis in the form of peritoneal dialysis. The mortality among children requiring RRT was similar to children not requiring RRT (42.9% vs. 42.3%) and the difference was not significant statistically. Requirement of RRT was not related to age or the etiology of AKI.

TABLE 9: AKI OUTCOME * RRT Cross tabulation

		RRT		
		NO	YES	Total
AKI OUTCOME	1	45 (73.8%)	16 (26.2%)	61 (100%)
	2	33 (73.3%)	12 (26.7%)	45 (100%)
Total		78 (73.6%)	28 (26.4%)	106 (100%)

1- SURVIVORS 2 – DEATH

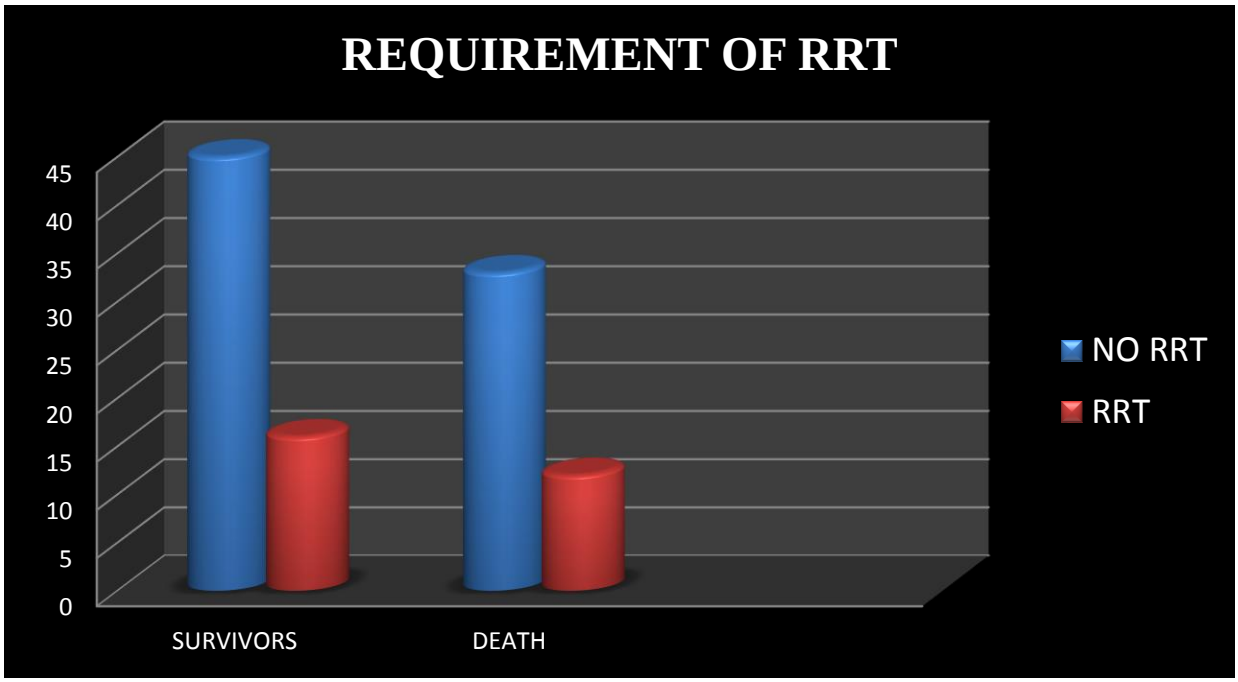


TABLE 11: Demographic parameters of critically ill children with AKI

PARAMETER	Baseline characteristics (n=106)
Age (months) [median(range)]	36 (2 – 144)
Sex	Male – 58 (54.7%) Female – 48 (45.3%)
PRISM III score (mean ± SD)	26.4 ± 8.3
Duration of stay (days) (mean ± SD) Survivors Non-survivors Overall	 11.1 ± 4.1 7.1 ± 4.0 9.4 ± 4.5
Mortality [N (%)]	45 (42.5%)
Mechanical Ventilation [N (%)]	59 (55.7%)
Shock [N (%)]	77 (72.6%)
Encephalopathy [N (%)]	33 (31.1%)
Renal replacement therapy [N (%)]	28 (26.4%)

LABORATORY DATA

TABLE 10: Laboratory characteristics of children with AKI.

VARIABLE	MEAN	STANDARD DEVIATION
UREA	64.2 mg/dl	50.1
CREATININE	2.1 mg/dl	1.7
SODIUM	135.3 meq/L	9.6
POTASSIUM	4.1 meq/L	0.97
HEMOGLOBIN	10.6 gm/dl	2.6
PLATELET COUNT	2.9 lakh cells/cu.mm	1.7

Hyponatremia (57.5%), Hypernatremia (16%), hypokalemia (22.6%), hyperkalemia (18.9%), anemia (55.7%), thrombocytopenia (23.6%), hypertension (15.1%) and metabolic acidosis (57.5%) were the associated complications and co-morbidities found in children with acute kidney injury in our study.

A total of 59 children (55.7%) out of 106 AKI children needed mechanical ventilation and 77 children (72.6%) had shock as co-morbidity.

TABLE 11: AKI OUTCOME * SODIUM LEVEL

	SODIUM LEVEL			
	NORMAL	HYPONATREMIA	HYPERNATREMIA	Total
AKI OUTCOME 1	17	36	8	61
2	11	25	9	45
Total	28 (26.5%)	61(57.5%)	17 (16%)	106

1- SURVIVORS 2 – DEATH

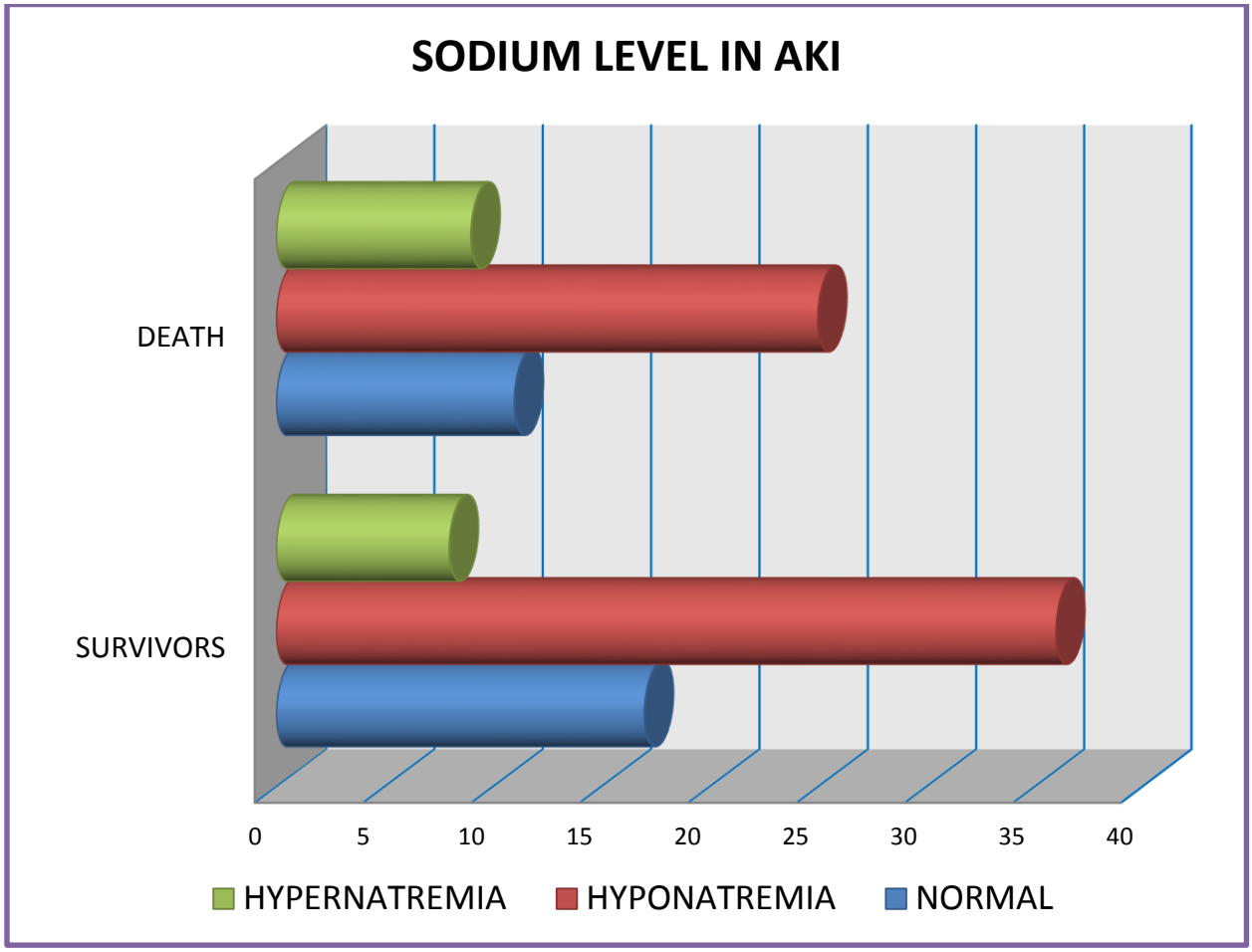


TABLE 12: AKI OUTCOME * POTASSIUM LEVEL

		POTASSIUM LEVEL			Total
		NORMAL	HYPOKALEMIA	HYPERKALEMIA	
AKI OUTCOME	**1**	36	14	11	61
	2	26	10	9	45
Total		62 (58.5%)	24 (22.6%)	20 (18.9%)	106

1- SURVIVORS 2 - DEATH

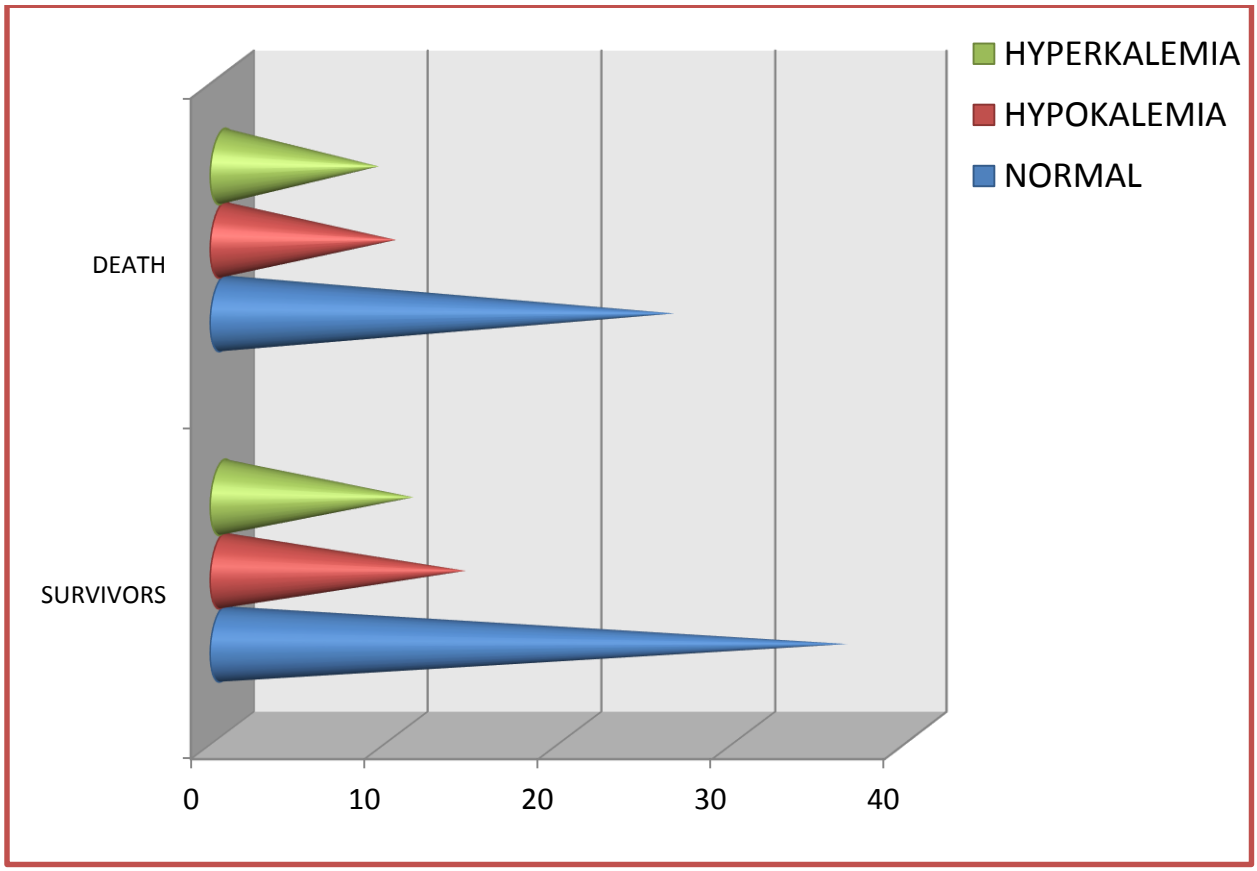


TABLE 13: AKI OUTCOME * ANEMIA

		Anemia		
		NO	**YES**	**Total**
AKI OUTCOME	**1**	30	31	61
	2	17	28	45
Total		47 (44.3%)	59 (55.7%)	106

1- SURVIVOR 2 – DEATH

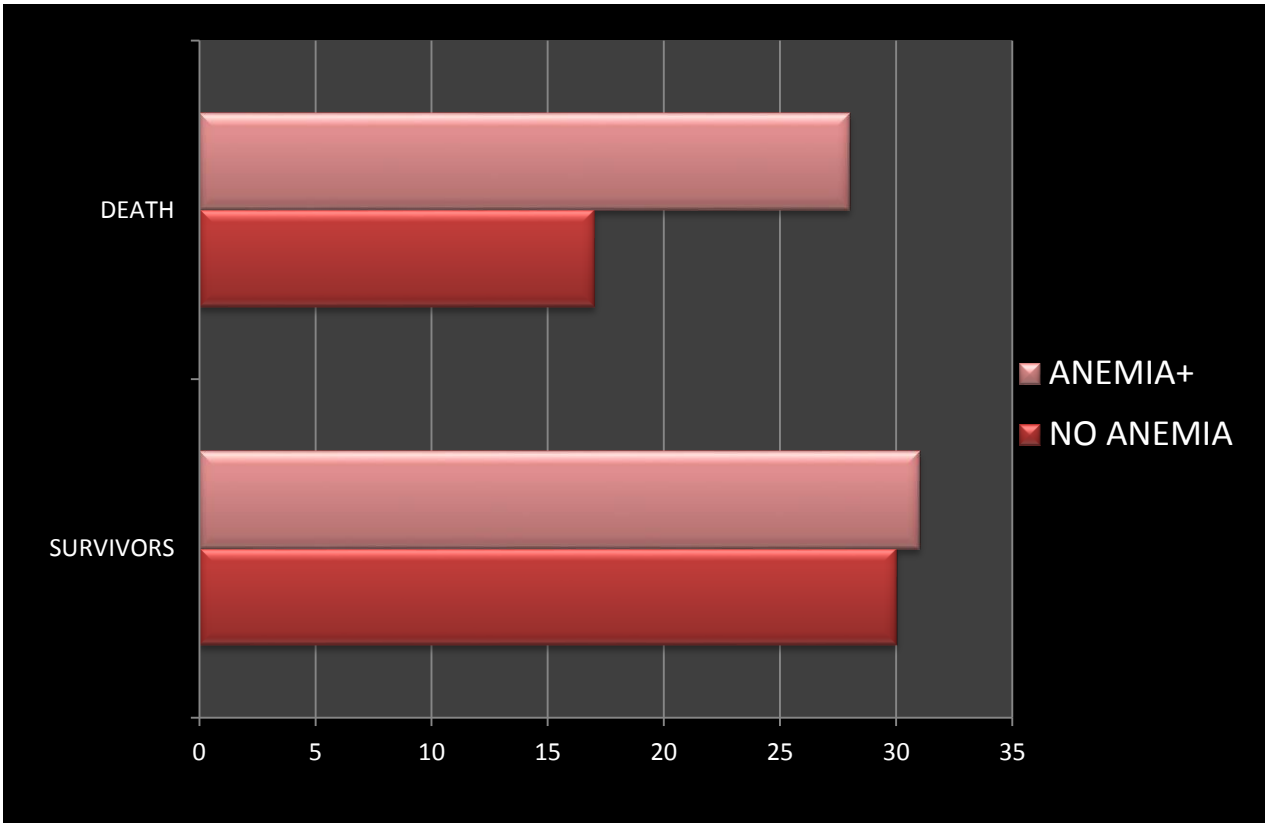


TABLE 14: AKI OUTCOME * THROMBOCYTOPENIA

		THROMBOCYTOPENIA		
		NO	YES	Total
AKI OUTCOME	1	46	15	61
	2	35	10	45
Total		81 (76.4%)	25 (23.6%)	106

1- SURVIVORS 2 – DEATH

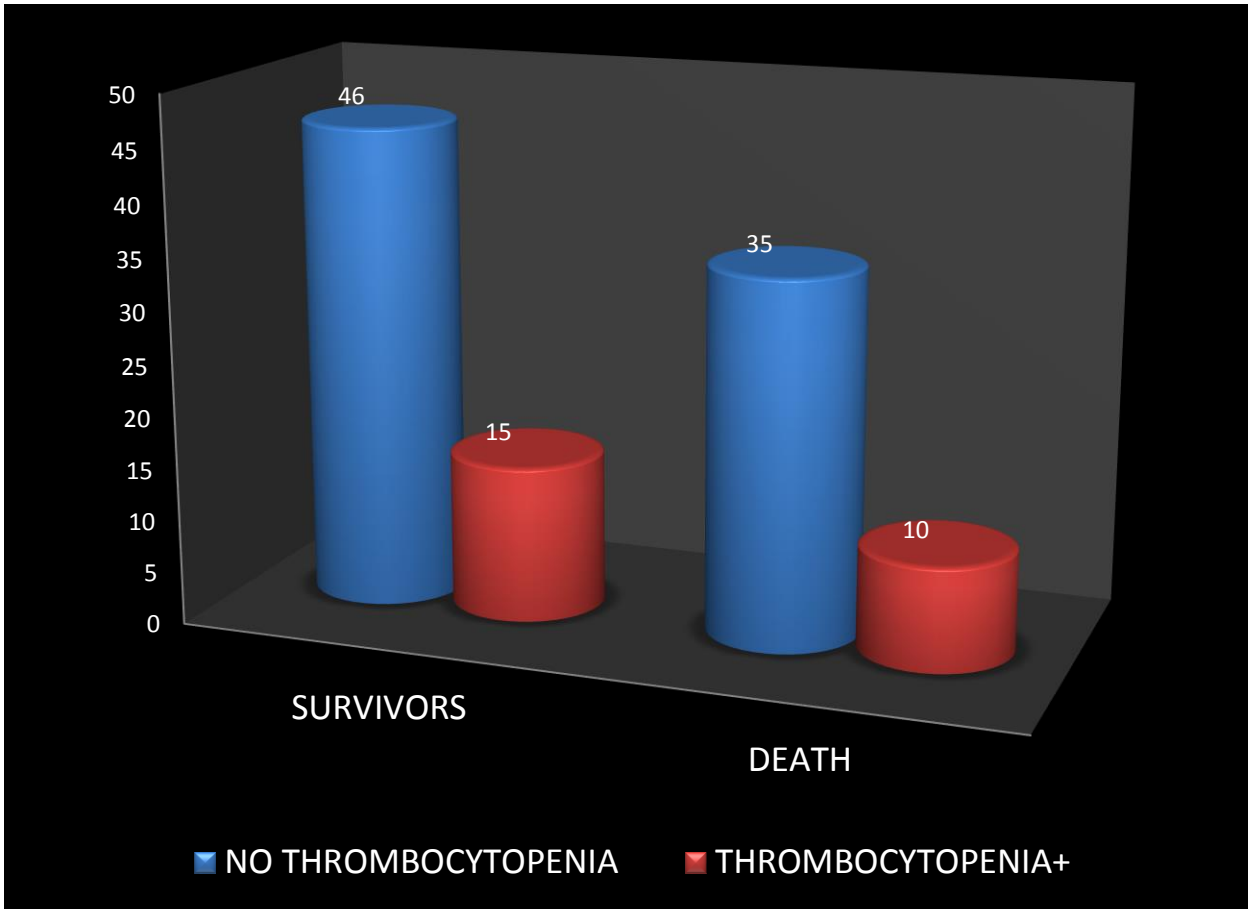


TABLE 15: AKI OUTCOME * HYPERTENSION

		Hypertension		
		NO	YES	Total
AKI OUTCOME	1	49	12	61
	2	41	4	45
Total		90 (84.9%)	16 (15.1%)	106

