“STUDY THE OUTCOME AND PROGNOSIS OF ACUTE KIDNEY INJURY IN PEDIATRIC PATIENTS”

By

DR CHARMEE JOSHI

DISSERTATION SUBMITTED TO THE

SUMANDEEP VIDYAPEETH, PIPARIA, VADODARA.

SBKS MEDICAL INSTITUTE & RESEARCH CENTRE

In partial fulfilment

of the requirements for the degree of

MD

PEDIATRICS

Under the guidance of

DR DULARI GANDHI

PROFESSOR & HOD

DEPARTMENT OF PEDIATRICS

SBKS MEDICAL INSTITUTE & RESEARCH CENTRE

PIPARIA

2015-18

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Dr. Niraj Pandit
Member Secretary
SV Institutional Ethics committee

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ABSTRACT

Title: Study the outcome and prognosis of acute kidney injury in pediatric patients.

Background: Acute renal failure (ARF) is associated with severe morbidity especially in children due to existence of more than 30 definitions of ARF in literature, leading to large variations in the reported incidence and outcome, the term ARF was replaced by AKI to provide uniform definition and classification and standardize patient care[1,2].

Aims and objectives: To study the etiological factors for acute kidney injury and assess associated acute kidney injury as a prognostic factor.

Material and methods: The study was conducted in Pediatric intensive care unit of Department of Pediatrics of SBKS medical college and Dhiraj hospital, Baroda.

Type of study: Prospective study. **Inclusion criteria**: patients aged one month to 17 years admitted to pediatric intensive care unit at Dhiraj hospital Baroda. **Exclusion criteria**: patients with known chronic kidney disease and patients not giving consent. Permission from institutional ethics committee, sbks medical college was taken, following this a well informed parental consent was taken and clinical history and examination was done. In consultation with chief of PICU etiological diagnosis was considered. Serum creatinine levels were estimated at admission and at daily intervals till discharge from PICU. Base line serum creatinine was determined by using backward calculation as recommended by KIDGO for given age within 48 hours of admission staging of AKI was done by pRIFLE and AKIN staging. Data of the

patients were reviewed and assigned pRIFLE and AKIN staging depending on eCrCland serum creatinine respectively.

Results: Of 50 patients included in the study 33(66%) male, 17(34%) female with maximum age of presentation 3.1 to 8 years and mean duration of stay 6.4 days.

Staging of AKI was done with pRIFLE and AKIN staging suggesting pRIFLE is more rapid in picking patients of AKI in I category in comparison to stage 2 of AKIN staging. Mortality risk increased with progression of staging of AKI.

Urine output was not useful in staging of AKI in our study. The commonest etiology for patients with AKI was sepsis 15(30%) of total patients of which 5(33.3%) patients had pneumonia.2nd most common etiology was acute tropical illness accounting total 7(14%) of which 6(85.7%) were of dengue febrile illness and 14.3%(1) with complicated malaria.3(6%) patients had metabolic acidosis, 1(2%) with hyperkalemia, 8(16%) with hypernatremia.32(64%) required inotrope support, 11(22%) were on mechanical ventilation,7(14%) were given renal replacement therapy. Patients on mechanical ventilation had poor outcome with 36.4% expired and 63.6% took DAMA.Of patients who were on renal replacement therapy (42.8%) were on peritoneal dialysis, (57.2%) were on hemodialysis. Overall outcome of patients was 26(52%) discharged,12(24%) went DAMA, 8(16%) referred,4(8%) expired.Of 4 patient expired following were the diagnosis, ARDS with AKI, sepsis with AKI, Subacute intestinal obstruction post op with sepsis,dengue with septic shock with AKI. Of 4 patients 2 were in INJURY and 2 were in failure category on the basis estimated creatinine clearance criteria where as on the serum creatinine criteria, stage 3 had 100% mortality.

Conclusion: There is paucity of AKI related literature and studies in Indianscenario.More studies are required to prove benefits of pRIFLE or AKIN staging. But the common cause for AKI was sepsis, next to it was acute tropical illness. Perhaps control on vectorborne disease may significantly reduce burden of aki. Also pRIFLE helps in picking up patients on AKI early as compared to AKIN staging.

Key words: ARF, AKI, AKIN, pRIFLE, eCrCl, KIDGO, RRT.

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INTRODUCTION

For decades we have used the term "**Acute Renal Failure (ARF)**" to denote a sudden and potentially reversible inability of the kidneys to perform their normal homeostatic functions. It refers to a damage that has already occurred and does not leave any capacity for early detection of "injury" or intervention, to prevent failure. Acute renal failure (ARF) is associated with severe morbidity especially in children due to existence of more than 30 definitions of ARF in literature, leading to large variations in the reported incidence and outcome, the term ARF was replaced by AKI to provide uniform definition and classification and standardize patient care[1,2]. Detection of incidence, etiological profile and outcome of AKI is important for commencement of preventive and therapeutic strategies, identifying patients early to avoid renal replacement therapy as well as comparison of epidemiological studies for improved clinical decision making.

It is recognized that the epidemiology of AKI in developing countries differs from that of the developed world in many important ways [3,4,5]. Whereas in developed regions elderly patients predominate [3], in developing countries, AKI is a disease of the young [6,7] and children [8,9], in whom volume-responsive "prerenal" mechanisms are common [10,11]. In these developing countries, AKI affects predominantly the young and children and mortality is high [12,13]

"**Acute Kidney Injury (AKI)"** was adopted and criteria to define early renal injury and stage different levels of renal injury categorically were sought. AKI may now be defined objectively by the criteria proposed by the AKI Network (AKIN)[1] as an abrupt (within 48 hours) reduction in kidney function, involving:

- an absolute increase in serum creatinine> 0.3 mg/dL from baseline OR
- an increase in serum creatinine> 50%(1.5-fold from baseline) OR
- a reduction in urine output < 0.5 mL/kg/hrfor more than 6 hours).

The RIFLE criteria for Acute Kidney Injury (AKI) were proposed by the Acute Dialysis Quality Initiative (ADQI) Group[2] in 2004 and modified for pediatric use (pRIFLE)[16] in 2007. Further modifications resulted in the AKIN (Acute Kidney Injury Network) criteria in 2007. The major limitations of these scores however, are that they depend on a rise in serum creatinine from baseline and on urine output. Serum creatinine is recognized to rise only late in the evolution of renal damage and additionally at presentation, the baseline (well-state) serum creatinine is often not known. Urine output guidelines are erroneous in detection of AKI in non-oliguric renal failure which is more common in children, particularly neonates. However, until better biomarkers of early renal damage become available, these are the best objective criteria available and importantly, known to have effect on clinical outcome. The above mentioned definitions and scores have been validated subsequently although in mainly adult studies, with definitive proof that clinical outcome gets worse with rising RIFLE and AKI scores.[17]

Considering the limited data available on clinical profile of pediatric AKI from Indian children, the fallacies of retrospective studies and the regional variations in the profile of AKI the present prospective study was conducted.

AIMS AND OBJECTIVES

1. To study the etiological factors for acute kidney injury
2. To assess associated acute kidney injury as a prognostic factor.

REVIEW OF LITERATURE

History:

Richard bright - father of nephrology. Established the relationship between symptoms and pathology of renal failure[22].

Thomas graham- first described the process of dialysis using a hoop from dialyser in 1861[23]

Geoge haasin- first dialysis procedure was performed on human by him in 1924[23]

Kidney transplantation was first reported to be performed for acute renal failure in 1933[23]

Richard ruben- first used peritoneal dialysis as a treatment of end stage renal disease in 1959[24]

First successful peritoneal dialysis antedated the first successful hemodialysis by 7 years.[24]

Scenario of acute kidney injury in india:

The urgent issues in child health in our country encompass control of infection, malnutrition and diarrheal diseases. However, with improvements in public health especially in urban communities, the pattern of childhood mortality is changing resulting in a shift in causes of mortality from infections to other chronic diseases.