1- SURVIVOR 2 - DEATH

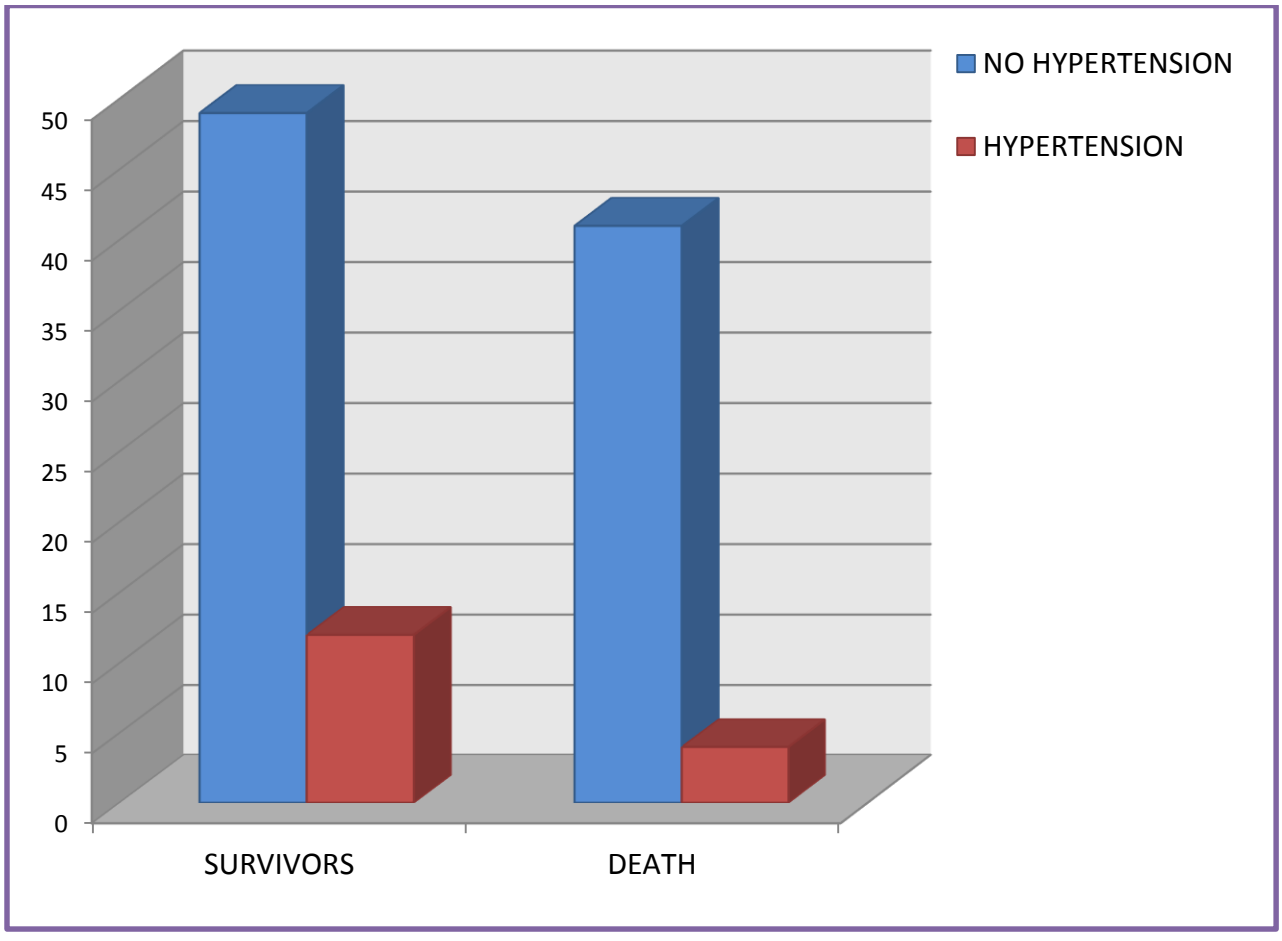


TABLE 16: AKI OUTCOME * ABG ANALYSIS

		ABG ANALYSIS			
		Not done	Normal	Acidosis	Total
AKI OUTCOME	1	4	29	28	61
	2	1	11	33	45
Total		5 (4.7%)	40 (37.8%)	61 (57.5%)	106

1- SURVIVORS 2 - DEATH

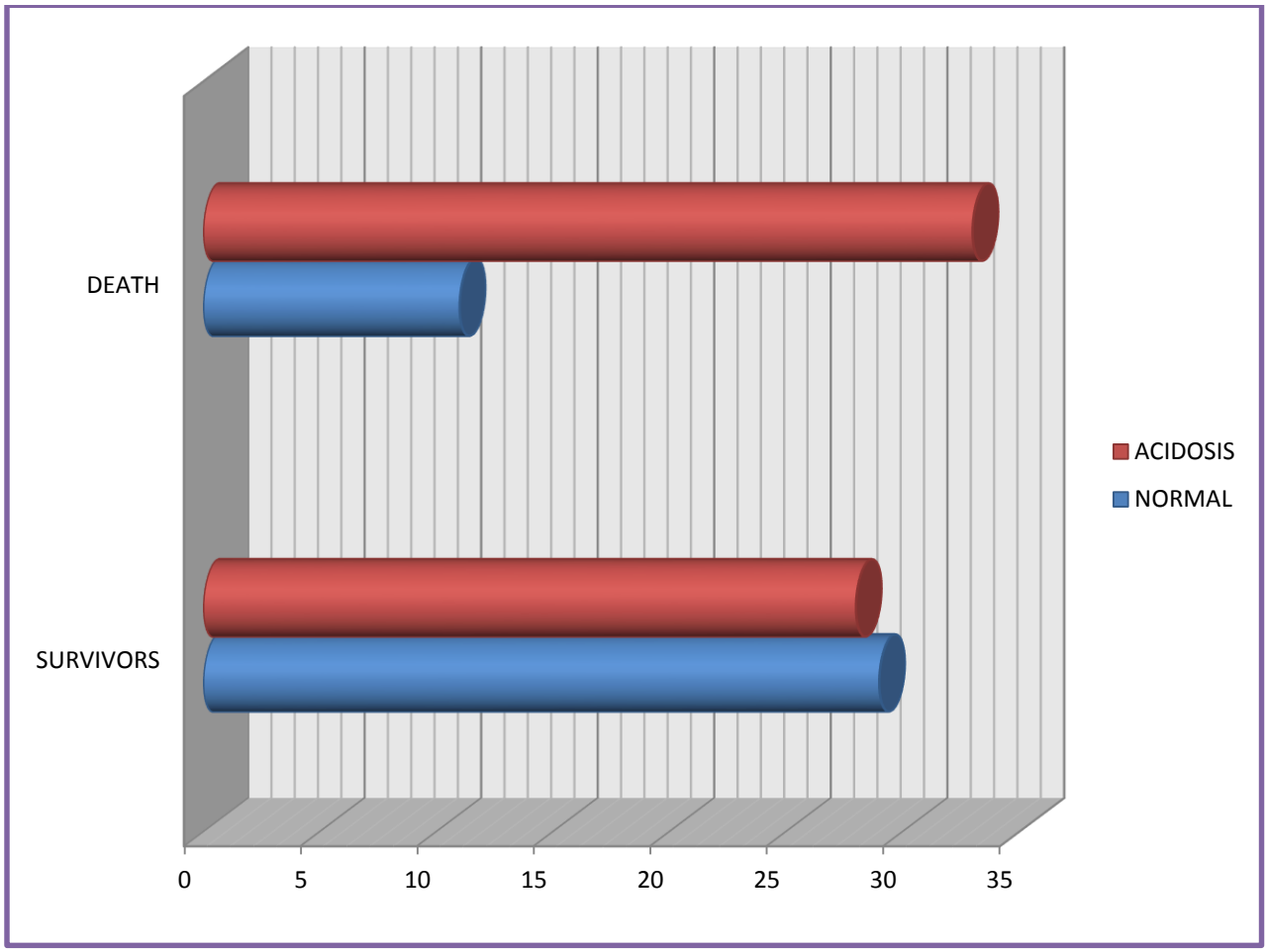


Table 17:AKI OUTCOME * MECHANICAL VENTILATION

		MECHANICAL VENTILATION		
		No	Yes	Total
AKI OUTCOME	1	42	19	61
	2	5	40	45
Total		47 (44.3%)	59 (55.7%)	106

1- SURVIVOR 2- DEATH

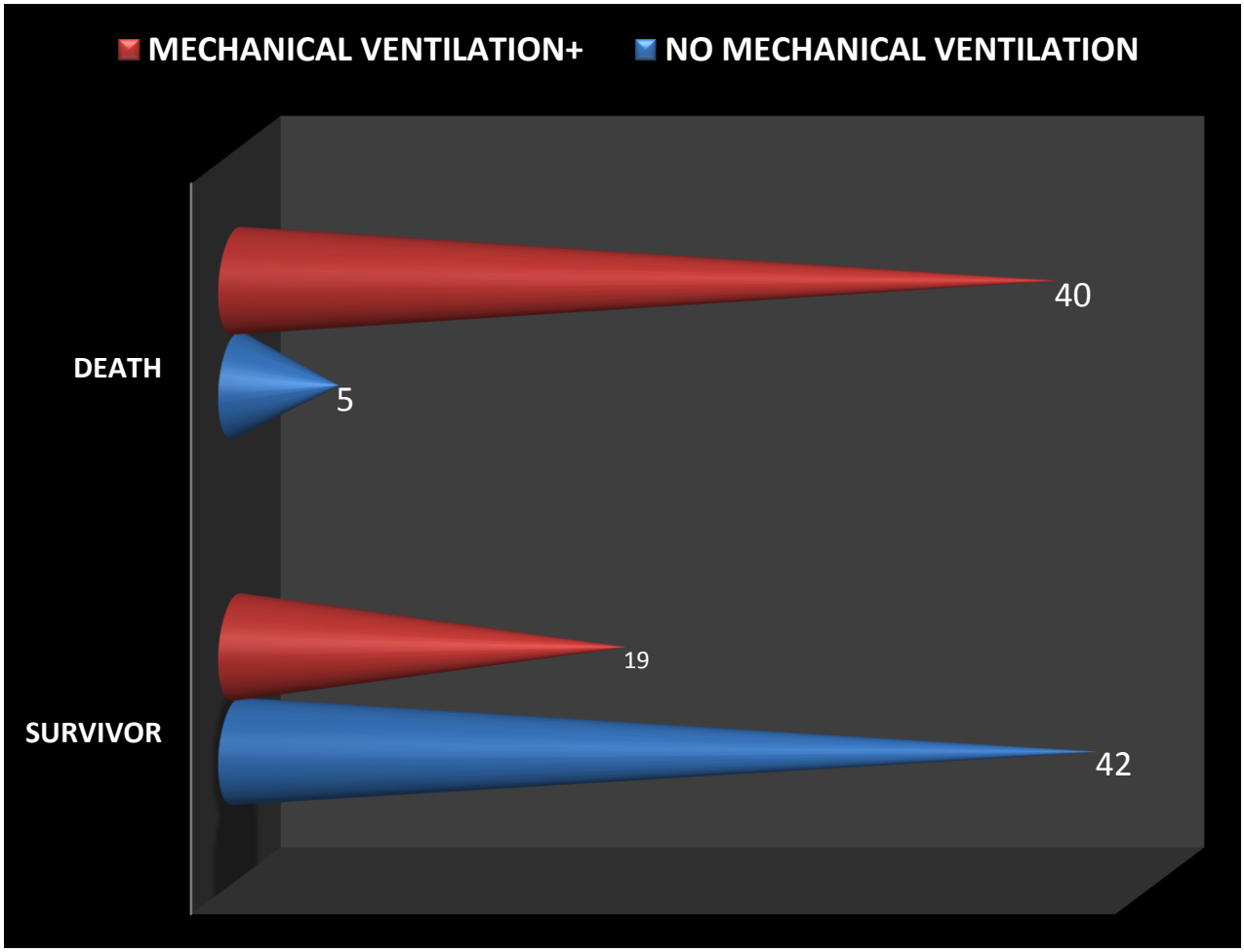


TABLE 18: AKI OUTCOME * SHOCK

		INOTROPE USE		TOTAL
		NO	YES	
AKI OUTCOME	1	26	35	61
	2	3	42	45
TOTAL		29 (27.4%)	77 (72.6%)	106

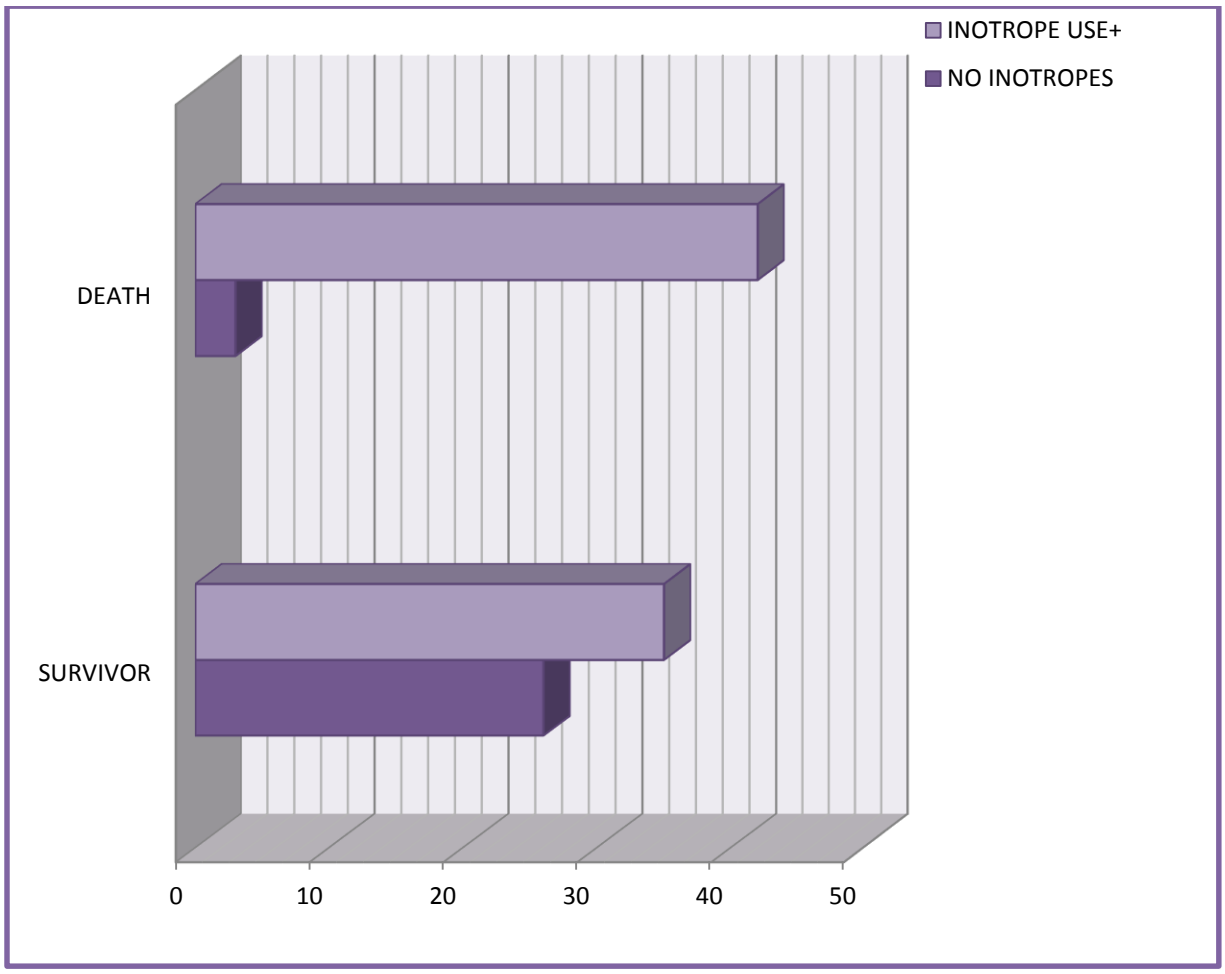


TABLE 19

Comparison of survivors and deaths in critically ill children with AKI (n=106)

Parameter	**Survivors (n=61)**	**Deaths (n=45)**	**P value**
Age (months) [median(range)]	42 (2-144)	24 (2-144)	0.02
Sex [N (%)]	Male – 31 Female – 30	Male -27 Female - 18	0.348
Duration of stay (days) (mean±SD)	11.1 ± 4.1	7.1 ± 4.0	0.001
PRISM III score (mean±SD)	21.05 ± 5.77	33.56 ± 5.04	0.0001
Mechanical ventilation [N (%)]	40 (88.9%)	19 (31.1%)	0.0001
Shock [N (%)]	35 (57.4%)	42 (93.3%)	0.0001
Renal Replacement Therapy [N (%)]	16 (26.2%)	12 (26.7%)	0.960
Maximum creatinine value (mean±SD)	1.9 ± 1.4	2.4 ± 2.1	-
Morbidities [N (%)]			
Bronchopneumonia	2 (18.2%)	9 (81.8%)	
Meningoencephalitis	5 (31.3%)	11 (68.7%)	
Sepsis	8 (44.4%)	10 (55.6%)	
AGN	3 (75%)	1 (25%)	
Snake envenomation	6 (85.7%)	1 (14.3%)	
Scorpion sting	4 (100%)	0	
Nephrotic syndrome	3 (100%)	0	

TABLE 20

Predictors of mortality in Acute Kidney Injury (Univariate analysis)

PARAMETER	DEATH	SURVI-VORS	ODDS RATIO	95% CI	P value
Age					
<10 months	**16**	**11**	**2.51**	**0.94-6.75**	**0.0406#**
>10 months	**29**	**50**			
Gender					
Male	**27**	**31**	**1.45**	**0.68-3.41**	**0.347**
Female	**18**	**30**			
Stage of AKI					
Stage 1	**15**	**28**	**0.59**	**0.24-1.41**	**0.192**
Stage 2	**11**	**17**	**0.84**	**0.32-2.20**	**0.692**
Stage 3	**19**	**16**	**2.06**	**0.84-5.08**	**0.083**
Oliguria	**19**	**21**	**1.39**	**0.58-3.32**	**0.413**
Metabolic acidosis	**33**	**28**	**3.24**	**1.31-8.13**	**0.004#**
Hyponatremia	**25**	**36**	**0.87**	**0.37-2.03**	**0.721**
Hypernatremia	**9**	**8**	**1.66**	**0.52-5.29**	**0.339**
Hyperkalemia	**9**	**11**	**1.14**	**0.38-3.35**	**0.798**
Hypertension	**4**	**12**	**0.40**	**0.10-1.48**	**0.125**
Mechanical ventilation	**40**	**19**	**17.68**	**5.50-60.79**	**0.0001#**
Anemia	**28**	**31**	**1.59**	**0.68-3.77**	**0.242**
Thrombocytopenia	**10**	**15**	**0.88**	**0.32-2.39**	**0.776**
Requirement of RRT	**12**	**16**	**1.02**	**0.39-2.67**	**0.959**
Shock	**42**	**35**	**10.4**	**2.9-37.3**	**0.0001#**

#P value significant, RRT: Renal Replacement Therapy, CI: Confidence interval, AKI: Acute Kidney Injury

The predictors of mortality on univariate analysis were: Age less than 10 months, shock and requirement of mechanical ventilation, presence of metabolic acidosis as shown in table. In the multivariate model, requirement of mechanical ventilation was found to be an independent predictor of mortality. (odds ratio: 15.011; 95% Confidence Interval 3.086-73.008; P value 0.001)

RENAL ULTRASOUND

Ultrasound findings of children with AKI: 40 children (37.7%) had abnormal echo pattern (increased cortical echoes), 4 children (3.7%) had B/L enlarged kidneys. 4 children (3.7%) had anatomical abnormality in the form of pelvicalyceal dilatation, hydroureteronephrosis, PUJ obstruction.

DISCUSSION

Our study was a single-centre study with an objective to determine the incidence and clinical profile of AKI using both AKIN AND p-RIFLE classification. Knowledge on incidence, clinical profile and outcome of AKI is vital for initiation of appropriate therapeutic management and also to compare epidemiological studies for improved clinical decision making.

Study	**Incidence by AKIN staging**
Present study	**31%**
Sriram Krishnamurthy et al[19]	25.1%
Mehta et al[20]	36.1%
Srinivasa et al[38]	35.5%

From our observational study, the incidence of Acute Kidney Injury in critically ill children admitted to PICU in our institute was found to be 31% using AKIN staging. The incidence in this study was comparable with a study done by Sriram krishnamurty et al at JIPMER[19], where the incidence was reported to be 25.1% in PICU and by Mehta P et al at AIIMS[20], where the incidence was 36.1%. This study used serum creatinine and urine output that has been used in several similar studies in children.

The incidence of AKI in critically ill children admitted to PICU of our institute using p-RIFLE classification was found to be 30.1%. This was

Study	Incidence by p-RIFLE classification
Present study	**30.1%**
Srinivasa et al[38]	26.1%
Shweta naik et al[113]	40.9%
Gil-Ruiz et al, 2014	20%

comparable with the incidence of a study done by Srinivasa S et al at KIMS, Bangalore using p-RIFLE classification which showed an incidence of 26.1%[38]. Another study by Shweta naik et al[113] using p-RIFLE classification showed an incidence of 40.9%. This study used serum creatinine and estimated creatinine clearance / glomerular filtration rate using Schwartz formula that has been used in several studies in children. This study however reports a slightly lower incidence of AKI using p-RIFLE classification similar to a study by Akobye Winnie et al done at mulago because of the use of glomerular filtration rate, which takes into account a child's age, height and weight. This study has the benefit of having calculated the glomerular filtration rate, a better index of kidney function than serum creatinine. However, there occurs an over-estimation of the GFR, another measure like urine output is recommended to improve the sensitivity of the pRIFLE criteria. When using both, either the urine output criteria or the creatinine criteria that shows the worst possible outcome should be considered. Thus,

pRIFLE is useful in determining severity, and thus predicting the mortality, and for monitoring the progress of AKI[112].

SEVERITY OF ACUTE KIDNEY INJURY

By AKIN staging

STAGING	PRESENT STUDY	Sriram krishnamurthy et al[19]	Srinivasa et al[38]	Mehta et al[20]
STAGE 1	43 (40.6%)	19 (35.2%)	93 (37.5%)	48 (65.8%)
STAGE 2	28 (26.4%)	14 (25.9%)	88 (35.5%)	13 (17.8%)
STAGE 3	35 (33%)	21 (38.9%)	67 (27%)	12 (16.4%)
TOTAL	106 (100%)	54 (100%)	248 (100%)	73 (100%)

From the above table, it is evident that our study is comparable with similar studies conducted in Indian children. Assessing the severity of AKI is useful in predicting mortality rates and the need for renal replacement therapy. Our study includes maximum cases in stage 1 and the results are similar to other studies.

By p-RIFLE classification

The following table compares the classification of AKI cases according to p-RIFLE criteria in our study and similar kind of studies conducted elsewhere.

CLASSIFICATION	PRESENT STUDY	Srinivsa et al[38]	Shwet naik et al[113]
RISK	35 (34%)	108 (60.7%)	39 (37.9%)
INJURY	31 (30.1%)	51 (28.7%)	37 (35.9%)
FAILURE	37 (35.9%)	19 (10.6%)	27 (26.2%)
TOTAL	103 (100%)	178 (100%)	103 (100%)

In our study, maximum RIFLE score was achieved in almost all children within 72 hours of admission to PICU which was again comparable to study by shweta naik et al. Schneider et al reported that almost 50% patients developed their maximum RIFLE score within 24 hours of admission and about 75% achieved it by 7th day of PICU stay. Due to the decreasing sensitivity and increasing specificity as one moves through the categories from Risk, to Injury, to Failure[112], it is possible that many more patients were categorised as pRIFLE-risk, while some were missed as we consider the pRIFLE-Failure category.

CLINICAL CHARACTERISTICS AND RISK FACTORS

The median age group of children with Acute Kidney Injury in our study was found to be 36 months (IQR 2-144). The median age of survivors with AKI was 42 months (IQR 2-144) and the median age of non survivors was 24 months (IQR 2-144). In a study by Sriram Krishnamurthy et al[19], the median age of survivors was 30 months and the median age of non survivors was 10 months.

Similar results was given by Mehta et al[20] and Shweta naik et al[113]. The mortality in children < 10 months of age was found to be high as compared with age group of >10 months and this difference was statistically significant (p value 0.0406) and this was also comparable with study done by Sriram Krishnamurthy et al[19]. Thus it is evident that, younger age is an independent risk factor for Acute kidney injury.

. Of the 106 children who developed AKI, 58 (54.7%) were male and 48 (45.3%) were female. In the study by Sriram Krishnamurthy et al[19], among the AKI population, 53.7% were males and 46.3% were females. In a study by Shweta naik et al[113], males were of 62.1% and females were 37.9% among the AKI population.

The mean PRISM III score of children with AKI was found to be 26.4 ± 8.3 and in children without AKI it was 10.2 ± 6.5 in our study. The mean PRISM III score of children who survived with AKI was 21.05 ± 5.77 and the mean PRISM III score of non survivors was 33.56 ± 5.04. The difference was statistically significant (p value <0.0001) in our study. Similar observation was made in a study by Sriram Krishnamurthy et al[19], the mean PRISM III score who survived was 16.6 ± 12.3 and the mean PRISM III score of non survivors was 31.2 ± 14.1 (p value <0.0001). In a study by Shweta naik et al[113], they found that the mean PRISM III scores were higher in the group of children with Acute Kidney injury than the children without Acute Kidney Injury.

The length of stay in PICU was higher in children with AKI than children without AKI in our study. The length of stay of the entire study population was found to be 9.4 ± 4.5 days**.** The length of stay in children with AKI was 9.4 ± 4.5 days which was higher than the length of stay in children without AKI, which was 5.6 ± 3.2 days. Also the length of stay of children who survived was 11.1 ± 4.1 days and that of the non-survivors was 7.1 ± 4.1 days.

PARAMETER	**PRESENT STUDY**	**Sriram Krishnamurthy et al[19]**
OVERALL	9.4 ± 4.5 days	8.4 ± 5.4 days
SURVIVORS	11.1 ± 4.1 days	10.1 ± 5.8 days
NON SURVIVORS	7.1 ± 4.1 dayS	6.3 ± 4.3 days

From the above table it is evident that the length of stay was higher in Acute Kidney Injury group. Similar observations were made in studies done by Shweta naik et al and Srinivasa et al. These studies showed longer period of stay in PICU in children with Acute kidney Injury.

ETIOLOGICAL PROFILE

In our study, the most common cause of Acute Kidney Injury was infections (56.6%). Of which, sepsis constitutes 30% (overall 17%). Other common

etiologies being meningoencephalitis (15.1%), bronchopneumonia (10.4%), cardiac causes (8.5%), UTI (6.6%), snake envenomation (6.6%), viral hemorrhagic fever and status epilepticus constitute 4.7% each, AGN (3.8%), scorpion sting (3.8%), HUS d+ (2.8%), nephritic syndrome (2.8%). Previous studies show sepsis, glomerulonephritis and HUS as predominant etiologies in developing countries , which have been replaced by hemato-oncological complications and pulmonary failure as causes of AKI in west[19,114]. In our study sepsis, meningoencephalitis and bronchopneumonia accounted for majority of all infections. They were also associated with high mortality whereas diarrhea, scorpion sting and nephrotic syndrome had no mortality. Increased risk of developing AKI has been mentioned with pneumonia, but seems to have been under-reported in children[115]. In a prospective study from Scotland, out of 1241 adults with pneumonia, 18% had AKI[116]. In the study by Shweta naik et al[113], sepsis, bronchopneumonia, status epilepticus, gastroenteritis and renal pathologies were identified as significant causes of AKI. In the study by Sriram krishnamrthy et al[19], pneumonia accounted for about 66.7% as a cause of AKI. Other significant causes found to be associated with AKI were sepsis, meningoencephalitis in that study. In a study by Garuda Rama et al[122], the most common associated etiology with AKI was sepsis. 7 cases of snake envenomation and 4 cases of scorpion sting were reported in our study to be associated with AKI which is a common problem in some parts of India[117].

MORTALITY

The mortality in AKI in children also has been reported to vary widely from 16% to 43.8%[6,14,16,18,118]. In our study, it was 42.5% (by AKIN staging) and 43.7% (by p-RIFLE classification), which is comparable to a recent study from Kuwait reporting 43.8% mortality[118]. The mortality rate in a study by Sriram Krishnamurthy et al[19] was found to be 46.3%. In the study by Shweta naik et al[113], mortality was found to be 15.5% in AKI group. In a study by Mehta et al[20], the mortality was 37% in AKI group. In a study by Miklaszewka et al[119], 2014 the mortality was found to be 40%. In a study by Martin et al[120], 2013 the mortality was 44%. Studies by Miklaszewka and Martin used p-RIFLE class to define AKI.

AKIN STAGE	SURVIVO-RS	DEATH	TOTAL	ODDS RATIO	95% CI	P value
STAGE 1	28 (65.1%)	15 (34.9%)	43 (100%)			
STAGE 2	17 (60.7%)	11 (39.3%)	28 (100%)	**0.84**	**0.32-2.20**	**0.692**
STAGE3	16 (45.7%)	19 (54.3%)	35 (100%)	**2.06**	**0.84-5.08**	**0.083**
TOTAL	61 (57.5%)	45 (42.5%)	106(100%)			

RIFLE CLASS	SURVIVO-RS	DEATH	TOTAL	ODDS RATIO	95% CI	P value
RISK	22 (62.9%)	13 (37.1%)	35 (100%)			
INJURY	19 (61.3%)	12 (38.7%)	31 (100%)	**0.502**	**0.19-1.28**	**0.152**
FAILURE	17 (46%)	20 (54%)	37 (100%)	**1.863**	**0.70-4.91**	**0.209**
TOTAL	58 (56.3%)	45 (43.7%)	103(100%)			

From the above tables which compare the mortality in each stage of the disease classifications, it is evident that there was no statistical significance between AKIN staging and RIFLE classification. Similar observation was made in a study by Srinivasa et al. In a study by Scott M. Sutherland et al[121], they found that pRIFLE, AKIN, and KDIGO result in different incidences and substantial disparities in staging. All three definitions correlate highly with outcomes and demonstrate excellent interstage discrimination. In a study by Talita Machado Levi et al, they found that the RIFLE, AKIN and KDIGO scores were all good predictors of mortality in critically ill patients, and there were no differences among them in terms of predicting death. These scores are good predictors of death.

Apart from mortality, the other short term outcomes that were observed in our study were the complete and partial renal recovery. A total of 49 children of the survivors with AKI (80.3%) had complete renal recovery while 12 children

(19.7%) of the survivors had partial renal recovery at discharge. Majority of partial renal recovery belong to stage 3, indicating towards significant morbidity associated with Acute Kidney Injury. This observation was comparable with the study by Sriram Krishnamurthy et al[19], where 20.7% children who survived with AKI had partial renal recovery at the time of discharge. In a study by Shweta naik et al[113], 38.8% children had partial renal recovery. In our study, we followed up the children who were discharged with partial renal recovery for a period of 3 months. 4 had hypertension and 8 had elevated creatinine levels at the time of discharge. 1 child with hypertension recovered completely while 1 still have hypertension (controlled with medications). 2 children were lost to follow up. Out of 8 children with elevated creatinine levels, 1 child died and 3 lost to follow up and other 4 had elevated creatinine levels at 2 months follow up. (serum creatinine levels 50% above baseline).

COMPLICATIONS

In our study, Hyponatremia (57.5%), Hypernatremia (16%), hypokalemia (22.6%), hyperkalemia (18.9%), anemia (55.7%), thrombocytopenia (23.6%), hypertension (15.1%) and metabolic acidosis (57.5%) were the associated complications and co-morbidities found in children with Acute Kidney Injury. A total of 59 children (55.7%) out of 106 AKI children needed mechanical ventilation and 77 children (72.6%) had shock as co-morbidity. Results were

comparable with other similar studies. In a study be Garuda Ram et al[122], Sepsis (50%), Encephalopathy (33.3%), Hypertension (33.3%), Metabolic Acidosis (33.3%), Fluid overload (19.8%), Hyperkalemia (13.6%) and bleeding tendency (13.6%) were the complications associated with AKI children. In a study by Sriram Krishnamurthy et al, the complications and co-morbidities observed in the group of children with AKI included severe metabolic acidosis in 32 (59.3%), hyponatremia in 14 (25.9%), hypernatremia in 8 (14.8%), hyperkalemia in 13 (24.1%), hypertension in 8 (14.8%), encephalopathy in 19 (35.2%), thrombocytopenia in 21 (38.9%), mechanical ventilation in 43 (79.6%) and shock in 47 (87%) children.

RENAL REPLACEMENT THERAPY

In our study, a total of 28 children (26.4%) required dialysis in the form of peritoneal dialysis. In a study by Sriram Krishnamurthy et al[19], a total of 27.8% required dialysis. Our study was comparable with other similar studies like a study by Mehta et al[20], where 15.1% required dialysis. In our study, the mortality among children requiring RRT was similar to children not requiring RRT (42.9% vs. 42.3%) and the difference was not significant. Similar observation was made in a study by Sriram Krishnamurthy et al[19], where the mortality among children requiring RRT was similar to children not requiring RRT (46.7% vs. 46.2%).

PREDICTORS OF MORTALITY

In our study, the predictors of mortality on univariate analysis were: Age less than 10 months, shock and requirement of mechanical ventilation, presence of metabolic acidosis. In the multivariate model, requirement of mechanical ventilation was found to be an independent predictor of mortality. In a study by Sriram Krishnamurthy et al[19], they observed that the predictors of mortality on univariate analysis were: Age less than 10 months, shock and requirement of mechanical ventilation while metabolic acidosis did not predict mortality. In the multivariate model, requirement of mechanical ventilation was found to be an independent predictor of fatality. Mehta et al[20] found that although crude mortality was higher in patients with AKI than those without AKI, AKI was not an independent predictor of mortality in multivariate analysis. Mehta et al found that shock was the only independent predictor of mortality. Shweta naik et al[113] found that need for mechanical ventilation and presence of multiorgan failure were the only independent risk factors of mortality in their study. Age below 2 years, shock, fluid overload, need for mechanical ventilation, multi-organ failure and late referral predicted poor outcomes in a study from Kuwait[118].