The major challenge in pediatrics nephrology still include the management of children with acute kidney injury (AKI) due to severe dehydration, sepsis and toxins.

In 2008, India had population of 1.15 billion people among which 40% of the population was compromised with children <19 years of age. The precise incidence of AKI in children is not well know.([25]) prospective studies from suggest an incidence of 4-6% of AKI in pediatric patients in general wards and upto 40% in picus and renal failure is an important factor in determining morbidity and mortality.

EPIDEMOLOGY

For several decades, clinicians have used the term acute renal failure [ARF] to designate the discrete event of a failed kidney, characterized by a rapid accumulation of blood urea nitrogen and creatinine. However, "ARF" over-emphasizes the failure of kidney function, and does not account for the diverse molecular, biochemical, and structural processes that transpire in an acutely injured kidney, well before the decline in function. Hence proposed the term "acute kidney injury" (AKI). This refers to a broad clinical syndrome encompassing various etiologies and occurring in a variety of clinical settings, with manifestations ranging from subtle biochemical and structural changes, to minimal elevation in serum creatinine, to anuric renal failure [26].

Embracing the spectrum of AKI and the importance of the early pre- clinical damage stage has resulted in several paradigm-shifting outcomes. there has been an improvement in our ability to define, classify, and stage pediatric AKI[26,27].

Historically, progress in pediatric AKI was hindered by the myriad definitions. During the past decade, two major classification systems have emerged (RIFLE and AKIN), based on serum creatinine and urine output criteria. Amodification of the RIFLE criteria was suggested for pediatric use (pRIFLE), substituting serum creatinine values with estimated creatinine clearance [using the Schwartz formula].

Recent pediatric AKI studies have employed the pRIFLE criteria to report on AKI incidence, severity of illness, length of hospital stay, and mortality [26,29-31].

Consequently, the precise incidence, prevalence, and outcomes of pediatric AKI still remain unclear.

Recognizing the need for a single consensus definition and staging system that could be applied to both children and adults, the Kidney Disease: Improving Global Outcomes (KDIGO) group has proposed the following definition for AKI[26]:

Increase in serum creatinine by ≥0.3 mg/dl [≥26.5 μmol/l] within 48 hours; OR Increase in serum creatinine to ≥1.5 times baseline within the prior 7 days; OR Urine volume <0.5 ml/kg/h for 6 hours

Stage	Serum creatinine criteria	Urine output criteria
1	1.5 to 1.9 times baseline or ≥0.3 mg/dl (≥ 26.5 μmol/l) increase	0.5 < mL/kg/hour for 6 to 12 hours
2	2.0 to 2.9 times baseline	<0.5 mL/kg/hour for ≥12 hours
3	3.0 times baseline or increase in serum creatinine to ≥4.0 mg/dl (≥ 353.6 μmol/l) or initiation of renal replacement therapy or in patients <18 years a decrease in eGFR to <35 ml/minute per 1.73 m^2	<0.3 mL/kg/hour for ≥24 hours or anuria for ≥12 hours

eGFR estimated glomerular filtration rate.

The KDIGO staging of AKI is illustrated in table. The KDIGO staging also allows for a child with eGFR <35 ml/min per 1.73 m^2 to be classified as Stage 3, in contrast with the adult criterion of ≥4 mg/dl serum creatinine (which would be unrealistic in infants and young children). The uniform adoption of the KDIGO definition and staging of

AKI holds significant promise for improving our understanding of pediatric AKI epidemiology, and therefore deserves our undivided attention.[32]

The etiology of AKI over past decades has shifted from primary renal disease to multifactorial causes like neonatal hypoxic ischemic injury, post cardiac surgery, increasing use of nephrotoxic agents, septicemia etc. AKI is frequent in picu,affects children who have sepsis and multiorgan failure and is independently associated with high mortality. Children undergoing major cardiac surgery and organ transplant are at risk. Since hospital acquired AKI accompanies other disease processes it complicates management and outcomes and requires aggressive interventions with initiation of renal replacement therapies. In developing world ,AKI is a disease of the young and secondary to a single predominant illness such as gastroenteritis with dehydration, malaria , leptospirosis, hemoltytic uremic syndrome , sepsis, envenomations and red cell enzyme deficiencies. The management in community acquired AKI is conservative and expectant and includes correcting the initial insult, providing supporting fluid and electrolyte therapy and awaiting spontaneous recovery of renal function.

The Acute Dialysis Quality Initiative in Recent developments in the diagnosis of AKI include

1) Use of the RIFLE (R-renalrisk, I-injury, F-failure, L-loss of kidney function, E-end stage kidney disease [ESKD])and AKIN (Acute Kidney Injury Network) criteria and

2) Use of biomarkers of AKI.

RIFLE or AKIN criteria (Table 1)

Small increases of serum creatinine levels in hospitalized patients are associated with

substantial morbidity and mortality[32] The Acute Dialysis Quality Initiative developed a consensus definition and classification of AKI based on creatinine increase and decrease in glomerular filtration rate (GFR) or urine output: the RIFLE criteria. More recently, the RIFLE criteria were modified by the AKIN. The criteria are identical to the first three stages of RIFLE, with the exception of a shorter time frame of AKIwithin 48 hours, and a lower creatinine threshold of greater than 0.3 mg/dL from baseline to peak value (Table 1). The prognostic values of the RIFLE and AKIN criteria have been validated for in-hospital mortality in numerous studies including cardiothoracic surgery, trauma, or critically ill patients and they provide a uniform definition of AKI.[32-35]A new study comparing the diagnostic and prognostic factors ofthe RIFLE and AKIN classifications for AKI after cardiac surgery showed that both are equally useful in diagnosis and accurate in prognosis.[36]

Table I RIFLE and AKIN criteria for diagnosis of AKI RIFLE criteria (within 7 days)AKIN criteria (within 48 hours)

pRIFLE criteria

Letter name	**Estimated creatinine clearance**	**Urine output**
Risk	Decrease by 25%	<0.5 ml/kg/h for 8h
Injury	Decrease by 50%	<0.5 ml/kg/h for 16h
Failure	Decrease by 75% or <35 ml/min/1.73 m^2	<0.3 ml/kg/h for 24 h or anuric for 12 h
Loss	Persistent failure >4 weeks	
End stage	End-stage renal disease (persistent failure >3 months)	

Table II RIFLE and AKIN criteria for diagnosis of AKI

Class	GFR criteria	Urine output criteria
R – risk	Creatinine increase x 1.5 or GFR loss > 25%	0.5 < mL/kg/hour x >6 hours
I – injury	Creatinine increase x 2 or GFR loss > 50%	0.5 < mL/kg/hour x >12 hours
F – failure	Creatinine increase x 3 or GFR loss > 75% or creatinine increase >4 mg/dL (acute increase >0.5 mg/dL)	0.3 < mL/kg/hour x >24 hours or anuria >12 hours
L – loss	Persistent loss of kidney function >4 weeks	
E – ESKD	EKSD > 3 months	—

AKIN criteria (within 48 hours)

Stage	Serum creatinine criteria	Urine output criteria
1	Creatinine increase x 1.5 or creatinine increase >0.3 mg/dL	0.5 < mL/kg/hour x >6 hours
2	Creatinine increase x 2	0.5 < mL/kg/hour x >12 hours
3	Creatinine increase x 3 or creatinine increase >4 mg/dL (acute increase >0.5 mg/dL)	0.3 < mL/kg/hour x >24 hours or anuria >12 hours

ETIO-PATHOGENESIS[37]

AKI has been conventionally classified into 3 categories: prerenal,intrinsic renal, and postrenal

PRERENAL

- Acute gastroenteritis
- Dehydration
- Hemorrhage,blood loss
- Sepsis
- Hypoalbuminemia,fulminant hepatits
- Cardiac failure
- Nephrotic syndrome
- Hepatorenal syndrome

INTRINSIC RENAL

- Glomerulonephritis
- postinfectious/poststreptococcal
- membranoproliferative
- anti–glomerular basement membrane
- Hemolytic-uremic syndrome
- Acute tubular necrosis
- Cortical necrosis
- Renal vein thrombosis
- Rhabdomyolysis
- Acute interstitial nephritis
- Tumor infiltration
- Tumor lysis syndrome
- Vascular renal vein thrombosis
- Renal arterial obstruction
- Prolongation of prerenal insult ,intravascular hemolysis, sepsis,nephrotoxic agents multi organ failure snake bite other envenomations ,falciparum malaria ,leptospirosis.