CONCLUSION AND RECOMMENDATION

- This dissertation has added to the literature regarding the incidence of acute kidney injury in critically ill children and helps to highlight on the importance and scope of AKI in this patient population.
- The incidence of Acute Kidney Injury among critically ill children admitted to ICH&RC, Madurai was 31%. Hence AKI is common in critically ill children.
- The mortality rate was 42.5% in critically ill children and 19.7% patients with AKI had partial renal recovery at the time of discharge. 26.4% patients required Renal replacement therapy.
- Frequency of AKI was more common among males than females.
- There is no difference between AKIN staging and p-RIFLE classification in identifying AKI cases. Both the criteria were good predictors of mortality.
- Children with AKI had significantly higher mortality rate and longer period of hospital stay than non AKI children irrespective of the criteria used.
- A clearer understanding of the long-term outcomes of this condition would allow optimization of follow-up strategies.
- No specific treatment for AKI exists. Preventing the development or worsening of AKI by removing risk factors when possible is currently the main method of reducing the incidence of AKI

- Existing diagnostic criteria (gold standard) for AKI depends on creatinine and urine output. Creatinine and Urine Output both are functional markers of glomerular filtration. As functional markers they lack sensitivity, specificity, and rapid timing to identify injury in the kidneys. The delay between the onset of injury and identifiable signs of AKI (loss of function) causes the therapeutic window, during which potential interventions could be tested or carried out, to be missed. More accurate ways of rapidly identifying acute damage in the kidneys are needed.
- Constructing AKI risk stratification models (Renal Angina) on the basis of existing data and implementing the models to clinical use could still increase awareness of AKI risk factors and help to further reduce the incidence of AKI.

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LIMITATIONS OF THE STUDY

Some of our study limitations were

- It was a single centre observational study. Therefore only associations can be shown, and no absolute causality.
- Being a tertiary care centre, we receive a lot of referral cases from peripheral hospitals. Lack of pre-referral treatment documentation concerning intravenous fluids, nephrotoxins like non steroidal anti-inflammatory drugs, gentamycin which could have changed the initial presentation.
- Diet, as a major factor influencing serum creatinine was not considered in this study.
- In our study, the estimation of a normal baseline GFR for age was used, since all patients did not have a prior GFR recorded.
- The worse part (either eCCl or FO) was used to stage AKI as in some cases Urine Output measurement was difficult in most cases and only eCCl or serum creatinine was used to stage AKI.
- We analyzed only short term outcomes of critically ill children with AKI. Children with AKI may have long-term residual renal injury e.g., microalbuminuria, hypertension or elevated creatinine levels. Lack of

information on the long-term outcome does not permit evaluation of the impact of mild AKI on renal function.

- We did not compare AKI and Non-AKI children and the data regarding Non-AKI cases were not collected and analyzed.
- Biomarkers were not used to predict AKI or its mortality in our study.
- Non availability of hemodialysis for children at our institute.

CHILD ON PERITONEAL DIALYSIS

BIBLIOGRAPHY

1. Andreoli SP. Acute kidney injury in children. Pediatr Nephrol. 2009 Feb;24(2):253-63.
2. Waikar Sushrut S., Bonventre Joseph V.: Acute Kidney Injury :Longo, Fauci, Kasper, Hauser, Jameson, Loscalzo, editors: Harrison's Principles of Internal Medicine, 18th ed, New York: Mc Graw-Hill; 2012. P2293-2308
3. Basu RK, Devarajan P, Wong H, Wheeler DS. An update and review of acute kidney injury in pediatrics. Pediatr Crit Care Med. 2011 May;12(3):339-47.
4. Schneider J, Khemani R, Grushkin C et al. Serum creatinine as stratified in the RIFLE score for acute kidney injury is associated with mortality and1length of stay for children in the pediatric intensive care unit. CritCareMed 2010; 38: 933–939
5. Bellomo R, Ronco C, Kellum JA, Mehta RL, Palevsky P. Acute renal failure - definition, outcome measures, animal models, fluid therapy and information technology needs: the Second International Consensus Conference of the Acute Dialysis Quality Initiative (ADQI) Group. Crit Care. [Consensus Development Conference Guideline Practice Guideline Review]. 2004 Aug;8(4):R204-12.

6. Akcan-Arikan A, Zappitelli M, Loftis LL, Washburn KK, Jefferson LS, Goldstein SL. Modified RIFLE criteria in critically ill children with acute kidney injury. Kidney international. [Research Support, Non-U.S. Gov't]. 2007 May;71(10):1028-35.
7. Schwartz G, J, Haycock G, B, Edelmann C, M, Jr, Spitzer A. A simple estimate of glomerular filtration rate in children derived from body length and plasma creatinine. Pediatrics. 1976;58(2):259-63.
8. Haengii M, H, Pelet J, Giugnard J, P. Estimation of glomerular filtration rate by formula GFR=K x T/Pc. Archives de pediatrie. 1999;6(2):165-72.
9. Mehta RL, Kellum JA, Shah SV et al. Acute Kidney Injury Network: report of an initiative to improve outcomes in acute kidney injury. Crit Care 2007; 11: R31.
10. Lassnigg A, Schmidlin D, Mouhieddine M et al. Minimal changes of serum creatinine predict prognosis in patients after cardiothoracic surgery: a prospective cohort study. J Am Soc Nephrol 2004; 15: 1597–1605.
11. Kidney Disease: Improving Global Outcomes (KDIGO) Acute Kidney InjuryWork Group: KDIGO Clinical Practice Guideline for Acute Kidney Injury. Kidney Inter Suppl 2012, 2:1–138.
12. Cruz DN, Bolgan I, Perazella MA, et al; North East Italian Prospective Hospital Renal Outcome Survey on Acute Kidney Injury (NEiPHROS-AKI)

Investigators. North East Italian Prospective Hospital Renal Outcome Survey on Acute Kidney Injury (NEiPHROS-AKI): targeting the problem with the RIFLE Criteria. Clin J Am Soc Nephrol. 2007;2:418–25.

13. Zappitelli M, Moffett BS, Hyder A, Goldstein SL. Acute kidney injury in non-critically ill children treated with aminoglycoside antibiotics in a tertiary healthcare centre: a retrospective cohort study. Nephrol Dial Transplant. 2011;26:144–50.

14. Plötz FB, Bouma AB, van Wijk JA, Kneyber MC, Bökenkamp A. Pediatric acute kidney injury in the ICU: an independent evaluation of pRIFLE criteria. Intensive Care Med. 2008;34:1713–7.

15. Palmieri T, Lavrentieva A, Greenhalgh D. An assessment of acute kidney injury with modified RIFLE criteria in pediatric patients with severe burns. Intensive Care Med. 2009;35:2125–9.

16. Duzova A, Bakkaloglu A, Kalyoncu M, et al; Turkish Society for Pediatric Nephrology Acute Kidney Injury Study Group. Etiology and outcome of acute kidney injury in children. Pediatr Nephrol.2010;25:1453–61.

17. Ratanarat R, Hantaweepant C, Tangkawattanakul N, Permpikul C. The clinical outcome of acute kidney injury in critically ill Thai patients stratified with RIFLE classification. J Med Assoc Thai.2009;92:S61–7.

18. Vachvanichsanong P, Dissaneewate P, Lim A, McNeil E. Childhood acute renal failure: 22-year experience in a university hospital in southern Thailand. Pediatrics. 2006;118:e786–91.
19. SriramKrishnamurthy, NiveditaMondal, Parameswaran Narayanan, NiranjanBiswal, SadagopanSrinivasan,RajendiranSoundravally. Incidence and etiology of acute kidney injury in southern India. Indian J Pediatr 2012 online publication.
20. Poonam Mehta, Aditi Sinha, Abdus Sami, Pankaj Hari, Mani Kalaivani, Ashima Gulati, Madhulika Kabra, Sushil K Kabra, Rakesh Lodha and Arvind Bagga from the Departments of Pediatrics and Biostatistics, All India Institute of Medical Sciences, New Delhi, India; 2011.
21. Lopes JA, Fernandes P, Jorge S, Goncalves S, Alvarez A, Costa e Silva Z, et al. Acute kidney injury in intensive care unit patients: a comparison between the RIFLE and the Acute Kidney Injury Network classifications. Critical Care. 2008;12:R110.
22. Bagshaw SM, George C, Bellomo R: A comparison of the RIFLE and AKIN criteria for acute kidney injury in critically ill patients. Nephrol Dial Transplant 23:1569Y1574. 2008.

23. Luo X, Li J, Ying W. The Beijing Acute Kidney Injury Trial (BAKIT) workgroup: A comparison of different diagnostic criteria of acute kidney injury in critically ill patients. Critical Care. 18:R144. 2014.
24. Adverse Nephrotoxic Events in Critically Ill Children: A Pharmacoepidemiologic Evaluation: Margan Brooke Slater, University of Toronto.
25. Lameire N, Van Biesen W, Vanholder R. Acute renal failure. Lancet; 365: 417-30; 2005.
26. Hoste, E. A. J., Clermont, G., Kersten, A., Venkataraman, R., Angus, D. C., De Bacquer, D., & Kellum, J. A. (2006). Rifle Criteria for Acute Kidney Injury Are Associated with Hospital Mortality in Critically Ill Patients: A Cohort Analysis. *Critical Care, 10*(3), R73.
27. Hoste, E. A. J., & De Corte, W. (2011). Clinical Consequences of Acute Kidney Injury. In J.A. Kellum, C. Ronco & J. L. Vincent (Eds.), *Controversies in Acute Kidney Injury* (Vol. 174, pp. 56-64). Basel: Karger.
28. Askenazi, D. (2011). Evaluation and Management of Critically Ill Children with Acute Kidney Injury. *Current Opinion in Pediatrics, 23*(2), 201-20
29. Hui-Stickle, S., Brewer, E. D., & Goldstein, S. L. (2005). Pediatric Arf Epidemiology at a Tertiary Care Center from 1999 to 2001. *American*

journal of kidney diseases : the official journal of the National Kidney Foundation, *45*(1), 96-101.

30. Symons, J. M., Chua, A. N., Somers, M. J., Baum, M. A., Bunchman, T. E., Benfield, M. R.,Goldstein, S. L. (2007). Demographic Characteristics of Pediatric Continuous Renal Replacement Therapy: A Report of the Prospective Pediatric Continuous Renal Replacement Therapy Registry. *Clinical journal of the American Society of Nephrology : CJASN*, *2*(4), 732-738

31. Alkandari, O., Eddington, K. A., Hyder, A., Gauvin, F., Ducruet, T., Gottesman, R.,Zappitelli, M. (2011). Acute Kidney Injury Is an Independent Risk Factor for Pediatric Intensive Care Unit Mortality, Longer Length of Stay and Prolonged Mechanical Ventilation in Critically Ill Children: A Two-Center Retrospective Cohort Study. *Critical Care, 15*(3), R146

32. Kavaz, A., Ozcakar, Z. B., Kendirli, T., Ozturk, B. B., Ekim, M., & Yalcinkaya, F. (2012). Acute Kidney Injury in a Paediatric Intensive Care Unit: Comparison of the Prifle and Akin Criteria. *Acta Paediatrica, 101*(3), e126-129.

33. Prodhan P., McCage, L. S., Stroud M. H., Gossett J, Garcia X., Bhutta, A. T, Blaszak R. T. (2012). Acute Kidney Injury Is Associated with Increased in-

Hospital Mortality in Mechanically Ventilated Children with Trauma. *The journal of trauma and acute care surgery, 73*(4), 832-837.

34. Aydin, S. I., Seiden, H. S., Blaufox, A. D., Parnell, V. A., Choudhury, T., Punnoose, A., & Schneider, J. (2012). Acute Kidney Injury after Surgery for Congenital Heart Disease. *The Annals of Thoracic Surgery, 94*(5), 1589-1595.

35. Blinder, J. J., Goldstein, S. L., Lee, V. V., Baycroft, A., Fraser, C. D., Nelson, D., & Jefferies, J. L. (2012). Congenital Heart Surgery in Infants: Effects of Acute Kidney Injury on Outcomes. *The Journal of Thoracic and Cardiovascular Surgery, 143*(2), 368-374.

36. Fadel, F. I., Abdel Rahman, A. M., Mohamed, M. F., Habib, S. A., Ibrahim, M. H., Sleem, Z. S., Soliman, M. M. (2012). Plasma Neutrophil Gelatinase-Associated Lipocalin as an Early Biomarker for Prediction of Acute Kidney Injury after Cardio-Pulmonary Bypass in Pediatric Cardiac Surgery. *Archives of medical science : AMS, 8*(2), 250-255.

37. Li, S., Krawczeski C. D., Zappitelli M., Devarajan P., Thiessen-Philbrook H., Coca S. G., Parikh, C. R. (2011). Incidence, Risk Factors, and Outcomes of Acute Kidney Injury after Pediatric Cardiac Surgery: A Prospective Multicenter Study. *Critical Care Medicine, 39*(6), 1493-1499.

38. A comparison of pRIFLE and AKIN criteria for acute kidney injury in pediatric intensive care unit patients; International Journal of Contemporary Pediatrics *2016 May;3(2):398-402* Srinivasa S., Reshmavathi V., Srividya G. S., Dept of Paediatrics, Kempegowda Institute of Medical Sciences
39. Ricci, Z., Cruz, D., & Ronco, C. (2008). The Rifle Criteria and Mortality in Acute Kidney Injury: A Systematic Review. *Kidney International, 73*(5), 538-546.
40. Askenazi, D. J., Feig, D. I., Graham, N. M., Hui-Stickle, S., & Goldstein, S. L. (2006). 3-5 Year Longitudinal Follow-up of Pediatric Patients after Acute Renal Failure. *Kidney International, 69*(1), 184-189
41. Coca, S. G., Singanamala, S., & Parikh, C. R. (2012). Chronic Kidney Disease after Acute Kidney Injury: A Systematic Review and Meta-Analysis. *Kidney International, 81*(5), 442-448.
42. Zappitelli, M., & Goldstein, S. L. (2009). Acute Kidney Injury: General Aspects. In S. G. Kiessling, J. Goebel & M. J. Somers (Eds.), *Pediatric Nephrology in the Icu*. Berlin: Springer.
43. Devarajan, P. (2013). Pediatric Acute Kidney Injury: Different from Acute Renal Failure but How and Why. *Current Pediatrics Reports, 1*(1), 34-40.

44. Amann, K., Wanner, C., & Ritz, E. (2006). Cross-Talk between the Kidney and the Cardiovascular System. *Journal of the American Society of Nephrology, 17*(8), 2112-2119.

45. Basu, R. K., & Wheeler, D. S. (2013). Kidney-Lung Cross-Talk and Acute Kidney Injury. *Pediatric Nephrology*.

46. Grams, M. E., & Rabb, H. (2012). The Distant Organ Effects of Acute Kidney Injury. *Kidney International, 81*(10), 942-948.

47. Singbartl K., Bishop J. V., Wen X., Murugan R., Chandra, S., Filippi, M. D., & Kellum, J. A. (2011). Differential Effects of Kidney-Lung Cross-Talk During Acute Kidney Injury and Bacterial Pneumonia. *Kidney International, 80*(6), 633-644.

48. Sutherland, S. M., Zappitelli, M., Alexander, S. R., Chua, A. N., Brophy, P. D., Bunchman, T. E., Goldstein, S. L. (2010). Fluid Overload and Mortality in Children Receiving Continuous Renal Replacement Therapy: The Prospective Pediatric Continuous Renal Replacement Therapy Registry. *American journal of kidney diseases : the official journal of the National Kidney Foundation, 55*(2), 316-325.

49. Bellomo R, Kellum JA, Ronco C. Acute kidney injury. Lancet; 380: 756-66; 2012.

50. Abuelo JG. Normotensive ischemic acute renal failure. N Engl J Med; 357: 797-805; 2007.
51. Chua HR, Glassford N, Bellomo R. Acute kidney injury after cardiac arrest. Resuscitation; 83: 721-7; 2012.
52. Molitoris BA, Sutton TA. Endothelial injury and dysfunction: role in the extension phase of acute renal failure. Kidney Int; 66: 496-9; 2004.
53. Devarajan P. Update on mechanisms of ischemic acute kidney injury. J Am Soc Nephrol; 17: 1503-20; 2006.
54. Thuillier R, Favreau F, Celhay O, Macchi L, Milin S, Hauet T. Thrombin inhibition during kidney ischemia-reperfusion reduces chronic graft inflammation and tubular atrophy. Transplantation; 90: 612-21; 2010.
55. Thurman JM. Triggers of inflammation after renal ischemia/reperfusion. Clin Immunol; 123: 7-13; 2007.
56. Sutton TA, Mang HE, Campos SB, Sandoval RM, Yoder MC, Molitoris BA. Injury of the renal microvascular endothelium alters barrier function after ischemia. Am J Physiol Renal Physiol; 285: F191-8; 2003.
57. Versteilen AM, Blaauw N, Di Maggio F, Groeneveld AB, Sipkema P, Musters RJ, Tangelder GJ. rho-Kinase inhibition reduces early microvascular leukocyte accumulation in the rat kidney following ischemia-

reperfusion injury: roles of nitric oxide and blood flow. Nephron Exp Nephrol; 118: e79-86; 2011.

58. Zuk A, Bonventre JV, Brown D, Matlin KS. Polarity, integrin, and extracellular matrix dynamics in the postischemic rat kidney. Am J Physiol; 275: C711-31; 1998.

59. Thurman JM, Lenderink AM, Royer PA, Coleman KE, Zhou J, Lambris JD, Nemenoff RA, Quigg RJ, Holers VM. C3a is required for the production of CXC chemokines by tubular epithelial cells after renal ishemia/reperfusion. J Immunol; 178: 1819-28; 2007.

60. Bonventre JV, Weinberg JM. Recent advances in the pathophysiology of ischemic acute renal failure. J Am Soc Nephrol; 14: 2199-210; 2003.

61. Schrier RW, Wang W, Poole B, Mitra A. Acute renal failure: definitions, diagnosis, pathogenesis, and therapy. J Clin Invest; 114: 5-14; 2004.

62. Wen X, Murugan R, Peng Z, Kellum JA. Pathophysiology of acute kidney injury: a new perspective. Contrib Nephrol; 165: 39-45; 2010

63. Wan L, Bagshaw SM, Langenberg C, Saotome T, May C, Bellomo R. Pathophysiology of septic acute kidney injury: what do we really know? Crit Care Med; 36: S198-203; 2008.

64. Takasu O, Gaut JP, Watanabe E, To K, Fagley RE, Sato B, Jarman S, Efimov IR, Janks DL, Srivastava A, Bhayani SB, Drewry A, Swanson PE,

Hotchkiss RS. Mechanisms of cardiac and renal dysfunction in patients dying of sepsis. Am J Respir Crit Care Med; 187: 509-17; 2013.

65. Jacobs R, Honore PM, Joannes-Boyau O, Boer W, De Regt J, De Waele E, Collin V, Spapen HD. Septic acute kidney injury: the culprit is inflammatory apoptosis rather than ischemic necrosis. Blood Purif; 32: 262-5; 2011.

66. Uchino S, Kellum JA, Bellomo R, Doig GS, Morimatsu H, Morgera S, Schetz M, Tan I, Bouman C, Macedo E, Gibney N, Tolwani A, Ronco C, Beginning, Ending Supportive Therapy for the Kidney I. Acute renal failure in critically ill patients: a multinational, multicenter study. JAMA; 294: 813-8; 2005.

67. Schrier RW, Wang W. Acute renal failure and sepsis. N Engl J Med; 351: 159-69; 2004.

68. Langenberg C, Wan L, Egi M, May CN, Bellomo R. Renal blood flow and function during recovery from experimental septic acute kidney injury. Intensive Care Med; 33: 1614-8; 2007.

69. Gomez H, Ince C, De Backer D, Pickkers P, Payen D, Hotchkiss J, Kellum JA. A unified theory of sepsis-induced acute kidney injury: inflammation, microcirculatory dysfunction, bioenergetics, and the tubular cell adaptation to injury. Shock; 41: 3-11; 2014.

70. Langenberg C, Bagshaw SM, May CN, Bellomo R. The histopathology of septic acute kidney injury: a systematic review. Crit Care; 12: R38; 2008.
71. Doi K, Leelahavanichkul A, Yuen PS, Star RA. Animal models of sepsis and sepsis induced kidney injury. J Clin Invest; 119: 2868-78; 2009.
72. Benes J, Chvojka J, Sykora R, Radej J, Krouzecky A, Novak I, Matejovic M. Searching for mechanisms that matter in early septic acute kidney injury: an experimental study. Crit Care; 15: R256; 2011.
73. Prowle JR, Molan MP, Hornsey E, Bellomo R. Measurement of renal blood flow by phase-contrast magnetic resonance imaging during septic acute kidney injury: a pilot investigation. Crit Care Med; 40: 1768-76; 2012.
74. Ishikawa K, May CN, Gobe G, Langenberg C, Bellomo R. Pathophysiology of septic acute kidney injury: a different view of tubular injury. Contrib Nephrol; 165: 18-27; 2010.
75. Gustot T. Multiple organ failure in sepsis: prognosis and role of systemic inflammatory response. Curr Opin Crit Care; 17: 153-9; 2011.
76. Murugan R, Karajala-Subramanyam V, Lee M, Yende S, Kong L, Carter M, Angus DC, Kellum JA, Genetic, Inflammatory Markers of Sepsis I. Acute kidney injury in non-severe pneumonia is associated with an increased immune response and lower survival. Kidney Int; 77: 527-35; 2010.

77. Knotek M, Rogachev B, Wang W, Ecder T, Melnikov V, Gengaro PE, Esson M, Edelstein CL, Dinarello CA, Schrier RW. Endotoxemic renal failure in mice: Role of tumor necrosis factor independent of inducible nitric oxide synthase. Kidney Int; 59: 2243-9; 2001

78. Ishikawa K, Bellomo R, May CN. The impact of intrarenal nitric oxide synthase inhibition on renal blood flow and function in mild and severe hyperdynamic sepsis. Crit Care Med; 39: 770-6; 2011.

79. May CN, Ishikawa K, Wan L, Williams J, Wellard RM, Pell GS, Jackson GD, Bellomo R. Renal bioenergetics during early gram-negative mammalian sepsis and angiotensin II infusion. Intensive Care Med; 38: 886-93; 2012.

80. Wan L, Langenberg C, Bellomo R, May CN. Angiotensin II in experimental hyperdynamic sepsis. Crit Care; 13: R190; 2009.

81. Loutzenhiser R, Griffin K, Williamson G, Bidani A. Renal autoregulation: new perspectives regarding the protective and regulatory roles of the underlying mechanisms. Am J Physiol Regul Integr Comp Physiol; 290: R1153-67; 2006.

82. Incidence, Biomarkers, and Outcome of Acute Kidney Injury in critically ill adults; sara nisula, University of Helsinki, Finland

83. Traynor, J., Mactier, R., Geddes, C. C., & Fox, J. G. (2006). How to Measure Renal Function in Clinical Practice. *British Medical Journal, 333*(7571), 733-737.

84. Stevens, L. A., & Levey, A. S. (2009). Measured Gfr as a Confirmatory Test for Estimated Gfr. *Journal of the American Society of Nephrology, 20*(11), 2305-2313.

85. Chantler, C., Garnett, E. S., Parsons, V., & Veall, N. (1969). Glomerular Filtration Rate Measurement in Man by the Single Injection Methods Using 51cr-Edta. *Clinical Science, 37*(1), 169-180.

86. Proulx, N. L., Akbari, A., Garg, A. X., Rostom, A., Jaffey, J., & Clark, H. D. (2005). Measured Creatinine Clearance from Timed Urine Collections Substantially Overestimates Glomerular Filtration Rate in Patients with Liver Cirrhosis: A Systematic Review and Individual Patient Meta-Analysis. *Nephrology, dialysis, transplantation : official publication of the European Dialysis and Transplant Association – European Renal Association*, *20*(8), 1617-1622.

87. "K/Doqi Clinical Practice Guidelines for Chronic Kidney Disease: Evaluation, Classification, and Stratification," 2002.

88. Coca SG, Yalavarthy R, Concato J, Parikh CR. Biomarkers for the diagnosis and risk stratification of acute kidney injury: a systematic review. Kidney Int; 73: 1008-16; 2008.

89. Dixon BS, Anderson RJ. Nonoliguric acute renal failure. Am J Kidney Dis; 6: 71-80; 1985.

90. Devarajan, P. (2011). Biomarkers for the Early Detection of Acute Kidney Injury. *Current Opinion in Pediatrics*, *23*(2), 194-200.

91. Cruz DN, Goh CY, Haase-Fielitz A, Ronco C, Haase M. Early biomarkers of renal injury. Congest Heart Fail; 16 Suppl 1: S25-31; 2010.

92. Haase M, Bellomo R, Haase-Fielitz A. Neutrophil gelatinase-associated lipocalin. Curr Opin Crit Care; 16: 526-32; 2010.

93. Mishra, J., Dent, C., Tarabishi, R., Mitsnefes, M. M., Ma, Q., Kelly, C., Devarajan, P. (2005). Neutrophil Gelatinase-Associated Lipocalin (Ngal) as a Biomarker for Acute Renal Injury after Cardiac Surgery. *Lancet*, *365*(9466), 1231-1238.

94. Parikh, C. R., Devarajan, P., Zappitelli, M., Sint, K., Thiessen-Philbrook, H., Li, S., Krawczeski, C. D. (2011). Postoperative Biomarkers Predict Acute Kidney Injury and Poor Outcomes after Pediatric Cardiac Surgery. *Journal of the American Society of Nephrology : JASN*, *22*(9), 1737-1747.

95. Krawczeski, C. D., Goldstein, S. L., Woo, J. G., Wang, Y., Piyaphanee, N., Ma, Q., Devarajan, P. (2011). Temporal Relationship and Predictive Value of Urinary Acute Kidney Injury Biomarkers after Pediatric Cardiopulmonary Bypass. *Journal of the American College of Cardiology, 58*(22), 2301-2309.

96. Bennett, M., Dent, C. L., Ma, Q., Dastrala, S., Grenier, F., Workman, R., Devarajan, P (2008). Urine Ngal Predicts Severity of Acute Kidney Injury after Cardiac Surgery: A Prospective Study. *Clinical Journal of the American Society of Nephrology, 3*(3), 665-673.

97. Du, Y., Zappitelli, M., Mian, A., Bennett, M., Ma, Q., Devarajan, P., Goldstein, S. L. (2011). Urinary Biomarkers to Detect Acute Kidney Injury in the Pediatric Emergency Center. *Pediatric Nephrology*, *26*(2), 267-274.

98. Wheeler, D. S., Devarajan, P., Ma, Q., Harmon, K., Monaco, M., Cvijanovich, N., & Wong, H. R. (2008). Serum Neutrophil Gelatinase-Associated Lipocalin (Ngal) as a Marker of Acute Kidney Injury in Critically Ill Children with Septic Shock. *Critical Care Medicine, 36*(4), 1297-1303.

99. Padiatric acute kidney injury: A syndrome under paradigm shift; Mohd Ashraf, Naveen Shahzad, Mohd Irshad, Sheikh quyoom Hussain, Parvez Ahmed, Indian Journal of Critical Care Medicine, 2014, vol 18, page 518-526

100. Haase, M., Bellomo, R., Devarajan, P., Schlattmann, P, & Haase-Fielitz, A. (2009). Accuracy of Neutrophil Gelatinase-Associated Lipocalin (Ngal) in Diagnosis and Prognosis in Acute Kidney Injury: A Systematic Review and Meta-Analysis. *American Journal of Kidney Diseases, 54*(6), 1012-1024.

101. Gourishankar, S., Courtney, M., Jhangri, G. S., Cembrowski, G., & Pannu, N. (2008). Serum Cystatin C Performs Similarly to Traditional Markers of Kidney Function in the Evaluation of Donor Kidney Function Prior to and Following Unilateral Nephrectomy. *Nephrology, dialysis, transplantation : official publication of the European Dialysis and Transplant Association - European Renal Association, 23*(9), 3004-3009.

102. Devarajan, P. (2008). The Future of Pediatric Acute Kidney Injury Management--Biomarkers. *Seminars in Nephrology, 28*(5), 493-498.

103. Renal angina: Concept and development of pretest probability assessment in acute kidney injury, Chawla et al, critical care (2015) 19:93

104. Joannidis M, Metnitz B, Bauer P, Schusterschitz N, Moreno R, Druml W, Metnitz PG. Acute kidney injury in critically ill patients classified by AKIN versus RIFLE using the SAPS 3 database. Intensive Care Med; 35: 1692-702; 2009.

105. Hofhuis JG, van Stel HF, Schrijvers AJ, Rommes JH, Spronk PE. The effect of acute kidney injury on long-term health-related quality of life: a prospective follow-up study. Crit Care; 17: R17; 2013.

106. Basu, R. K., Chawla, L. S., Wheeler, D. S., & Goldstein, S. L. (2012). Renal Angina: An Emerging Paradigm to Identify Children at Risk for Acute Kidney Injury. *Pediatric Nephrology, 27*(7), 1067-1078.

107. Bowers LD, Wong ET. Kinetic serum creatinine assays. II. A critical evaluation and review. Clin Chem.1980;26:555–61.

108. Nelson textbook of pediatrics; 20th edition, kleigman, Stanton, St Geme, Schor; table 109-8.

109. Goldstein B, Giroir B, Randolph A. International Consensus Conference on Pediatric Sepsis. International pediatric sepsis consensus conference: Definitions for sepsis and organ dysfunction in pediatrics. Pediatr Crit Care Med. 2005;6:2–8.

110. Lassnigg, A., Schmidlin, D., Mouhieddine, M., Bachmann, L. M., Druml, W., Bauer, P., & Hiesmayr, M. (2004). Minimal Changes of Serum Creatinine Predict Prognosis in Patients after Cardiothoracic Surgery: A Prospective Cohort Study. *Journal of the American Society of Nephrology : JASN, 15*(6), 1597-1605.

111. Bagshaw, S. M., George, C., Bellomo, R., & Committe, A. D. M. (2008). A Comparison of the Rifle and Akin Criteria for AKI in Critically Ill Patients. *Nephrology Dialysis Transplantation, 23*(5), 1569-1574.

112. Mishra SK, Das BS. Malaria and acute kidney injury. Semin Nephrol. 2008 Jul;28(4):395-408.

113. Acute kidney injury in critically ill children; risk factors and outcome, Indian Journal of Critical Care Medicine, March 2014 Vol 18 Issue 3, Shweta naik, Jyoti Sharma, Rameshwar yengkom, Vijay Kalroa, Atul Mulay.

114. . Srivastava RN, Bagga A, Moudgil A. Acute renal failure in north Indian children. Indian J Med Res.1990;92:404–8.

115. Muntner P, Warnock DG. Acute kidney injury in sepsis: Questions answered, but others remain. Kidney Int. 2010;77:485–7.

116. Akram AR, Singanayagam A, Choudhury G, Mandal P, Chalmers JD, Hill AT. Incidence and prognostic implications of acute kidney injury on admission in patients with community-acquired pneumonia. Chest. 2010;138:825–32.

117. Sinha R, Nandi M, Tullus K, Marks SD, Taraphder A. Ten-year follow-up of children after acute renal failure from a developing country. Nephrol Dial Transplant. 2009;24:829–33

118. Ghani AA, Al Helal B, Hussain N. Acute renal failure in pediatric patients: Etiology and predictors of outcome. Saudi J Kidney Dis Transpl. 2009;20:69–76.

119. Martin SM, Balestracci A, Aprea V, et al. Acute kidney injury in critically ill children: incidence and risk factors for mortality. Archivos Argentinos de Pediatria. 2013;111(5):411-416.

120. Miklaszewska M, Korohoda P, Sobczak A, et al. Acute kidney injury in a single pediatric intensive care unit in Poland: a retrospective study. Kidney & Blood Pressure Research. 2014;39(1):28-39.

121. AKI in Hospitalized Children: Comparing the pRIFLE, AKIN, and KDIGO Definitions; Scott M. Sutherland, John J. Byrnes, Manish Kothari, Christopher A. Longhurst, Sanjeev Dutta, Pablo Garcia, and Stuart L. Goldstein.

122. Study of Acute Kidney Injury in children; Its aetiology, clinical profile and outcome; Garuda Ram, Journal of Evidence based Medicine and Healthcare; Vol 2, Issue 11, March 16, 2015.

PROFORMA

NAME: AGE/SEX: ADDRESS:

DOA: DOD/DOE: DURATION OF STAY:

COMPLAINTS:

EXAMINATION FINDINGS:

HR	RR	BP	CRT	PERIPHERIES	SPO2

SYSTEMIC EXAMINATION:

PROVISIONAL DIAGNOSIS:

INVESTIGATIONS									
UREA CREATININE									
URINE ROUTINE EXAMINATION									
URINE C/S									
BLOOD C/S									
CRP									
Na K Cl HCO3									
Hb TC DC PLC PCV									
OTHERS(LFT, etc..)									

Date / Hourly									
Q6H									
Q6H									
Q6H									
Q6H									
TOTAL									

IMAGING STUDIES:

ABG ANALYSIS:

CO-MORBIDITIES: Sepsis / Hemorrhage / Dehydration / Cardiac failure / Nephrotoxins / Bladder outflow obstruction / Stones / Tumor / Glomerulonephritis / Tubulo-Interstititial Nephritis / Rhabdomyolysis / HUS / Malignant Hypertension / Mechanical Ventilation / others

MANAGEMENT:

OUTCOME:

FOLLOW UP:

ABBREVIATIONS

ADQI	**-**	**Acute Dialysis Quality Initiative**
AKI	**-**	**Acute Kidney Injury**
AKIN	**-**	**Acute Kidney Injury Network**
ARF	**-**	**Acute Renal Failure**
ATN	**-**	**Acute Tubular Necrosis**
ATP	**-**	**Adenosine Triphosphate**
BP	**-**	**Blood Pressure**
BUN	**-**	**Blood Urea Nitrogen**
CI	**-**	**Confidence Interval**
CO	**-**	**Cardiac Output**
eCCl	**-**	**estimated Creatinine Clearance**
FeNa	**-**	**Fractional Excretion of Sodium**
GFR	**-**	**Glomerular Filtration Rate**
HUS d+	**-**	**diarrheal type of Hemolytic Uraemic Syndrome**

IL-18	**-**	**Interleukin 18**
IQR	**-**	**Inter-Quartile Range**
KDIGO	**-**	**Kidney Disease: Improving Global Outcome**
KIM-1	**-**	**Kidney Injury Molecule 1**
NGAL	**-**	**Neutrophil Gelatinase-associated Lipocalin**
UTI	**-**	**Urinary Tract Infections**
NO	**-**	**Nitric Oxide**
PICU	**-**	**Paediatric Intensive Care Unit**
p-RIFLE	**-**	**pediatric Risk, Injury, Failure, Loss of kidney function, End stage renal disease**
PRISM III	**-**	**Paediatric Risk of Mortality Score**
PUJ	**-**	**Pelvi-Ureteric Junction**
RBF	**-**	**Renal Blood Flow**
RRT	**-**	**Renal Replacement Therapy**
SCr	**-**	**Serum Creatinine**
UO	**-**	**Urine Output**