POSTRENAL

- Posterior urethral valves
- Ureteropelvic junction obstruction
- Ureterovesicular junction obstruction
- Ureterocele
- Tumor
- Urolithiasis
- Hemorrhagic cystitis
- Neurogenic bladder

Prerenal AKI, also called prerenal azotemia, is characterized by diminished effective circulating arterial volume, which leads to inadequate renal perfusion and a decreased gfr. Evidence of kidney damage is absent. Common causes of prerenal AKI include dehydration, sepsis, hemorrhage, severe hypoalbuminemia, and cardiac failure.

If the underlying cause of the renal hypoperfusion is reversed promptly,renal function returns to normal. If hypoperfusion is sustained, intrinsic renal parenchymal damage can develop.

Intrinsic renal AKI includes a variety of disorders characterized by renal parenchymal damage, including sustained hypoperfusion and ischemia. Many forms of glomerulonephritis, including postinfectious glomerulonephritis, lupus nephritis, henoch-schönlein purpura nephritis, membranoproliferative glomerulonephritis, and anti–glomerular basement membrane nephritis, can cause AKI. Ischemic/hypoxic injury and nephrotoxic insults are the most common causes

Of intrinsic AKI in the united states, and are more common with an underlying comorbid condition; most are associated with cardiac, oncologic, urologic, renal, and genetic disorders or prematurity. Severe and prolonged ischemic/hypoxic injury and

nephrotoxic insult lead to acute tubular necrosis (atn), seen most often in critically ill infants and children. Mechanisms leading to ischemic AKI include hypotension/intravascular volume depletion (hemorrhage, third-space fluid losses, diarrhea), decreased effective intravascular volume (heartFailure, cirrhosis, hepatorenal syndrome, peritonitis, abdominal compartment syndrome), vasodilation/vasoconstriction (sepsis, hepatorenal syndrome), renal artery obstruction (thrombosis, embolization,stenosis), intrarenal artery disease (vasculitis, hemolytic-uremic syndrome,sickle cell anemia, transplant rejection), and impaired renal blood flow (cyclosporine, tacrolimus, angiotensin-converting enzyme[ace] inhibitors, angiotensin-receptor blocking agents, radiocontrastAgents).

The typical pathologic feature of atn is tubular cell necrosis, although significant histologic changes are not consistently seen in patients with clinical atn. The mechanisms of injury in atn can include alterations in intrarenal hemodynamics, tubular obstruction, and passive backleak of the glomerular filtrate across injured tubular cells into the peritubular capillaries.tumor lysis syndrome is a specific form of AKI related to spontaneous or chemotherapy-induced cell lysis in patients with lymphoproliferative malignancies. This disorder is primarily caused by obstruction

of the tubules by uric acid crystals. AcuteInterstitial nephritis is another common cause of AKI and is usually a result of a hypersensitivity reaction to a therapeutic agent or various infectious agents.

Postrenal AKI includes a variety of disorders characterized by obstruction of the urinary tract. In neonates and infants, congenital conditions, such as posterior urethral valves and bilateral ureteropelvic junction obstruction, account for the majority of cases of AKI. Other conditions, such as urolithiasis, tumor (intraabdominal lesion or

within the urinary tract), hemorrhagic cystitis, and neurogenic bladder, can cause AKI in older children and adolescents. In a patient with 2 functioning kidneys, obstruction must be bilateral to result in AKI.relief of the obstruction usually results in recovery of renal function, except in patients with associated renal dysplasia or prolonged urinary tract obstruction.

CLINICAL MANIFESTATIONS AND DIAGNOSIS[37]

The clinical features of AKI initially depend on the cause. It is a common event in patients who are critically ill and under multipleInterventions in the picu. Features of sepsis,Hypotension, edema and poor peripheralPerfusion may be present in such patients.

Signs of dehydration may be present in patients who have a history of vomiting or diarrhoea.Children with nephrotic syndrome are prone toHypovolemic episodes during untreated relapses, and if there is concurrent vomiting or diarrhea.

Abdominal pain, vomiting, tachycardia and coolperipheries may be the alerting symptoms. If theIntravascular volume can be restored and bloodPressure corrected, then many of these patientsWill improve.Children with hus too may have precedingDysentery. Patients with agn present withOliguria, hematuria and hypertension. Bright red urine with the presence of blood but not rbcs is indicative of hemoglobinuria as in g6pdDeficiency. Ain may be associated with systemicSymptoms like fever, arthritis, rash and uveitis. Obstructive uropathy may present with a poor Urinary stream, bladder or kidney mass, or with Renal colic. A history of drug intake should be taken in all patients.

Although oliguria is the most common Presenting feature, non-oliguric renal failure

can occur particularly in nephrotoxic AKI and acute Interstitial nephritis. Once intrinsic renal failure occurs, Fluid intake should be restricted to match output & insensible losses. If this balance is not Maintained, fluid overload ensues and the child may develop a raised blood pressure and jvp & peripheral oedema. More serious

Complications include left ventricular failure with pulmonary edema and hypertensive Encephalopathy. Rapidly increasing levels of urea can cause Confusion, anorexia and vomiting. Severe Hyperkalemia can cause cardiac arrhythmias & cardiac arrest. Acidosis can cause Hyperventilation. In the recovery stages, a period of excessive diuresis may occur

DETECTION AND DIAGNOSIS

BIOMARKERS FOR EARLY DIAGNOSIS[37]

In most cases of AKI severe impairment of renal function is present and only supportive management is possible untill recovery takes place. However, if AKI can be anticipated or incipient AKI detected early, preventive measures could be instituted. That situation is important in children undergoing major surgery,organ implantation, multiorgan failure, the neonates with hypoxia and sepsis,. Attempts have been to identify biomarkers, which would indicate renal injury before a rise in serum creatinine.the most promising of these include neutrophil gelatinase-associated lipocalin(ngal),interlukin-18 (il-18),kidney molecule-1 (kim-1) and cystine-c .high urine ngal levels hve been detected very early in children having major surgery who subsequently developed AKI and several other clinical situations complicated by AKI. Il-18 a prroinflamatory cytokine, is detected in urine in ischemic AKI. It is likely that easily measurable methods of these biomarkers would become available,which will help in management of patients,optimisation of intravascular

volume and renal perfusion,particular attention to medications being used and close monitoring of patients will help to prevent and promptly manage AKI.

RENAL ANGINA INDEX[38]

Since there is no reliable marker to determine the severity and outcome of AKI, the use of an index that combines clinical risk factors and signs of renal desease has been proposed to identify patients at risk for AKI. Analogous to the risk of myocardial infarction in a ptient having precordial pain,"renal angina" is defined as the presence of the established risk factors for AKI (e.g.,post cardiac surgery or bone marrow transplantation,sepsis,requiring mechanical ventilation,inotropes) and evidence of renal disease(fluid overload,changes in serum creatinine).the proposed renal angina criteria stratify patient in moderate - risk ,high risk and very high risk according to the underlying clinical condition. For each level of pre existing risk factors, there is a threshhold of evidence of injury that a patient must meet to be considered to have renal angina. Patient in very high risk category are likely to be at the greatest risk for severe AKI and benefit the most from strategies that attenuate renal injury and early renal replacement therapies. Renal angina index =risk of AKI× evidence of AKI. Although the score needs validation , it is likely to be useful in assisting management of patients of AKI.