NAME	AGE	SEX	DURATION OF STAY	PRISM III	CREATININE	AKIN STAGE	p-RIFLE CLASS	SODIUM LEVEL	POTASSIUM LEVEL	ANEMIA	THROMBOCYTOPENIA	HYPERTENSION	USG ABD	ABG ANALYSIS	RRT	MECHANICAL VENTILATION	INOTROPE USE	BLOOD CULTURE	URINE CULTURE	OUTCOME	FINAL OUTCOME	p-RIFLE PROGRESSION
Santhosh	3.5	2	11	28	1.6	1	3	2	0	2	1	1	B/L Renal Cortical Echoes increased	2	1	2	2	1	0	2	3	0
Mohitdharan	2	2	15	26	0.4	0	0	1	0	1	1	1	Normal	0	1	1	1	3	0	1	3	0
Rasiga	2	1	17	32	1.3	2	2	1	0	2	1	1	Normal	2	1	2	2	2	0	2	3	0
B/O kalaivani	2	2	20	11	0.4	0	0	0	0	1	1	1	Normal	0	1	1	1	1	0	1	0	0
Devika	120	1	6	8	0.7	0	0	0	0	1	2	1	Normal	0	1	1	1	0	0	1	0	0
Kayalvizhi	96	1	8	6	0.7	0	0	0	0	1	2	1	Normal	0	1	1	1	0	0	1	0	0
Muthulakshmi	60	1	14	6	0.8	0	0	0	0	2	2	1	Normal	0	1	1	1	0	0	1	0	0
Srivarsan	2	2	3	12	0.4	0	0	1	0	1	1	1	Normal	0	1	1	1	2	0	1	0	0
Santhosh kumar	42	2	16	23	1.5	2	2	0	0	1	1	1	Normal	2	1	1	1	2	1	2	2	0
Samrat	9	2	20	12	0.5	0	0	0	0	2	1	1	Normal	0	1	1	1	0	0	1	0	0
Ajay	84	2	17	36	1.2	1	1	0	0	2	1	1	Normal	2	1	1	1	1	3	2	2	0
Mutheeswari	5	1	4	6	0.4	0	0	1	0	1	1	1	Normal	0	1	1	1	0	0	1	0	0
Moushika	84	1	4	6	0.7	0	0	1	0	1	2	1	Normal	0	1	1	1	0	0	1	0	0
Jeevan	36	2	12	12	0.6	0	0	1	0	2	2	1	HSM+	0	1	1	1	1	0	1	0	0
Harish	132	2	5	6	0.8	0	0	0	0	1	2	1	Normal	0	1	1	1	0	0	1	0	0
Vijayasri	108	1	4	4	0.8	0	0	0	0	1	1	1	Normal	0	1	1	1	0	0	1	0	0
Ragini	120	1	3	10	0.7	0	0	0	0	2	2	1	Normal	0	1	1	1	0	0	1	0	0
Nithya	108	1	6	10	0.6	0	0	0	0	2	2	1	Normal	0	1	1	1	1	0	1	0	0
Maheshwari	72	1	7	16	0.5	0	0	0	0	2	1	1	Normal	0	1	1	1	1	0	1	0	0
Delamiyasri	7	1	9	26	0.8	1	1	0	1	1	2	1	Normal	1	1	1	1	1	0	2	2	0
Manoj Kumar	48	2	10	12	0.6	0	0	0	0	1	1	1	Normal	0	1	1	1	0	0	1	0	0
Aarthy	84	1	9	8	0.6	0	0	0	0	2	1	1	Normal	0	1	1	1	1	0	1	0	0
Arunraj	144	2	4	6	0.8	0	0	1	1	1	2	1	Normal	0	1	1	1	0	0	1	0	0
Ashwin	48	2	16	18	1.5	2	2	1	2	1	2	1	Normal	2	1	1	2	2	0	2	2	0

Kabilan	22	2	10	9	0.5	0	0	0	0	1	1	1	Normal	0	1	1	1	1	0	1	0	0
Kandavel	12	2	5	4	0.4	0	0	0	0	2	1	1	Normal	0	1	1	1	0	0	1	0	0
Harish Kumar	96	2	18	18	1.9	2	2	1	2	2	1	1	Normal	2	1	2	2	1	0	2	2	0
Diyasri	4	1	8	34	1.3	2	2	0	1	1	1	1	Normal	1	1	1	1	1	0	2	1	0
Apsana	3	1	21	35	1	2	2	0	0	2	2	1	Normal	2	1	1	1	2	0	2	3	0
Kathirmathiyan	3	2	5	7	0.5	0	0	1	0	1	1	1	Normal	0	1	1	1	1	0	1	0	0
Yogesh	36	2	3	8	0.6	0	0	0	0	2	1	1	HSM+	0	1	1	1	1	0	1	0	0
B/o Panju	5	2	7	15	1.3	2	2	2	1	1	1	1	Normal	2	1	1	1	1	0	2	2	0
Madan Kumar	132	2	5	6	0.7	0	0	1	0	1	2	1	Normal	1	1	1	1	0	0	1	0	0
Aaler Rijal	3	2	6	5	0.4	0	0	0	0	1	2	1	Normal	0	1	1	1	0	0	1	0	0
Abhiji ram	10	2	9	7	0.5	0	0	0	0	2	1	1	Normal	0	1	1	1	0	0	1	0	0
Lisanth	12	2	6	6	0.6	0	0	0	0	1	2	1	Normal	0	1	1	1	0	0	1	0	0
Suryaprakash	96	2	5	6	0.7	0	0	1	1	1	2	1	HSM+	0	1	1	1	0	0	1	0	0
Karupasamy	108	2	5	4	0.7	0	0	1	1	1	1	1	Normal	0	1	1	1	0	0	1	0	0
Swasthi	96	1	9	6	0.7	0	0	1	1	1	2	1	HSM+	0	1	1	1	0	0	1	0	0
Rakshana	48	1	5	4	0.6	0	0	0	0	1	1	1	Normal	0	1	1	1	0	0	1	0	0
Sri Sakthi Sophia	6	1	5	11	0.7	2	2	2	2	1	1	1	Normal	2	1	1	1	0	0	2	2	0
Renuka devi	132	1	18	23	0.8	1	1	0	0	1	1	1	Normal	2	1	1	1	0	6	2	2	0
Sowmya	12	1	16	21	0.5	0	0	0	0	1	1	1	Normal	0	1	1	1	0	0	1	0	0
Karthick	144	2	6	39	5	3	3	1	1	2	1	1	B/L Renal Cortical Echoes increased, B/L Enlarged Kidneys	2	2	2	2	1	1	2	1	0
Nithya	36	1	6	17	1.4	1	1	0	0	2	2	1	GB wall edema+	1	1	1	2	1	0	2	2	0
Dhavapriyan	2	2	8	13	0.6	2	2	0	2	1	1	1	Normal	0	1	1	1	1	0	2	2	0
Balaji	36	2	13	13	1	2	2	1	0	1	1	2	Cystitis	1	1	1	1	1	4	2	1	0
Mahathi	7	1	16	12	1.5	2	2	2	1	1	2	1	Normal	2	1	1	1	1	0	2	1	0
Malaisamy	12	2	19	24	3.6	3	3	1	0	2	2	1	B/L Renal Cortical Echoes Increased	2	2	2	2	4	0	2	2	0
Yogeshwari	2	1	14	23	1.1	2	2	1	0	1	2	1	Normal	0	1	1	1	2	1	2	2	0
Muthukumaran	10	2	17	16	4.1	3	3	1	2	2	1	2	Right Hydroureteronephrosis	2	2	1	1	1	5	2	1	0
Boopesh	10	2	15	15	3.9	3	3	0	0	1	1	2	PUJ obstruction	1	2	1	1	1	2	2	1	0

Saran	5	2	4	42	6.7	3	3	0	0	2	2	1	B/L renal cortical echoes increased	2	2	2	2	3	1	2	3	0
Sabeerabegam	48	1	6	17	5.4	3	3	1	2	2	2	1	B/L renal cortical echoes increased	2	2	1	1	1	1	2	1	0
Lavanya	30	1	9	23	3.6	3	3	1	0	2	2	1	B/L renal cortical echoes increased	2	2	2	2	1	1	2	2	0
Sankar	132	2	12	21	1.7	2	2	2	1	1	1	2	Normal	1	1	2	2	1	0	2	2	0
B/O Gangalaxmi	2	1	17	19	1	2	2	0	0	1	1	1	Normal	0	1	1	1	1	0	2	2	0
Saranya	144	1	6	31	0.8	0	0	1	0	2	2	1	Normal	2	1	2	2	1	0	1	3	0
Isanthuraj	96	2	12	37	2	2	2	1	1	1	1	1	Normal	2	1	2	2	1	0	2	3	0
Bharathi raja	48	2	5	33	1.4	2	1	1	0	2	1	1	Normal	2	1	2	2	5	1	2	3	3
B/O Sujatha	2	1	9	27	0.6	1	1	1	0	2	1	1	Normal	1	1	2	2	1	0	2	3	0
Tamilarasan	15	2	5	6	0.6	0	0	0	0	2	1	1	Normal	1	1	1	1	1	0	1	0	0
Lahimiya Sri	96	1	8	13	2	1	1	1	2	1	2	1	GB wall edema+	1	1	1	2	1	0	2	2	3
Harshana Sri	6	1	4	29	8	3	3	1	2	2	1	1	B/L renal cortical echoes increased	2	2	2	2	1	0	2	3	0
Pon Vishnu	48	2	4	25	1.2	2	2	2	0	2	2	1	Normal	2	1	2	2	1	0	2	3	0
kaajakishan	2	2	5	4	0.5	0	0	0	0	2	1	1	Normal	1	1	1	1	1	0	1	0	0
Mathinilavan	120	2	11	11	1.4	2	2	0	0	2	1	2	Normal	1	1	1	1	1	0	2	1	0
Pradhesh	18	2	10	4	0.6	0	0	0	0	2	1	1	Normal	0	1	1	1	1	0	1	0	0
Jesima	108	1	9	13	1	1	1	2	2	1	1	1	Normal	1	1	1	2	1	0	2	2	0
Sathish pandi	60	2	6	11	0.6	0	0	0	0	1	1	1	Normal	0	1	1	1	5	0	1	0	0
Dharshan pandi	5	2	5	10	0.5	0	0	0	0	2	1	1	Normal	0	1	1	1	2	0	1	0	0
Madhanasri	36	1	5	8	0.6	0	0	1	1	1	1	1	Normal	0	1	1	1	0	0	1	0	0
Santhiya	120	1	6	6	0.8	0	0	0	0	1	2	1	Normal	0	1	1	1	0	0	1	0	0
Rohith	4	2	4	9	0.5	0	0	1	0	2	1	1	Normal	0	1	1	1	2	0	1	0	0
Sivasri	9	1	6	6	0.5	0	0	0	0	1	1	1	Normal	0	1	1	1	1	0	1	0	0
B/O Lakshmi	9	1	9	25	1.7	2	1	1	0	2	2	1	Normal	1	1	2	2	1	0	2	3	3
Meenakshi	132	1	12	19	2	1	1	1	0	2	1	1	Normal	1	1	1	1	1	0	2	2	2
Mouna guru	42	2	7	9	0.5	0	0	0	0	1	1	1	Normal	0	1	1	1	4	0	1	0	0
Deepak Raja	108	2	5	11	0.6	0	0	0	0	2	1	1	Normal	0	1	1	1	1	0	1	0	0
Nagaselvan	24	2	9	16	0.8	1	0	1	0	1	1	1	Normal	1	1	1	1	0	0	2	2	0
Jeevan	2	2	3	12	0.4	0	0	1	0	2	1	1	Normal	0	1	1	1	5	0	1	0	0

Kaviashri	6	2	4	22	1.2	2	2	2	2	2	1	1	Normal	2	1	2	2	1	0	2	3	0
Dhanalaxmi	84	1	4	27	1.1	1	1	1	0	1	2	1	Normal	0	1	2	2	2	0	2	3	0
Balakumar	72	2	4	21	3.4	3	3	1	1	2	2	2	B/L renal cortical echoes increased	1	2	2	2	1	1	2	2	0
Tamilarasi	36	1	3	33	1.6	2	2	0	0	2	1	1	Normal	1	1	2	2	1	0	2	3	0
Vijayaragavan	5	2	4	9	0.5	0	0	0	0	1	1	1	Normal	0	1	1	1	3	0	1	0	0
Muthulakshmi	12	1	4	27	2	2	2	0	0	2	1	1	Normal	1	1	2	2	7	0	2	3	0
Nandakumar	108	2	4	16	0.8	0	0	1	1	1	1	1	Normal	1	1	1	1	2	0	1	0	0
Balamurugan	144	2	5	13	1.2	1	1	0	0	1	1	2	B/L PCS dilatation noted	1	1	1	1	1	1	2	1	0
Arun Prasath	18	2	4	9	0.5	0	0	0	0	2	2	1	Normal	0	1	1	1	0	0	1	0	0
Karupasamy	96	2	7	7	0.6	0	0	2	0	2	1	1	Normal	0	1	1	1	1	0	1	0	0
Azhaguraja	24	2	9	15	0.8	2	0	0	0	2	1	1	Normal	1	1	1	1	1	0	2	2	0
Lokesh pandi	12	2	5	9	0.5	0	0	0	0	2	2	1	Normal	0	1	1	1	1	0	1	0	0
Lakshmi	96	1	9	23	1	1	1	0	1	1	2	1	Normal	1	1	1	1	1	1	2	2	0
Mahathi	11	1	4	11	0.5	0	0	0	0	2	2	1	HSM+	0	1	1	1	1	0	1	0	0
Sudhan Bala	48	2	3	15	0.6	0	0	1	1	2	1	1	Normal	1	1	1	1	1	0	1	0	0
Tharun	18	2	4	41	0.9	2	1	0	1	2	1	2	Normal	2	1	2	2	1	1	2	3	0
Dharakesh	10	2	5	37	3.6	3	3	0	0	2	1	2	Normal	2	1	1	2	4	1	2	3	0
Maheshwari	132	1	15	28	1	1	1	1	0	1	1	1	Normal	2	1	1	2	1	1	2	2	0
Kishore	48	2	4	9	0.5	0	0	1	1	1	1	1	Normal	0	1	1	1	1	3	1	0	0
Krishnamurthy	2	2	4	15	0.4	0	0	0	0	1	1	1	Normal	0	1	1	1	1	0	1	0	0
Abhinaya	132	1	16	23	7.7	3	3	1	0	2	1	1	B/L Enlarged kidneys with increased echoes	2	2	2	2	1	2	2	2	0
Savipriya	10	1	6	8	0.5	0	0	1	0	1	1	1	Normal	1	1	1	1	1	0	1	0	0
Pon Issakiyammal	2	1	8	7	0.4	0	0	0	0	1	1	1	Normal	0	1	1	1	2	0	1	0	0
Veemaraja	8	2	6	39	0.8	1	1	1	0	2	1	1	Normal	2	1	2	2	1	0	2	3	0
Hasini	15	1	4	8	0.6	0	0	0	0	2	1	1	Normal	0	1	1	1	1	0	1	0	0
surya	120	2	3	11	0.8	0	0	1	0	1	1	1	Normal	0	1	1	1	1	1	1	0	0
siva	42	2	11	27	3.5	3	3	1	0	2	1	2	B/L renal cortical echoes increased	1	2	2	2	1	6	2	2	0
Hoshika	12	1	10	27	0.6	1	0	1	0	2	1	1	Normal	2	1	1	2	1	1	2	2	0
Logesh	48	2	14	23	1	1	1	1	0	1	2	1	Normal	1	1	1	2	1	1	2	2	0

Nandakumar	120	2	14	25	3.4	3	3	1	2	1	1	1	B/L renal cortical echoes increased	2	2	2	2	1	1	2	2	0
Deepadharsini	96	1	6	19	1	1	1	1	0	1	2	1	HSM+	1	1	1	2	1	1	2	2	0
Anusiya	10	1	6	11	0.5	0	0	1	0	2	1	1	Normal	0	1	1	1	3	1	1	0	0
Jaswanth	3	2	6	7	0.4	0	0	0	0	2	2	1	Normal	0	1	1	1	1	1	1	0	0
Mugileshwari	18	1	8	25	1.1	1	3	1	0	2	1	1	B/L renal cortical echoes increased	2	1	1	2	1	1	2	2	0
Dheena	120	2	11	24	1.6	1	2	0	2	1	1	1	Normal	2	1	1	2	4	1	2	2	0
Akshaya shri	5	1	9	25	0.8	1	2	0	2	2	1	1	Normal	1	1	1	1	1	1	2	2	0
Ilakia	2	1	5	33	0.7	1	2	0	0	1	1	1	Normal	2	1	2	2	1	1	2	3	0
Jeyanisha	3	1	5	11	0.5	0	0	1	0	1	1	1	Normal	0	1	1	1	2	0	1	0	0
Kavinraj	4	2	4	37	1	3	3	0	0	1	1	1	B/L increased renal cortical echoes	2	1	2	2	5	1	2	3	0
Kanmani	3	1	4	9	0.4	0	0	0	1	1	1	1	Normal	0	1	1	1	0	0	1	0	0
Reshma	120	1	4	9	0.8	0	0	0	1	2	1	1	Normal	0	1	1	1	0	0	1	0	0
Akshaya	8	1	6	6	0.5	0	0	1	1	1	1	1	Normal	0	1	1	1	1	0	1	0	0
Sabari vasagan	24	2	5	35	0.9	1	2	1	0	2	1	1	Normal	2	1	2	2	1	1	2	3	0
Chandran	132	2	8	21	1	1	1	1	0	2	1	1	B/L increased renal cortical renal echoes	2	1	1	1	1	2	2	2	0
Muthulakshmi	84	1	5	36	8.6	3	3	1	2	2	1	2	B/L renal cortical echoes increased	2	2	2	2	1	1	2	3	0
Balaji	12	2	4	31	1.3	1	1	2	1	2	2	1	B/L renal cortical echoes increased	2	1	2	2	1	1	2	3	0
Kathija	36	1	11	7	0.6	0	0	0	0	1	1	2	Normal	0	1	1	1	1	0	1	0	0
Madhan kumar	4	2	6	9	0.4	0	0	0	0	1	1	1	Normal	0	1	1	1	1	0	1	0	0
Valagurunathan	60	2	10	30	1.2	1	2	1	0	1	1	1	Normal	1	1	1	1	1	1	2	3	0
Karupayi	108	1	6	29	2	3	3	1	2	1	1	1	B/L renal cortical echoes increased	2	1	2	2	1	0	2	3	0
Sivagiri	32	2	7	37	0.8	1	1	1	0	1	1	1	Normal	1	1	2	2	0	0	2	3	0
Mohammed harish	46	2	5	33	8.7	3	3	1	2	2	2	1	B/L renal cortical echoes increased	2	2	2	2	1	0	2	3	0
Sandhiya	96	1	16	34	0.7	0	0	0	0	1	1	1	Normal	2	1	2	2	4	1	1	3	0
Hameetha	32	1	3	40	0.5	0	0	0	0	2	1	1	HSM+	2	1	2	2	1	0	1	3	0
Rakesh	84	2	3	9	0.6	0	0	0	0	1	2	1	GB wall edema+	1	1	1	1	1	0	1	0	0
Selvam	132	2	4	31	0.8	0	0	0	0	1	1	1	Normal	1	1	2	2	1	0	1	3	0
Sugan	8	2	12	37	1.2	1	1	1	0	1	2	1	Normal	1	1	2	2	1	0	2	3	0