CLINICAL AND LABORATORY EVALUATION[39]

In a child having oligoanuria, it is important to look for prerenal factors that lead to renal hypoperfusion. History of diarrhea, vomiting,fluid or blood loss should be taken and an assessment of fluid intake in the previous 24 hours made.

In prerenal AKI renal blood flow and glowmerular filtration rate decline , but tubular reabsorption of salt and water continues.thus,there is oliguria with no urine sodium,high urine osmolality,increased plasma urea/creatinine ratio and low fractional excretion of sodium. The rise in blood urea/creatinine ratio occurs because oliguria with decreased tubular flow results in greatly increased urea reabsorption,while that of creatinine is not affected.the level of blood urea (and urea/creatinine ratio)is also elevated when there is increased urea production(e.g., due to excessive protien breakdown, infection or high dose steroid therapy).in atn in a setting of renal hypoperfusion , there is deminished tubular function with a high urine sodium and dilute urine. Several indices help to differentiate parental from established renal failure ;fractional excretion of sodium is most sensitive and reliable. These indices are, however,not useful in patient with non oliguric renal failure, and those recieving diuretics.in prerenal AKI, expansion of intravascular volume leads to improved renal perfusion and increase in urine output.the dehydration is corrected by infusion of 20 to 30ml /kg of an isotonic solution (normal saline or ringer lactate)over45 to 60 minutes. During this period, the vital signs are monitored and caretaken to avoid overhydration. Central venous pressure (cvp) should be meassured to detremine the adequacy of fluid replacement if clinical assessment of hypovolemia is difficult.if urine output is less than 1ml/kg/hr after 2 hrs and there is no sign of intravascular deficit, furosemide is given (1-2mg/kg).if no urine output occurs in the folllowing hour, intrinsic renal failre is suspected.

INVESTIGATIONS[39]

Appropriate investigations are performed to confirm the diagnosis . Peripheral blood smear examination showing features of microangiopathic hemolysis, thrombocytopenia and reticulocytosis indicates hus . Throat culture for streptococci, aso titer and other streptocoocal antibodies ,and serum c3 level should be examined in patients suspected to have acute gn. In glomerular and vascular disease, urinary protein is elevated (>1g/m2/24 hr) along with red cell and casts. Eosinophils in the urinary sediment suggest interstitial nephritis. The presence of renal tubular epithelial cells ,cellular debris and muddy brown tubular cells cast support the diagnosis of atn.

Ultrasonography is the ideal imaging tool in renal failure because of its non dependence on renal function. It allows visualization of pelvicalyceal system and assessment of renal size and screens for structural anomalies and calculi. Renal biopsy may be required in an occasional patient with AKI ,the indications being : (a) patients in whom the etiology of AKI is not identified, particularly in the context of a systemic disease; (b) unremitting AKI lasting longer than 2 to 3 weeks, where the biopsy may be useful for diagnosis(e.g., crescentic gn), or assessing the extent of renal damage and outcome (tubular or cortical necrosis); (c) suspected drug induced AKI in a patient receiving therapy with a potentially nephrotoxic drug (eg; renal transplant recipient treated with cyclosporine).

MANAGEMENT

Preventive measures Studies from different geographic regions of Nigeriademonstrated that the most common cause of AKI in children was volume depletion and that the AKI was due to preventable cause . Since dialytic resources were

scare, the mortality rate in these studies was quite high . Thus, on a global scale, the prevention of AKI is likely to have a larger impact on mortality rates than other measures.[40,41]

The principles of management include treatment of life threatening complications, maintenance of fluid and electrolyte balance and nutritional support. Specific management of the underlying disorder should be instituted . Patients with urinary tract obstruction need to be managed urgently. Definitive surgery is performed after complications of AKI have been treated.

Immediate treatment of complications[39,42]In a child with AKI and oligoanuria, immediate attention is directed towards detection and management of life threatening complications that already exist. These include hyperkalemia, pulmonary edema, hypertensive emergencies, severe acidosis and anemia. Clinical evaluation involves measurement of blood pressure ,fundus examination and a search for signs of congestive heart failure ,fluid overload, acidosis and anemia. Immediate investigations include estimation of blood levels of hemoglobin ,urea, creatinine, electrolytes and bicarbonate. Electrocardiogram should be done to detect potassium toxicity and x-ray film of the chest for pulmonary edema.

Supportive care[43]In a child with AKI in whom serious complications are absent or have been adequately treated, supportive care is instituted. Management is based on close attention to intake of fluid and electrolytes, provision of proper nutrition , prevention, and treatment of infections, careful monitoring and dialysis.

Fluid and electrolyte imbalance[43]AS per arvind bagga,Fluid and electrolyte in a patient with AKI is crucial and must be meticulously regulated. The daily fluid requirements amount to insensible water losses (300ml/m2),urinary output and extrarenal fluid losses. Insensible fluid loss should be replaced with 10 percent glucose solution. Urine output should measured without resorting to catheterization. Urinary losses and those from extrarenal sources should be replaced with 0.3 to 0.45 percent saline in 5 percent glucose. Potassium containing fluids must not be given. It is preferable to administer the required amount of fluid by mouth. If there is persistent vomiting, intravenous route may be necessary.

Fluids : amount given equals insensible losses plus urine volume and other losses.

Nutrition:protein intake of 1 to 2g/kg & energy of 60 to 70 cal/kg and micronutrients. Maintenance of nutrition is crucial in hypercatabolic states or if AKI is prolonged.Prevent infections; treat with appropriate antibiotics in correct dosage. Weigh daily accurately; prevent weight gain. Monitor urine output; investigations are necessary.

NUTRITION SUPPORT:

Nutritional supportParenteral nutrition compared to other modalities of nutritionin critically ill patients has not been proven to be of benefit.With multiorgan dysfunction uremia is known to acceleratecatabolism due to a variety of factors including acidosis, alteredcounter-regulatory hormonal status, and insulin resistance.A prospective double-blind study randomizing 30 patientswith AKI to three isocaloric regimens: glucose alone, glucoseplus essential amino acids, or glucose plus essential andnon-essentialamino acids has been performed.[44] All patientsremained in negative nitrogen balance and remained so throughoutthe study and no difference in recovery of renal

function orsurvival between treatment groups was noted. In patients oncontinuous RRT, despite an intake of 2.5 g/kg/day of protein,these patients remained in negative nitrogen balance.[45]

In recent reviews of the topic, the following recommendationswere made: 1) protein and non protein calories shouldbe provided to meet calculated energy expenditures and at arate not to exceed 1.5 g/kg/day protein intake, 2) nutritionalrecommendations should not be different from that ofcritically ill patients as a whole, 3) total parentral nutritionshould be administered only to patients who are severelymalnourished or patients expected to be unable to eat forgreater than 14 days, and 4) enteral feeding is the preferredmeans of nutritional supplementation.[46]

Patients with AKI have increased metabolic needs and are usually catabolic . Adequate nutritional support is desirable with maximization of caloric intake, but limited by the volume restriction necessary during the oliguric phase. A diet containing 1 to 2g/kg protein in infants and 0.8 to 1.2 g/kg in older children, and 60 to 70 cal/kg should be given. The latter requirement can be met by adding liberal amounts of carbohydrates and fats to the diet . Once dialysis is initiated , dietary protein ,fluid and electrolyte intake is increased. Supplements of vitamins (thiamine, riboflavin, pyridoxine ,folic acid, cynacobalamin, ascorbic acid) and micronutrients should be provided. If oral intake is inadequate, parenteral administration is considered.

MANAGEMENT OF INFECTIONS[39,42]

Patients with AKI are more susceptible to develop infectious because of depressed immune system induced by azotemia and concomitant malnutrition , and invasive procedures. Various infections (respiratory and urinary tract , peritonitis and septicemia) are the immediate cause of death in majority of patients. All procedures should be performed with aseptic techniques, intravenous lines carefully watched, and skin puncture sites cleaned and dressed. Oral hygiene should be ensured. Devitalized tissue and collection of blood should be removed. Prolonged catheterization of bladder is avoided. Sepsis is suggested by hypothermia ,persistent hypotension, hyperkalemia and a disproportionate rise of blood urea compared to creatinine. The patient should be frequently examined for infections, which may present without fever.if infection is suspected, appropriate specimens are taken for culture and antibiotics started.

USE OF MEDICATIONS[39,42]

Medications that increase severity of renal damage or delay recovery of function, e.g., aminoglycosides, radiocontrast media, nsaids and amphotericin b, should be avoided. Agents that reduce renal perfusion, e.g., ace inhibitors and indomethacin are not recommended in patients with renovascular disease or following renal transplantation.

The dose and dosing interval of antibiotics should be modified depending on the severity of renal injury . The dose of medications should be adjusted based on residual renal function, in order to avoid toxic accumulation of drugs and their metabolites, and to avoid worsening the AKI. When glomerular filtration rate <50 percent of normal, most drugs excreted by the kidney will require modifications in

dose or scheduling. Patients with marked oliguria and rising creatinine should be assumed to have a gfr <10 ml/min and medications dosed accordingly, since there is a delay in serum creatinine to rise to steady state levels. A number of medications are cleared by dialytic therapies, with clearance varying among various modalities. While assessment of drug levels is useful, monitoring is often not available, and there is limited ability to determine if these levels are approaching toxicity.

Diuretics and dopamine receptor agonistDiuretics and 'renal-dose' dopamine are commonly used toprevent or limit AKI. There have been several clinicalstudies using mannitol, diuretics, and 'renal-dose' dopaminefor AKI[47-54]. The stimulation of urine output easesmanagement of AKI, but conversion of oliguric to nonoliguricAKI has not been shown to alter the course of renalfailure[47]. Furosemide may increase the urine flow rate todecrease intratubular obstruction and will inhibit Na-KATPase,which will limit oxygen consumption in alreadydamaged tubules with a low oxygen supply. In a randomizedcontrolled trial, two groups of adult patients with AKIrequiring dialysis were given furosemide therapy or placebo;diuresis was achieved in a significantly shorter time in thegroup that received furosemide than in the group that hadreceived placebo [47]. However, there was not a differencein the number of dialysis sessions, time on dialysis, orpatients'survival [47]. In patients who do respond to diuretictherapy with an increase in urine output, continuousinfusions may be associated with less toxicity than bolusadministration[48]. A retrospective study actually demonstratedthat the use of diuretics in AKI was associated withadverse outcomes [49]. Since high doses of furosemide cancause ototoxicity, continued use in individual patients withAKI needs to take into consideration the risks and potentialbenefits or lack of benefits.

The use of ‘renal-dose’ dopamine (0.5 μg/kg per minuteto 3-5 μg/kg per minute) to improve renal perfusionfollowing an ischemic insult has become very common inintensive care units. While dopamine increases renal bloodflow by promoting vasodilatation and may improve urineoutput by promoting natriuresis, there have been nodefinitive studies to demonstrate that low doses ofdopamine are effective in decreasing the need for dialysisor improve survival times in patients with AKI[50-54]. Infact, a placebo controlled randomized study of low doses ofdopamine in adult patients demonstrated that low doseswere not beneficial and did not confer clinically significantprotection from renal dysfunction [50]. Other studies havedemonstrated that ‘renal-dose’ dopamine is not effective inthe therapy of AKI, and one study demonstrated that lowdoses worsened renal perfusion and renal function [52].

Three separate meta-analyses have shown no benefit ofdopamine in AKI[51, 53, 54]. Fenoldopam is a potent,short-acting, selective, dopamine-1 receptor agonist thatdecreases vascular resistance while increasing renal bloodflow[55]. A recent meta-analysis of 16 trials of fenoldopamconcluded that therapy with fenoldopam decreased theincidence of acute kidney injury, decreased the need forrenal replacement therapy, decreased ICU stay and decreasedthe number of deaths from any cause [56].

Fenoldopam has been used in a few children with acutekidney injury, including two children receiving therapywith a ventricular-assist device as a bridge to cardiactransplantation; therapy with fenoldopam was thought toavoid the need for renal replacement therapy in one child[57]. Additional studies utilizing fenoldopam need to beperformed on children with acute kidney injury.

Therapies to decrease injury and promote recoveryWhile there is no current specific therapy to prevent renalinjury or promote recovery in human ATN, severalpotential

therapies are being studied, and future managementof AKI may also include antioxidant, anti-adhesionmolecule therapy and the administration of vascularmediators or mesenchymal stem cells to prevent injuryand/or promote recovery [58-62].

Diuretics: Diuretics may be useful in volume overload in AKI. Nonoliguricpatients with AKI are better than oliguric patients.[55]However conversion of oliguria to nonoliguria has not beenshown to decrease mortality. Diuretics have not been shownto prevent AKI or improve outcomes in AKI.[56] In fact, in amulticenter retrospective study of 552 patients with AKI inthe ICU, diuretics were used in 326 patients (59%) at the timeof nephrology consultation and with adjustment for relevantcovariates and propensity scores, diuretic use was associatedwith a significant increase in the risk of death or nonrecoveryof renal function.[57]

As per arvind bagga There is no evidence that diuretics improve renal function or the prognosis of intrinsic renal failure. Diuretics may improve urine output but not the glomerular filtration rate. They may be useful in instances where a high urine flow is required to prevent intratubular precipitation as with intravascular hemolysis, hyperuricemia and myoglobinuria. Furosemide can cause ototoxicity, interstitial nephritis, hypotension and persistent patent ductus arteriosus in the newborn.

Calcium channel blockers, antioxidants, thyroxine, peptide growth factors and cytokines have been used to attenuate renal injury or enhance recovery of renal function in experimental models. However, none of these agents has a place in the management of AKI.

MONITORING: The child with AKI should be closely monitored. Accurate record of intake and output and weight should be maintained. Laboratory tests are done depending upon the patient's condition, progression of AKI and presence of complications. Careful physical examination is done as often as necessary.

Biochemical derangement with persistent acute kidney injury

Hyponatremia : Serum sodium level below 130 meq/l may be present initially in AKI or develop later during management. In both instances, hyponatremia is the result of excessive fluid administration rather than sodium loss. Sodium administration is not required to correct AKI associated hyponatremia, and restriction of fluid intake is advised. Sodium administration is hazardous in patients with excessive body water and may cause hypertension and congestive cardiac failure. However, severe hyponatremia when associated with sensorial alteration or seizures requires prompt correction. Serum sodium concentration should be increased by 5 to 10 meq/l over 30 to 90 minutes by infusion of 3 percent saline; 12 ml/kg of this solution will raise serum sodium by 10 meq/l.

Hyperkalemia: Serum potassium levels should be measured every 12 to 24 hr and ecg obtained as necessary. Hypercatabolic states and extensive tissue breakdown lead to increase in blood potassium levels. Potassium rich foods are avoided and hyperkalemia controlled by administration of potassium exchange resins. Sodium or calcium polystyrene sulfonate is administered orally with sorbitol or lactulose, or by enema. One g resin/kg body weight reduces serum potassium by 1 meq/l. Side effects include anorexia, nausea, hypokalemia and sodium retention. Patients with oligoanuria and hyperkalemia usually require dialysis.