Shivani	4	1	6	9	0.3	0	0	1	0	2	1	1	Normal	1	1	1	1	1	0	1	0	0
Mukesh	30	2	14	21	1.1	1	1	1	0	2	1	1	Normal	1	1	1	2	1	0	2	2	2
Mahalaxmi	144	1	7	9	0.9	0	0	1	0	1	1	1	GB wall edema+	0	1	1	1	1	0	1	0	0
Saran anand	2	2	8	8	0.5	0	0	0	0	2	1	1	Normal	0	1	2	2	1	0	1	0	0
Mohammed abi	48	1	8	31	0.9	1	1	1	1	2	1	1	B/L renal cortical echoes increased	1	1	1	1	1	0	2	3	0
Hariharan	42	2	5	29	2.2	3	3	0	0	2	2	2	B/L renal cortical echoes increased	1	2	2	2	1	7	2	1	0
Karthick	96	2	6	11	0.6	0	0	0	0	2	1	1	Normal	0	1	1	1	1	0	1	0	0
B/O karupayi	2	2	4	9	0.3	0	0	0	0	1	1	1	Normal	0	1	1	1	1	0	1	0	0
Harikrishna	4	2	3	13	0.4	0	0	0	0	1	2	1	Normal	0	1	1	1	1	0	1	0	0
Devesh	2	2	3	7	0.5	0	0	0	0	2	2	1	Normal	0	1	1	1	4	0	1	0	0
Hariharan	18	2	4	7	0.6	0	0	1	0	1	1	1	Normal	0	1	1	1	1	0	1	0	0
Vasumathi	15	1	4	7	0.7	0	0	0	1	2	1	1	Normal	0	1	1	1	1	0	1	0	0
Satyapriya	24	1	8	20	0.9	1	1	1	0	1	1	1	Normal	1	1	1	2	1	1	2	2	0
Kajira begum	144	1	4	9	0.9	0	0	0	0	1	2	1	Normal	0	1	1	1	1	0	1	0	0
Ramakrishnan	144	2	6	13	0.6	0	0	1	1	1	2	1	Moderate ascitis	0	1	1	1	1	0	1	0	0
Sasmitha	48	1	7	12	0.6	0	0	1	1	2	1	1	Normal	0	1	1	1	1	0	1	0	0
Deivanayagi	4	1	5	9	0.4	0	0	0	0	1	1	1	Normal	0	1	1	1	2	1	1	0	0
Deva kumar	120	2	11	32	1	1	1	1	0	2	1	1	Normal	2	1	2	2	3	1	2	3	0
saravanan	108	2	9	30	2.8	3	3	1	2	2	1	1	B/L renal cortical echoes increased	2	1	2	2	1	0	2	3	0
Thashika	4	1	4	11	0.5	0	0	0	0	1	1	1	Normal	0	1	1	1	1	0	1	0	0
B/O umadevi	2	2	4	15	0.4	0	0	0	0	1	1	1	Normal	0	1	1	1	1	0	1	0	0
Vignesh	132	2	5	43	3.3	3	3	2	1	1	1	1	B/L renal cortical echoes increased	2	2	2	2	2	1	2	3	0
Surya	96	2	3	36	1.2	1	1	0	0	1	2	1	Normal	1	1	2	2	1	0	2	3	0
Nivetha	108	1	14	26	2	2	2	1	1	1	1	1	normal	1	1	1	2	1	0	2	2	0
Bharani	120	1	17	23	1.8	1	1	1	1	2	1	2	B/L renal cortical echoes increased	1	1	1	1	1	1	2	2	0
Jeyabalaji	108	2	4	9	0.8	0	0	1	1	2	1	1	normal	1	1	1	1	1	0	1	0	0
Dharun	18	2	5	17	0.7	0	0	0	0	2	1	1	Normal	0	1	1	1	1	0	1	0	0
Durgesh	18	2	7	12	0.6	0	0	0	0	2	1	1	normal	0	1	1	1	1	0	1	0	0
Nithya sri	3	1	7	9	0.4	0	0	0	0	1	1	1	Normal	0	1	1	1	3	0	1	0	0

Thangam	120	1	10	19	1	1	1	1	1	2	1	1	B/L renal cortical echoes increased	1	1	1	2	1	0	2	2	0
Leo	96	2	7	9	0.6	0	0	0	0	1	2	1	Normal	0	1	1	1	1	1	1	0	0
Sakthivel	84	2	5	9	0.7	0	0	0	1	1	2	1	Normal	0	1	1	1	1	0	1	0	0
Kabilesh pandi	10	2	11	21	1.3	2	2	1	0	1	1	2	B/L renal cortical echoes increased	2	1	2	2	1	0	2	2	0
Sathiyabala	84	2	7	17	0.5	0	0	0	0	1	1	1	Normal	1	1	1	1	1	0	1	0	0
Arumuga doss	144	2	18	22	2	2	2	1	0	1	1	1	B/L renal cortical echoes increased	2	1	2	2	1	3	2	2	0
Kishore	48	2	5	35	1.4	1	1	1	0	1	1	1	B/L renal cortical echoes increased	2	1	2	2	1	1	2	3	2
saravanakumar	12	2	5	35	2.5	2	2	2	0	1	1	1	B/L renal cortical echoes increased	2	1	2	2	1	1	2	3	0
Sivadharsika	84	1	5	11	0.5	0	0	0	0	1	1	1	Normal	0	1	2	2	1	0	1	0	0
Ravikumar	36	2	5	30	3.4	3	3	1	0	2	1	1	B/L renal cortical echoes increased	2	2	2	2	1	0	2	3	0
Ramya	144	1	15	43	4.4	3	3	2	1	1	1	2	B/L renal cortical echoes increased	2	2	2	2	1	1	2	3	0
Aruna devi	132	1	17	26	3.6	3	3	2	0	1	2	1	B/L renal cortical echoes increased	2	2	2	2	1	1	2	2	0
Rishitha	24	1	4	40	4	3	3	2	2	2	2	1	B/L renal cortical echoes increased	2	2	2	2	1	0	2	3	0
Mohan	10	2	8	15	1.4	1	2	2	0	2	1	1	Normal	1	1	1	1	1	0	2	2	0
Chitradevi	5	1	5	11	0.4	0	0	0	0	1	1	1	Normal	1	1	1	1	1	0	1	0	0
Sai nihitha	2	1	8	27	1.5	3	3	0	0	2	1	1	B/L renal cortical echoes increased	1	1	2	2	1	0	2	3	0
Maria Sebastin	8	2	4	39	4.8	3	3	2	2	1	1	1	B/L renal cortical echoes increased with B/L enlarged kidneys	2	2	2	2	5	1	2	3	0
Prajith	36	2	13	33	3.8	3	3	1	1	2	1	1	B/L renal cortical echoes increased with B/L enlarged kidneys	2	2	2	2	4	1	2	3	0
Praveen Kumar	102	2	4	14	0.6	0	0	0	0	1	1	1	P/O Right Renal Abscess	1	1	1	1	1	1	1	0	0
Ajay	6	2	5	34	1.5	1	2	1	1	1	1	1	B/L acute medical renal disease	2	1	1	2	1	0	2	3	3
Asma	36	1	13	19	3	3	3	1	1	2	1	1	B/L renal cortical echoes increased	2	2	1	2	1	0	2	2	0
Meena	72	1	7	29	0.7	0	0	0	0	2	2	1	B/L renal cortical echoes increased	2	1	2	2	1	0	1	3	0
Gopinath	3	2	7	37	0.3	0	0	0	1	1	1	1	Normal	1	1	1	1	3	0	1	3	0
Subashree	2	1	7	30	0.4	0	0	0	0	1	1	1	Normal	1	1	1	1	1	0	1	3	0
Nandhini	36	1	5	27	0.5	0	0	0	0	1	1	1	Normal	1	1	1	1	1	0	1	3	0

Dharsana	3	1	4	27	0.4	0	0	0	0	1	1	1	Normal	1	1	1	1	2	0	1	3	0
Pooja	84	1	9	26	1.2	1	1	1	0	2	1	1	Normal	1	1	2	2	1	0	2	2	0
Perundevi	48	1	6	39	3.5	3	3	1	2	1	1	1	B/L renal cortical echoes increased	2	2	2	2	1	0	2	3	0
Sivavishnu	24	2	4	8	0.6	0	0	0	0	1	1	1	Normal	1	1	1	1	1	0	1	0	0
Pandimahalakshmi	48	1	8	21	0.9	1	1	1	0	2	1	1	Normal	1	1	2	2	1	0	2	2	0
B/O Nivedha	2	1	5	31	0.3	0	0	0	0	1	1	1	Normal	2	1	1	1	1	0	1	3	0
Pandeeshwaran	60	2	4	7	0.6	0	0	0	0	1	1	1	Normal	1	1	1	1	1	0	1	0	0
Islon	90	2	8	33	2.5	3	3	0	0	2	1	1	Normal	1	1	2	2	2	1	2	3	0
Ponnusamy	132	2	12	22	1.3	1	1	2	0	1	1	1	Normal	1	1	2	2	1	0	2	2	0
Sathuragiri	48	2	10	25	0.5	0	0	0	0	2	1	1	Normal	2	1	2	2	1	0	1	3	0
Ashok	36	2	4	11	0.5	0	0	0	0	2	1	1	Normal	1	1	2	2	1	0	1	0	0
Subramani	144	2	7	9	0.7	0	0	0	0	1	1	1	Normal	1	1	2	2	1	0	1	0	0
B/o Ramu	9	1	8	9	0.4	0	0	0	0	1	1	1	Normal	1	1	2	2	1	0	1	0	0
Elakiya	24	1	3	7	0.5	0	0	0	0	1	1	1	Normal	2	1	2	2	1	0	1	0	0
Aadhi	9	2	6	26	0.5	0	0	0	1	2	2	1	Normal	2	1	2	2	2	0	1	3	0
B/O Anusha	2	2	5	30	0.4	0	0	1	0	1	1	1	Normal	2	1	2	2	0	0	1	3	0
Dharun	48	2	3	21	0.5	0	0	0	0	1	1	1	Normal	2	1	2	2	1	0	1	3	0
Dharshith	2	2	3	25	0.3	0	0	0	0	1	1	1	Normal	2	1	2	2	1	0	1	3	0
Nithish	6	2	3	22	0.6	0	0	0	0	1	1	1	Normal	1	1	2	2	1	0	1	3	0
Harshitha	84	1	3	10	0.6	0	0	0	0	2	1	1	Normal	1	1	2	2	1	0	1	0	0
Yamuna devi	4	1	4	7	0.4	0	0	0	0	1	1	1	Normal	1	1	2	2	1	0	1	0	0
Ponnar	2	2	5	8	0.4	0	0	0	0	1	1	1	Normal	2	1	2	2	1	0	1	0	0
Ramakrishnan	60	2	4	20	0.6	0	0	0	0	1	1	1	Normal	2	1	2	2	2	0	1	3	0
Kamalesh	6	2	6	21	0.5	0	0	0	0	1	1	1	Normal	2	1	2	2	3	0	1	3	0
Sathyapriya	132	1	6	6	0.6	0	0	0	0	2	1	1	Normal	1	1	1	2	1	0	1	0	0
Archana	7	1	8	23	0.9	1	1	1	0	2	1	1	Normal	2	1	2	2	1	0	2	2	0
Subhashni	42	1	13	38	2	3	3	1	1	2	1	1	B/L Renal Cortical echoes increased	2	2	2	2	1	1	2	3	0
Surya	48	2	8	27	0.5	0	0	0	0	1	1	1	Normal	2	1	2	2	1	0	1	3	0
Yashini	4	1	4	22	0.5	0	0	0	0	1	1	1	Normal	1	1	2	2	1	0	1	3	0

Abinash	9	2	14	13	0.5	0	0	0	0	1	1	1	Normal	1	1	1	1	1	0	1	0	0
Devasanjay	108	2	4	8	0.7	0	0	0	0	1	2	1	Normal	0	1	2	2	1	0	1	0	0
Balakishore	48	2	4	9	0.5	0	0	0	0	2	1	1	Normal	0	1	2	2	3	0	1	0	0
Rohith	4	2	3	7	0.5	0	0	0	0	1	2	1	Normal	0	1	1	1	1	0	1	0	0
Antony	84	2	11	7	0.6	0	0	0	0	1	1	1	Normal	0	1	1	1	5	0	1	0	0
Madhupriyan	2	2	8	6	0.4	0	0	0	0	1	1	1	Normal	0	1	1	1	1	0	1	0	0
Dilon	96	2	4	5	0.7	0	0	0	0	2	1	1	Normal	0	1	1	1	1	0	1	0	0
Mahithasri	5	1	4	5	0.5	0	0	0	0	1	1	1	Normal	0	1	1	1	1	0	1	0	0
Karthikeyan	108	2	7	8	0.7	0	0	0	0	1	1	1	Normal	1	1	1	1	1	0	1	0	0
Abhishek	2	2	3	8	0.4	0	0	0	0	1	1	1	Normal	1	1	1	1	1	0	1	0	0
Suhasini	3	1	3	7	0.4	0	0	0	0	2	1	1	Normal	0	1	1	1	1	0	1	0	0
Mugesh	7	2	3	11	0.4	0	0	1	0	1	2	1	Normal	0	1	1	1	1	0	1	0	0
B/O Kaleeshwari	2	2	7	12	0.3	0	0	0	0	1	1	1	Normal	1	1	1	1	1	0	1	0	0
B/O Panchavarnam	3	2	9	10	0.6	0	0	0	1	1	1	1	Normal	0	1	1	1	1	0	1	0	0
Neeleshwari	9	1	4	9	0.6	0	0	0	0	2	1	1	Normal	0	1	1	1	1	0	1	0	0
Srilatha	60	1	3	8	0.6	0	0	0	0	1	2	1	Normal	0	1	1	1	1	0	1	0	0
Keerthigasri	9	1	3	5	0.5	0	0	0	0	1	1	1	Normal	0	1	1	1	1	0	1	0	0
Oviya	12	1	3	5	0.5	0	0	0	0	1	1	1	Normal	0	1	1	1	1	0	1	0	0
Kesavan	9	2	3	8	0.5	0	0	0	0	1	1	1	Normal	0	1	1	1	1	0	1	0	0
Marimuthu	24	2	5	12	0.5	0	0	1	0	1	1	1	Normal	1	1	1	1	1	0	1	0	0
Dhivyaarani	120	1	8	13	0.6	0	0	1	0	1	1	1	Normal	1	1	1	1	1	0	1	0	0
Ramkumar	108	2	4	9	0.7	0	0	1	0	1	1	1	Normal	0	1	1	1	1	0	1	0	0
Kalidas	144	2	7	9	0.7	0	0	1	0	1	1	1	Normal	1	1	1	1	2	0	1	0	0
Ponnathaal	144	1	8	5	0.8	0	0	0	0	2	1	1	Normal	0	1	1	1	1	0	1	0	0
Sasikala	5	1	4	5	0.5	0	0	0	0	1	2	1	Normal	0	1	1	1	1	0	1	0	0
Apoorva	48	1	4	9	0.6	0	0	0	0	1	1	1	Normal	0	1	1	1	1	0	1	0	0
Bhuvana	6	1	4	5	0.3	0	0	0	0	1	1	1	Normal	0	1	1	1	4	0	1	0	0
Deepika	12	1	5	5	0.5	0	0	0	0	1	1	1	Normal	0	1	1	1	1	0	1	0	0
Saranya	72	1	4	5	0.6	0	0	0	0	1	1	1	Normal	0	1	1	1	1	0	1	0	0
Devasri	3	1	3	7	0.5	0	0	1	0	1	1	1	Normal	1	1	1	1	1	0	1	0	0

Sriram	16	2	3	7	0.3	0	0	1	0	1	1	1	Normal	0	1	1	1	1	0	1	0	0
Nathish	72	2	4	6	0.7	0	0	0	0	1	1	1	Normal	0	1	1	1	1	0	1	0	0
Rajesh	3	2	3	6	0.4	0	0	0	0	1	1	1	Normal	0	1	1	1	1	0	1	0	0
Velmurugan	6	2	12	22	2.2	3	3	1	1	2	1	1	B/L Renal Cortical Echoes increased	2	2	2	2	4	0	2	1	0
Mukunthan	12	2	4	11	0.6	0	0	0	0	1	1	1	Normal	1	1	1	1	1	0	1	0	0
Dhivyalakshmi	132	1	5	11	0.7	0	0	0	0	1	1	1	Normal	1	1	1	1	1	0	1	0	0
Muthukumaran	12	2	5	25	1.6	3	3	1	1	2	1	1	B/L Renal Cortical Echoes increased	2	2	2	2	4	1	2	1	0
B/o Anandhi	2	2	6	21	0.4	0	0	0	0	1	1	1	Normal	1	1	1	1	1	0	1	3	0
Sahayam	24	2	4	6	0.6	0	0	0	0	1	1	1	Normal	1	1	1	1	1	0	1	0	0
Karthick	24	2	6	11	0.5	0	0	1	0	1	1	1	Normal	1	1	1	1	1	0	1	0	0
Eswaran	132	2	6	5	0.5	0	0	0	0	1	1	1	not done	0	1	1	1	1	0	1	0	0
Manikandan	4	2	5	4	0.5	0	0	0	1	1	1	1	not done	0	1	1	1	2	0	1	0	0
Gokulakrishnan	42	2	7	10	0.5	0	0	0	0	1	1	1	not done	0	1	1	1	1	0	1	0	0
Karupasamy	18	2	4	10	0.5	0	0	1	0	1	1	1	not done	0	1	1	1	1	0	1	0	0
Boomika	60	1	3	10	0.5	0	0	0	1	2	1	1	Normal	0	1	1	1	1	0	1	0	0
Muthupandi	2	2	6	12	0.5	0	0	0	0	1	1	1	Normal	0	1	1	1	4	0	1	0	0
Pramila	5	1	4	8	0.5	0	0	1	0	1	1	1	not done	0	1	1	1	1	0	1	0	0
Udayasri	22	1	6	7	0.7	0	0	0	1	1	1	1	not done	0	1	1	1	2	0	1	0	0
Chinnadurai	84	2	3	7	0.7	0	0	0	0	2	1	1	not done	0	1	1	1	1	0	1	0	0
Arulraj	48	2	7	6	0.7	0	0	1	1	1	1	1	Normal	0	1	1	1	1	0	1	0	0
Koushikan	2	2	3	7	0.4	0	0	1	1	1	1	1	not done	0	1	1	1	1	0	1	0	0
Hariprasad	108	2	4	5	0.6	0	0	0	0	1	1	1	Normal	0	1	1	1	1	0	1	0	0
Muthusri	30	1	3	8	0.5	0	0	1	0	1	1	1	not done	0	1	1	1	1	0	1	0	0
karthickraj	56	2	8	9	0.5	0	0	0	0	1	1	1	Normal	0	1	1	1	2	1	1	0	0
Ayyanar	10	2	5	7	0.5	0	0	0	1	1	1	1	not done	0	1	1	1	1	0	1	0	0
Padmapriya	48	1	4	6	0.5	0	0	1	0	2	1	1	Normal	0	1	1	1	1	1	1	0	0
Deva	7	2	5	6	0.5	0	0	0	0	1	1	1	not done	0	1	1	1	1	0	1	0	0
B/O Logarani	2	2	5	9	0.5	0	0	0	1	1	1	1	not done	0	1	1	1	5	0	1	0	0
Angelin	36	1	7	11	0.6	0	0	1	0	2	1	1	Normal	0	1	1	1	1	0	1	0	0