Hyperphosphatemia, hypermagnesemia and hypocalcemia: Once AKI has persisted for few days, hyperphosphatemia (6-8 mg/dl) and hypermagnesemia may occur. The former is aggravated if a hypercatabolic state or rhabdomyolysis is present. Hypocalcemia usually occurs but is mostly asymptomatic. Tetany or convulsions may be precipitated by excessive alkali therapy. Calcium gluconate or carbonate is given at a dose of 30 to 50 mg/ kg elemental calcium. A diet containing low phosphate is given. If serum phosphate levels are above 7 mg/dl, administration of aluminum hydroxide or calcium based agents that chelate dietary phosphate may be useful. Dialysis is considered if dietary modification and phosphate binders fail to reduce levels of serum phosphate.Hyperuricemia is common in AKI. If uric acid levels are below 15 mg/dl, no treatment is required.

Diuretic phase in acute kidney injury: The clinical course of uncomplicated AKI (acute tubular necrosis) is characterized by 3 phases: oligoanuria, diuresis and recovery. The duration of oliguria may be a few hours to several weeks, but in uncomplicated atn it usually lasts for 5 to 10 days. During the diuretic phase there is a progressive rise in urine output which may reach 2 to 3 l per day. Such high output is often due to excessive replacement of fluids and overhydration. A profound diuresis may be seen following relief of obstruction in postrenal AKI. During the diuretic phase the levels of blood urea and creatinine may continue to increase and decline only after several days. Urine has low levels of urea and creatinine and large amounts of electrolytes. Complications such as infections, gastrointestinal bleeding, convulsions and electrolyte abnormalities (e.g., hypokalemia) are frequent. The diuretic phase should be managed by replacement of urinary output with 0.45 percent saline and potassium if necessary. In uncomplicated am oligoanuria may last for 7 to 10 days at the end of which the urine output progressively increases. Such patients

may require only a single dialysis. If AKI is prolonged beyond 2 to 3 weeks multiple dialysis sessions may be required. In these cases, maintenance of nutrition and prevention of infections are crucial, since the patient may die of infection and inanition before recovery of renal function.

DIALYSIS :**Renal replacement therapies**[63]

In the presence of AKI complications such as hypervolemiaeg, acute pulmonary edema or large cumulative positive fluidbalance, hyperkalemia, metabolic acidosis (pH less than 7.1)and uremic symptoms (persistent nausea and vomiting, pericarditis,neuropathy, or an otherwise unexplained decline inmental status) dialysis should be considered as a mainstaytherapy. Modalities of RRT include intermittent hemodialysis(IHD), continuous renal replacement therapies (CRRTs),and hybrid therapies, such as sustained low-efficiencydialysis (SLED). Despite these varied techniques, mortalityin patients with AKI remains greater than 50% in severely illpatients. It is possible that variations in the timing of initiation,modalities, and/or dosing of RRT may affect clinicaloutcomes, particularly survival.

The main considerations when starting a patient with AKIon dialysis are the following: 1) timing of initiation of dialysis,2) the modality of dialysis, and 3) dose of dialysis.

Urgent indications for dialysis include severe or persistent hyperkalemia, congestive heart failure, pulmonary edema, severe acidosis and neurological abnormalities (secondary to uremia or hyponatremia) . However, these features are usually late manifestations of severe kidney dysfunction, and it is important that renal replacement therapy be initiated prior to the appearance of these complications. Dialysis must begin early to prevent occurrence of metabolic complications and

improve outcomes. It is important to evaluate the clinical situation and anticipate the course of AKI in each case, depending upon the severity of renal injury and urine output. Early dialysis should be performed in hypercatabolic states, e.g., extensive trauma, burns and infections. Data from the pediatric prospective continuous renal replacement therapy registry suggests that fluid overload (>15%) should be prevented, since it is an independent risk factor for mortality. Fluid overload is calculated as follows:

Percent fluid overload = [[fluid in (liters) - fluid out (liters)] / admission weight (kg)]*100

All dialyses modalities (peritoneal dialysis pd, hemodialysis hd and continuous hemofiltration) can be used to ensure equivalent solute clearance and ultrafiltration. Hd and pd are equally effective in the management of AKI. The choice of procedure depends on (i) age and size of the patient, (ii) cardiovascular status, (iii) availability of vascular access, (iv) integrity of peritoneal membrane and abdominal cavity, and (v) expertise available. In patients who are hemodynamically stable, intermittent therapies are as effective as crrt, while in patients who are hemodynamically unstable, crrt is the modality of choice.

Most children with AKI who need dialyses are treated with pd, which is easier to perform and requires minimal equipment and infrastructure. Moreover, the gradual rate of fluid removal and correction of metabolic derangement provided by pd is often an advantage in critically sick children or small infants. Pd is also an effective treatment for severe hyperphosphatemia. Children with cardiovascular compromise tolerate this procedure better than hd. The wide range of acute pd catheters available and their ease of insertion make pd technically feasible even in the smallest infant.

CONTINUOUS RENAL REPLACEMENT THERAPIES

Technological advances have led to availability of several forms of continuous renal replacement therapies (crrt). These require expensive equipment and expertise. Continuous hemofiltration employs a filtration cartridge with a high ultrafiltration coeeficient that permits efficient removal of excess fluid in the form of protein free plasma. The patients's cardiac output provides the driving force for the hemofilter. The ultrafiltered volume is replaced by a balanced electrolyte solution . Crrt therapies require a vascular access with large size catheters to allow blood inflow and outflow. Systemic heparanization is necessary .

Continuous arteriovenous hemofiltration (cavh) and its modifications such as pump assisted continuous venovenous hemofiltration (cvvh) and arteriovenous hemodiafiltration (cavdh) has been useful to remove excess fluid and solutes. Cvvh is particularly useful for the management of AKI with fluid overload and pulmonary edema, and in patients with major surgical procedures, burns, heart failure, and septic shock, especially when the hd and pd are not possible. Patients with tumor lysis syndrome, hyperammonemia, ingestion of dialyzable toxins and hypercatabolic states are better managed with hd or crrt. Continuous hemofiltration provides an efficient and smooth control of ultrafiltered volume and gradual correction of metabolic abnormalities , even in haemodynamically unstable patients with multiorgan failure.

OUTCOME

Specific therapies for AKI Although the mortality in patients with AKI has declinedbetween 1988 and 2002,[64] the mortality of AKI in the ICUremains high.[65] Most interventional therapeutic trials in AKIeg, furosemide,[66] dopamine and furosemide,[67] anaratide,[68,69]insulin-like growth factor-1,[70] and fenoldopam[71] have

failedin humans. A possible reason for the failure of interventionaltrials in AKI is the dependence on serum creatinineto diagnose AKI. Alterations in serum creatinine may lag24–48 hours behind actual changes in GFR.[64] Ideally, inthe future, early diagnosis of AKI using urine or plasmabiomarkers may allow early initiation of specific therapieseg, erythropoietin to treat or prevent worsening of AKI.

Optimal management and dialysis can reverse the derangements caused by AKI.The outcome depends chiefly on the underlying condition. The prognosis is favourable in atn from volume depletion; intravascular hemolysis,diarrhea related hus ,acute interstitial nephritis, and drug or toxins related AKI, when complicating factors are absent. In crescentic gn , atypical hus ,and AKI associated with sepsis, multiorgan failure and following major cardiac surgery, the prognosis is less satisfactory.

Long term outcome: Although complete recovery usually occurs in uncomplicated AKI, many patients have a significant risk of developing chronic renal damage. That especially applies to patients with seemingly mild d+ (typical) hus, and AKI secondary to medications and toxins , and perinatal hypoxia. Patients with AKI should be observed for several years with regular monitoring of blood pressure ,urinalysis and estimation of blood levels of creatinine.

Prevention of acute kidney injury: Several conditions that cause AKI can be prevented. Important measures include prompt rehydration therapy in acute diarrhea , judicious use of nephrotoxic drugs, careful observation of patients receiving antimalarial drugs and maintainence of proper hydration for patients undergoing diagnostic procedures with radiocontrast media. Forced diuresis with use of allopurinol is effective in preventing AKI in patients with tumor lysis syndrome.

MATERIALS AND METHODS

The study was conducted in Pediatric intensive care unit of Department of Pediatrics of SBKS medical college and Dhiraj hospital Baroda.

Study period: 31/12/2015 to 30/6/2017.

Settings: Pediatric Intensive Care Unit of Dhiraj hospital, a tertiary level hospital Baroda.

Type of study: Prospective study

Inclusion criteria: Patients aged one month to 17 years admitted to pediatric intensive care unit at Dhiraj hospital Baroda

Exclusion criteria: patients with known chronic kidney disease and patients not giving consent.

Methodology:

Permission from institutional ethics committee, SBKS medical college was taken, following this a well informed parental consent was taken and clinical history and examination was done.

In consultation with chief of PICU etiological diagnosis was considered. Serum creatinine levels were estimated at admission and at daily intervals till discharge from PICU. Serum creatinine was estimated on ERBA XL systems by creatinine enzymatic method. Base line serum creatinine was determined by using backward calculation as recommended by KIDGO for given age within 48 hours of admission.

NORMAL VALUES FOR GLOMERULAR FILTERATION RATE (AS PER SCHWARTZ FORMULA)[18]

AGE	**MEAN GFR±SD(ml/min/1.73m^2)**
Neonate <34 weeks gestation	
1 week	15.3±5.6
2-8 weeks	28.7±13.8
>8 weeks	51.4
Neonate>34 weeks gestation	
1 week	41±15
2-8 weeks	66±25
>8weeks	96±22
Children and adolescents	
2-12 years (males and females)	133±27
13-21 years (males)	140±30
13-21 years (females)	126±22

NORMAL VALUE FOR SERUM CREATININE[(19)]

Age	**Range (mg/dl)**
Cord	0.6-1.2
New born	0.3-1.0
<3 years	0.17-0.35
3-5 years	0.26-0.42
5-7 years	0.29-0.48
7-9 years	0.34-0.55
9-11 years	0.35-0.64
11-13 years	0.42-0.71
13-15 years	0.46-0.81
Adult male	0.7-1.3
Adult female	0.6-1.1

The above mentionednormal values of glomerular filteration rate values given for age in pediatric population. The standardized gfr (estimate creatinine clearance) values

were also taken as the baseline GFR for purpose of staging of patients according to pRIFLE criteria.

Schwartz formula: GFR=δL/Scr.

GFR: glomerular filtration rate in ml/min/1.73m^2

L: LENGHTH

Scr: serum creatinine in milligram per deciliter

δ: constant of proportionality is age and sex dependent.

Values for δ :

- For preterm infants:0.34
- Term infants :0.45
- Children and adolescent girls:0.55
- Adolescent boys:0.70

Data of the patients were reviewed and assigned to AKIN staging based on serum creatinine and pRIFLE as per eCrcl during stay in PICU.

pRIFLE criteria

Letter name	**Estimated creatinine clearance**	**Urine output**
Risk	Decrease by 25%	<0.5 ml/kg/h for 8h
Injury	Decrease by 50%	<0.5 ml/kg/h for 16h
Failure	Decrease by 75% or <35 ml/min/1.73 m^2	<0.3 ml/kg/h for 24 h or anuric for 12 h
Loss	Persistent failure >4 weeks	
End stage	End-stage renal disease (persistent failure >3 months)	

Table I RIFLE and AKIN criteria for diagnosis of AKI

Class	**GFR criteria**	**Urine output criteria**
R – risk Stage 1	Creatinine increase x 1.5 or GFR loss > 25%	0.5 < mL/kg/hour x >6 hours
I – injury Stage 2	Creatinine increase x 2 or GFR loss > 50%	0.5 < mL/kg/hour x >12 hours
F – failure Stage 3	Creatinine increase x 3 or GFR loss > 75% or creatinine increase >4 mg/dL (acute increase >0.5 mg/dL)	0.3 < mL/kg/hour x >24 hours or anuria >12 hours
L – loss	Persistent loss of kidney function >4 weeks	
E – ESKD	EKSD > 3 months	—

STATISTICAL ANALYSIS:

In study time of 18 months, 393 patients were screened and 50 were enrolled. Descriptive statistics like frequency, percentage, mean, SD used to summarize data. Pearson's Chi-Square test is used to check association between two qualitative variables and One Way ANOVA test is used to compare more than two group of means. Significance level is set at 5%.

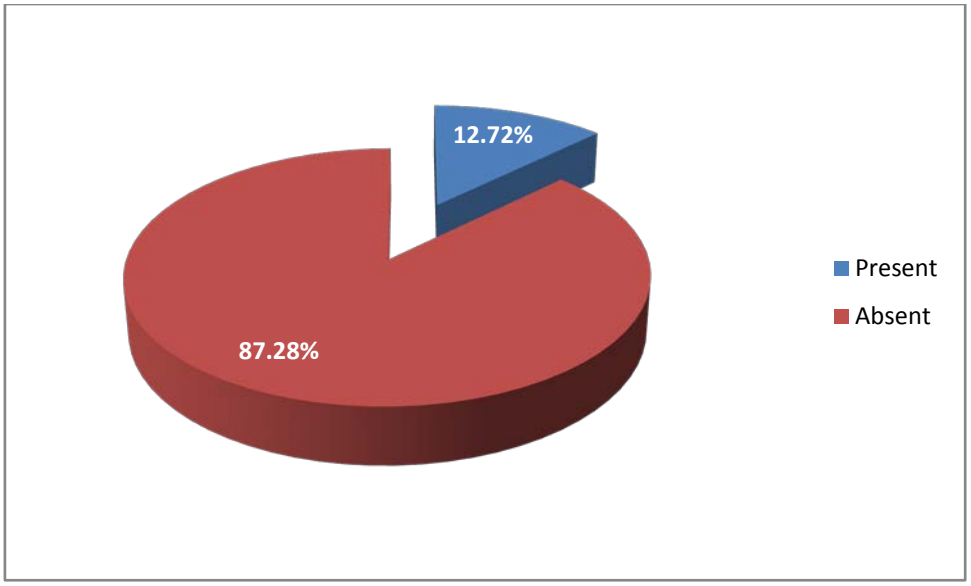
RESULTS AND DISCUSSION

Incidence of AKI against PICU admissions:

Total patients 393 out of which 50 were included in our study who met inclusion criteria, suggesting incidence of 12.72% of AKI in critically ill patient admitted in PICU at dhiraj hospital. Maximum stage of AKI was found in pRIFLE: **F**:27 pts (54%),**I**: 21 pts (42%) :**R**: 2pts (4%)and in AKIN staging stage 3 :36pts (72%) stage 2 :19pts (8%) and stage 1 :5 pts (10%.) within 48hours of admission.

TABLE 1: INCIDENCE OF AKI

	No of patients	**Percentage**
Present	50	17.12
Absent	343	82.88
Total	393	100.0



Poonam Mehta et al[72]from the departments of pediatrics all India institute of medical sciences, New Delhi, found that incidence of (36.1%) of AKI in critically ill patients and

(9.0%) patients who were not critically ill developed AKI (p <0.001); the maximal stage of AKI was stage 1 in 48(65.8%) patients, stage 2 in 13 (17.8%) and stage 3 in 12(16.4%) patients with AKIN grading.

Another study by **Sriram krishnamurthy et al**[73] suggested that the incidence of AKI was 5.2 % in the pediatric wards and 25.1 % in the PICU of a tertiary hospital in southern india.