Nitheesh Varman	2	2	4	5	0.3	0	0	0	0	1	2	1	Normal	0	1	1	1	1	0	1	0	0
Andisamy	132	2	7	11	0.7	0	0	0	1	2	1	1	not done	1	1	2	2	2	1	1	0	0
Madhunisha	4	1	7	9	0.5	0	0	0	0	1	1	1	not done	0	1	2	2	1	1	1	3	0
Muthuraman	72	2	7	10	0.5	0	0	1	0	1	1	1	Normal	0	1	2	2	1	0	1	0	0
Pavan Keerthana	42	1	4	7	0.5	0	0	0	0	1	1	1	Normal	0	1	1	1	1	0	1	0	0
Gokul Raj	8	2	6	7	0.5	0	0	0	1	1	1	1	not done	0	1	1	1	1	0	1	0	0
Brinda Devi	7	1	9	9	0.5	0	0	0	0	2	1	1	not done	1	1	1	1	1	0	1	0	0
Thasrika	4	1	8	7	0.4	0	0	1	1	1	1	1	not done	0	1	1	1	2	1	1	0	0
Pandy vettaiyan	2	2	5	11	0.4	0	0	0	0	1	1	1	Normal	0	1	2	2	1	0	1	0	0
Vinosh Raj	108	2	4	26	0.6	0	0	0	0	1	2	1	Normal	0	1	2	2	1	0	1	3	0
Jerome	48	2	7	11	0.5	0	0	1	1	2	1	1	not done	1	1	2	2	1	0	1	0	0
Devanayagi	4	1	4	9	0.4	0	0	0	0	1	1	1	not done	0	1	1	1	1	0	1	0	0
Pradeep	2	2	4	9	0.4	0	0	0	0	1	1	1	not done	0	1	1	1	1	0	1	0	0
Kavin Kumar	2	2	7	11	0.4	0	0	0	0	1	1	1	Normal	0	1	1	1	1	1	1	0	0
Anbuselvam	3	2	3	13	0.4	0	0	1	1	2	1	1	Normal	0	1	1	1	1	1	1	0	0
Deepan Raj	10	2	3	6	0.4	0	0	0	0	1	1	1	not done	0	1	2	2	1	0	1	0	0
Angalaparameshwari	72	1	5	6	0.7	0	0	0	0	1	1	1	Normal	1	1	2	2	4	1	1	0	0
Angel Theresa	12	1	6	5	0.5	0	0	1	1	1	2	1	not done	0	1	1	1	1	0	1	0	0
Deepak Shanmugam	2	2	3	6	0.4	0	0	0	0	2	1	1	not done	0	1	1	1	0	0	1	3	0
Bharath Kumar	144	2	4	4	0.8	0	0	1	1	1	2	1	not done	0	1	2	2	0	0	1	0	0
B/O Poomari	5	2	4	8	0.5	0	0	0	0	1	1	1	not done	0	1	1	1	6	0	1	0	0
Subha	132	1	7	7	0.7	0	0	0	0	2	1	1	not done	0	1	2	2	1	0	1	0	0
Amsavalli	24	1	3	6	0.4	0	0	0	0	1	1	1	Normal	0	1	1	1	1	1	1	0	0
Mohammed Iliyaas	11	2	3	7	0.4	0	0	1	1	1	1	1	Normal	0	1	1	1	1	1	1	0	0
Parvathy	9	1	4	4	0.4	0	0	0	0	1	2	1	Normal	0	1	2	2	1	1	1	0	0
Isakiraja	132	2	4	4	0.6	0	0	0	0	1	1	1	Normal	0	1	1	1	1	1	1	0	0
Suresh	72	2	4	4	0.5	0	0	0	0	1	1	1	not done	1	1	1	1	1	0	1	0	0
Karthiga Manju	30	1	8	6	0.5	0	0	1	1	1	1	1	not done	0	1	1	1	1	0	1	0	0
Nagarani	120	1	9	5	0.6	0	0	0	0	2	2	1	not done	1	1	1	1	1	0	1	0	0
Yazhini	12	1	6	9	0.5	0	0	0	0	1	1	1	Normal	1	1	1	1	1	1	1	0	0

Madhumitha	84	1	6	11	0.7	0	0	0	1	1	1	1	Normal	0	1	1	1	1	1	1	0	0
Mahendran	72	2	6	5	0.6	0	0	0	1	1	1	1	Normal	0	1	1	1	1	1	1	0	0
Ramana	132	2	3	5	0.7	0	0	1	1	2	1	1	not done	0	1	1	1	1	0	1	0	0
Prasath	22	2	3	11	0.5	0	0	0	0	1	1	1	Normal	0	1	1	1	1	1	1	0	0
Siddarth	132	2	6	10	0.8	0	0	0	0	1	1	1	Normal	0	1	1	1	0	1	1	0	0
yogan	12	2	4	7	0.6	0	0	0	0	2	1	1	not done	0	1	1	1	0	0	1	0	0
Ranjitha	7	1	3	8	0.5	0	0	1	0	1	2	1	Normal	0	1	1	1	2	0	1	0	0
Supriya	42	1	3	11	0.5	0	0	0	0	2	1	1	Normal	0	1	1	1	0	1	1	0	0
Vasanth Kumar	24	2	4	9	0.5	0	0	0	0	1	1	1	not done	0	1	1	1	1	0	1	0	0
Markandan	2	2	10	6	0.5	0	0	1	0	1	1	1	not done	0	1	1	1	4	0	1	0	0
Krishnaprabha	132	1	4	8	0.7	0	0	0	0	2	1	1	not done	0	1	1	1	1	0	1	0	0
Isaiamuthan	32	2	12	19	0.9	1	1	1	0	2	1	1	GB wall edema+	0	1	1	2	6	0	2	2	0
Dharshan pandi	24	2	3	9	0.7	0	0	0	0	1	1	1	Normal	0	1	1	1	1	1	1	0	0
Karuthapandi	2	2	9	7	0.5	0	0	0	0	2	1	1	Normal	1	1	1	1	0	1	1	0	0
Keshana	11	1	7	18	2.7	3	3	0	0	2	1	2	B/L Hydronephrosis	2	2	1	1	1	2	2	2	0
Sakthi Mari	30	1	6	7	0.5	0	0	0	0	1	1	1	not done	0	1	1	1	1	0	1	0	0
Siva	12	2	3	9	0.5	0	0	1	1	1	1	1	not done	0	1	1	1	0	0	1	0	0
Princy	48	1	3	5	0.6	0	0	0	0	2	1	1	not done	0	1	1	1	0	0	1	0	0
Anbarasan	56	2	4	5	0.7	0	0	0	1	2	1	1	Normal	0	1	1	1	0	1	1	0	0
Inbasri	2	1	4	35	0.4	3	3	1	1	1	1	1	Normal	2	1	2	2	0	1	2	3	0
Raja	120	2	4	7	0.7	0	0	1	0	1	1	1	Normal	1	1	1	1	0	1	1	0	0
Thandeeshwari	96	1	30	7	0.7	0	0	0	1	1	1	1	Normal	1	1	1	1	0	1	1	0	0
B/O Sivaranjani	2	2	3	6	0.5	0	0	0	1	1	1	1	not done	1	1	1	1	3	0	1	0	0
Pavithra	60	1	5	9	0.4	0	0	0	0	1	1	1	not done	0	1	1	1	0	0	1	0	0
Gugasri	48	1	3	11	0.5	0	0	0	1	2	1	1	not done	0	1	1	1	0	0	1	0	0
Santhoshkumar	60	2	3	4	0.6	0	0	0	0	1	1	1	Normal	1	1	1	1	0	1	1	0	0
Kamaludin	18	2	4	4	0.4	0	0	1	0	1	2	1	Normal	1	1	1	1	0	1	1	0	0
Arunthathi	132	1	4	4	0.7	0	0	0	0	2	1	1	not done	0	1	1	1	0	0	1	0	0
Revathy	120	1	8	5	0.6	0	0	0	0	1	1	1	not done	0	1	1	1	0	0	1	0	0
Poornatharan	36	2	3	9	0.6	0	0	1	0	1	1	1	Normal	0	1	1	1	0	1	1	0	0

Vetrivelmurugan	72	2	6	6	0.6	0	0	0	0	2	1	1	Normal	0	1	1	1	0	1	1	0	0
Selvi	60	1	5	7	0.6	0	0	0	0	2	1	1	not done	0	1	1	1	0	0	1	0	
																	1					

Key to Master Chart

Gender	1	Female
	2	Male
AKIN Stage	0	No Acute Kidney Injury
	1	Stage 1
	2	Stage 2
	3	Stage 3
p-RIFLE Classification	0	No Acute Kidney Injury
	1	Risk
	2	Injury
	3	Failure
Sodium Level	0	Normal
	1	Hyponatremia
	2	Hypernatremia
Potassium Level	0	Normal
	1	Hypokalemia
	2	Hyperkalemia
Anemia	1	Nil
	2	Present

Thrombocytopenia	1	Nil
	2	Present
Hypertension	1	Nil
	2	Present
ABG Analysis	0	Not done
	1	Normal
	2	Metabolic acidosis
Renal Replacement Therapy	1	Not required
	2	Required
Mechanical Ventilation	1	Nil
	2	Yes
Inotrope Use	1	Nil
	2	Yes
Blood Culture	0	Not done
	1	No growth
	2	Coagulase Negative Staphylococcus aureus
	3	Klebsiella pneumonia

	4	Non fermentative gram negative bacillus
	5	Staphylococcus aures
	6	E. coli
	7	Others (commensals, enterococcus)
Urine Culture	0	Not done
	1	No growth
	2	Profuse growth of E. coli
	3	Profuse growth of CONS
	4	Growth of proteus
	5	Growth of staphylococcus aureus
	6	Growth of Non fermentative gram negative bacillus
	7	Others (candida)
Outcome	1	No Acute Kidney Injury
	2	Acute Kidney Injury +
Final Outcome	0	Discharged
	1	Partial renal recovery
	2	Complete renal recovery
	3	Death

MADURAI MEDICAL COLLEGE

MADURAI, TAMILNADU, INDIA -625 020

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Course	:	PG in MD., PAEDIATRICS
Period of Study	:	2014 - 2017
College	:	MADURAI MEDICAL COLLEGE
Research Topic	:	**A STUDY ON INCIDENCE, CLINICAL PROFILE AND OUTCOME OF ACUTE KIDNEY INJURY IN CHILDREN ADMITTED TO PAEDIATRIC INTENSIVE CARE UNIT (PICU) OF A TERTIARY CARE CENTRE**

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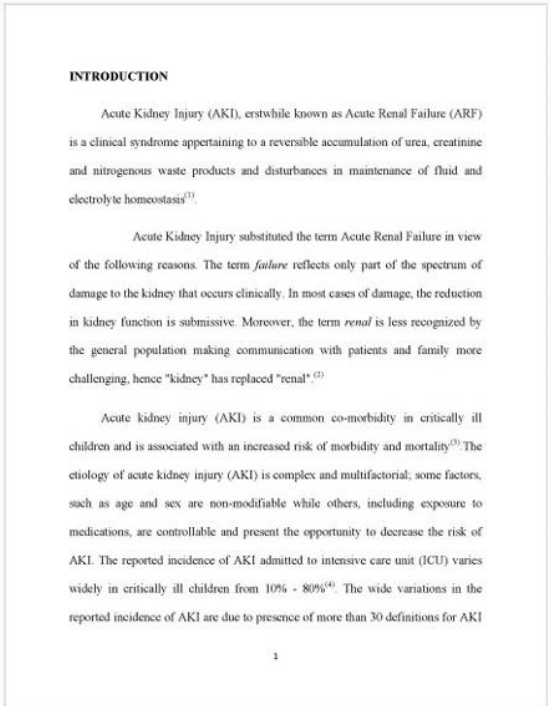

INTRODUCTION

Acute Kidney Injury (AKI), erstwhile known as Acute Renal Failure (ARF) is a clinical syndrome appertaining to a reversible accumulation of urea, creatinine and nitrogenous waste products and disturbances in maintenance of fluid and electrolyte homeostasis[1].

Acute Kidney Injury substituted the term Acute Renal Failure in view of the following reasons. The term *failure* reflects only part of the spectrum of damage to the kidney that occurs clinically. In most cases of damage, the reduction in kidney function is submissive. Moreover, the term *renal* is less recognized by the general population making communication with patients and family more challenging, hence "kidney" has replaced "renal".[2]

Acute kidney injury (AKI) is a common co-morbidity in critically ill children and is associated with an increased risk of morbidity and mortality[3].The etiology of acute kidney injury (AKI) is complex and multifactorial; some factors, such as age and sex are non-modifiable while others, including exposure to medications, are controllable and present the opportunity to decrease the risk of AKI. The reported incidence of AKI admitted to intensive care unit (ICU) varies widely in critically ill children from 10% - 80%[4]. The wide variations in the reported incidence of AKI are due to presence of more than 30 definitions for AKI

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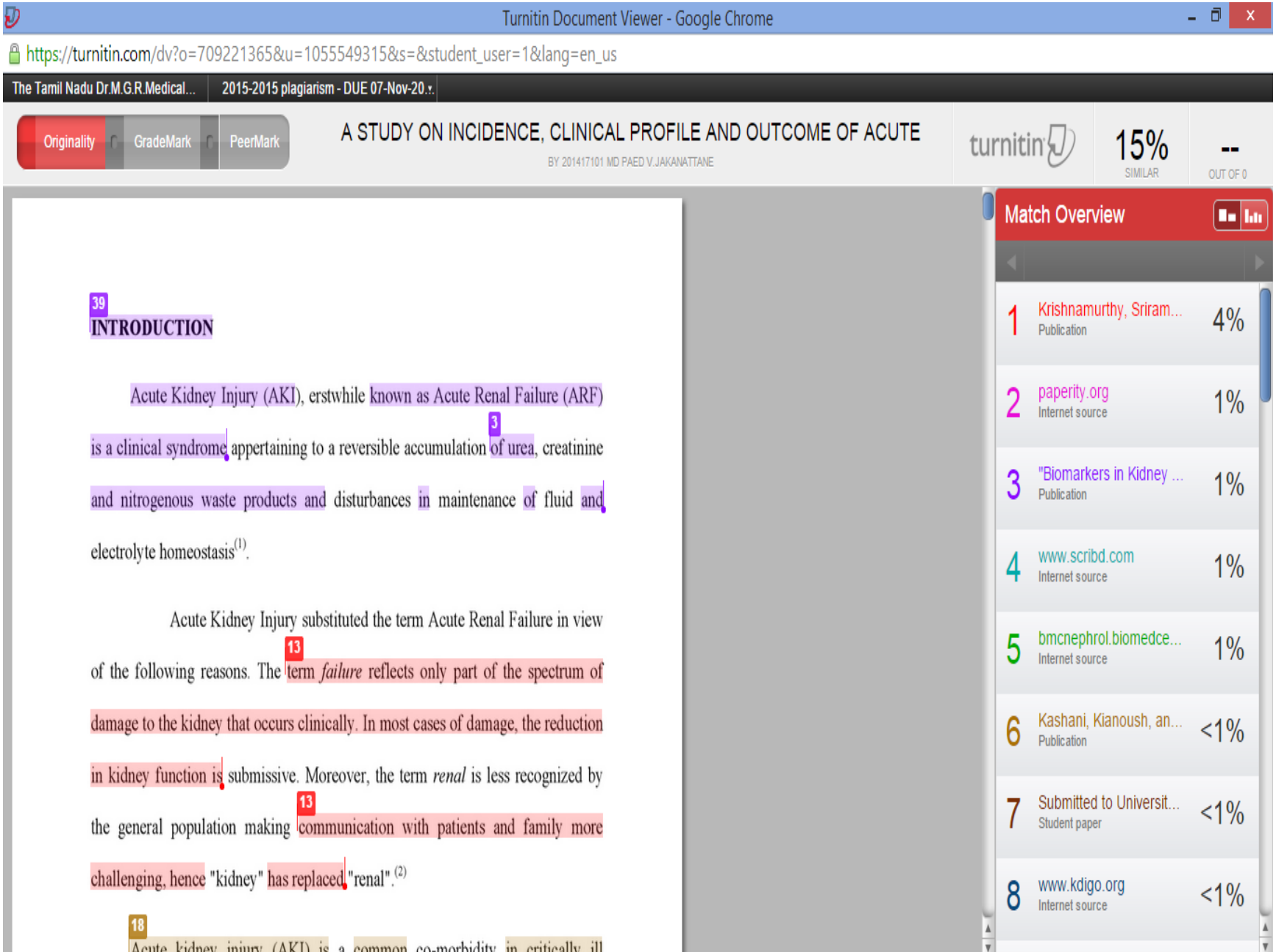
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INTRODUCTION
Acute Kidney Injury (AKI), erstwhile known as Acute Renal Failure (ARF) is a clinical syndrome appertaining to a reversible accumulation of urea, creatinine and nitrogenous waste products and disturbances in maintenance of fluid and electrolyte homeostasis(1).
Acute Kidney Injury substituted the term Acute Renal Failure in view of the following reasons. The term failure reflects only part of the spectrum of damage to the kidney that occurs clinically. In most cases of damage, the reduction in kidney function is submissive. Moreover, the term renal is less recognized by the general population making communication with patients and family more challenging, hence "kidney" has replaced "renal".(2)
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ஆராய்ச்சி ஒப்புதல் படிவம்

தீவிரமான சிறுநீரக காயம் சம்பந்தமான ஆராய்ச்சி.

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எனக்கு இந்த ஆராய்ச்சியின் செயல்முறையும், இந்த ஆராய்ச்சியில் பங்கு கொள்ளவில்லை என்றால் எனது குழந்தையின் சிகிச்சையில் எந்தத் தடையும் இருக்காது என்பதையும், இந்த ஆராய்ச்சியில் இருந்து எந்த நேரத்திலும் வெளியே செல்லலாம் என்பதையும், மேலும் இந்த ஆராய்ச்சி விளக்கங்களும் அதன் நோக்கங்களும் முழுமையாக எனக்கு விரிவுரைக்கப்பட்டது. எனவே இந்த ஆராய்ச்சிக்க குழந்தையின் பெற்றோர் என்ற முறையில் முழு மனதுடன் சம்மதம் தெரிவிக்கிறேன்.

பெற்றோர் கையொப்பம்