These studies had taken first day creatinine as baseline and in those cases where either it was missed or not available it was calculated using MDRD formula. In our study baseline serum creatinine and base line glomerular filteration rate is taken from standard range for serum creatinine and GFR given by KIDGO. Based on s.creatinine AKIN staging was done and in reference to estimated creatinine clearance and urine output pRIFLE staging was done, since few patients had non oliguric AKI and few were on furosemide, urine output was not useful in our study.

SEX AND AGE DISTRIBUTION AGAINST NO. OF CASES:

Of 50 patients included in the study 33patients(66%) were male ,17(34%) were female, with maximum age of presentation being 3 years to 8 years and mean duration of stay was 6.4 days.

TABLE 2: AGE DISTRIBUTION AGAINST STUDY POPULATION.

		Sex		Total
		Male	**Female**	
Age group	1 m to 3 yrs	9	7	16
		56.2%	43.8%	100.0%
	3.1-8 yrs	11	2	13
		84.6%	15.4%	100.0%
	8.1-12 yrs	4	3	7
		57.1%	42.9%	100.0%
	12.1-17 yrs	9	5	14
		64.3%	35.7%	100.0%
Total		33	17	50
		66.0%	34.0%	100.0%

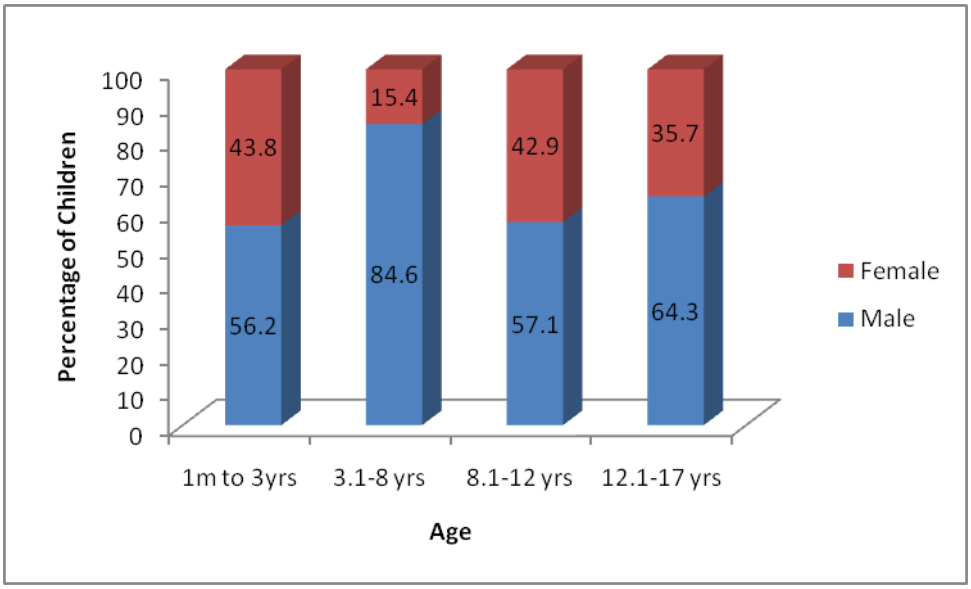
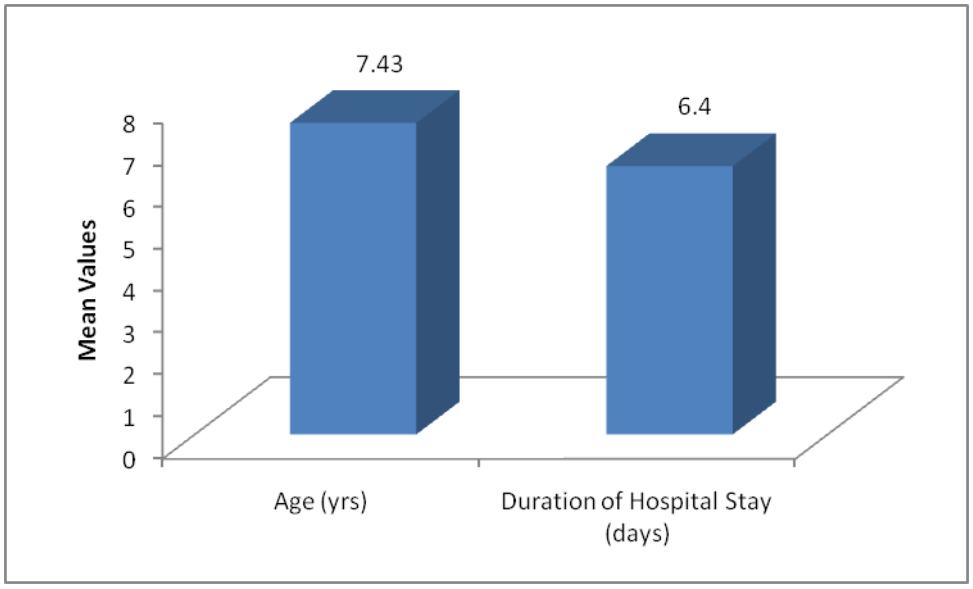


TABLE 3: MEAN DURATION OF HOSPITAL STAY:

	N	Minimum	Maximum	Mean	Std. Deviation
Age	50	.17	17.00	7.4283	5.65966
Duration of stay in days	50	1.00	21.00	6.4000	4.41241

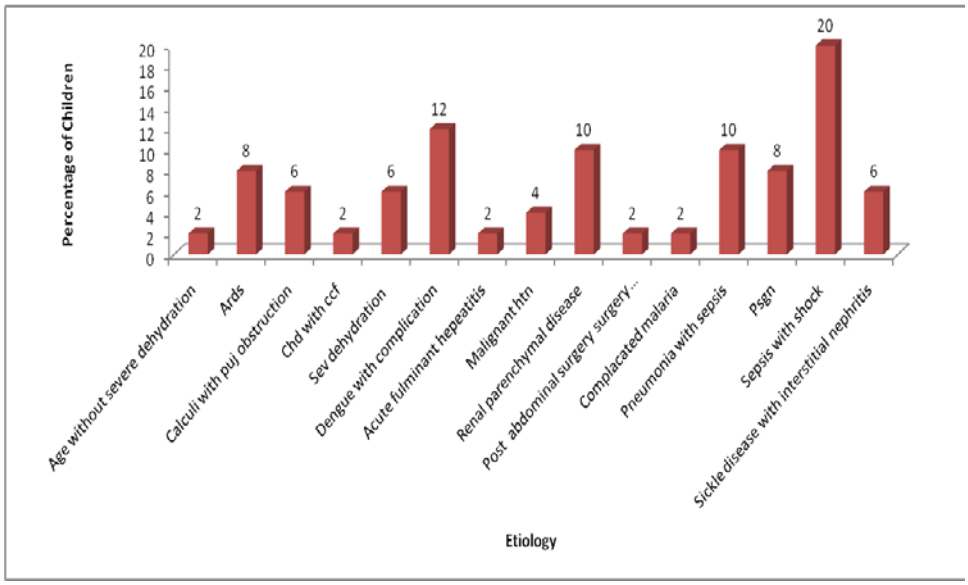


The median age of patients with AKI was 42 months and 54.2% were boys in study by **Sriram krishnamurthy et al**[73].Another study by **A Ackan-Arikan et al**[74] showed median age of 4 years with 56% boys, which is comparable to our study. There was no statistical significance noted in age and sex paramters. (pvalue:0.400)

TABLE 4: ETIOLOGY OFAKI:

Etiology	Frequency
AGE	1(2%)
ARDS	4(8%)
Calculi with PUJ obstruction	3(6%)
Chd with CCF	1(2%)
Sev dehydration	3(6%)
Dengue with complication	6(12%)
Acute fulminant hepeatitis	1(2%)
Malignant HTN	2(4%)
Renal parenchymal disease	5(10%)
Post abdominal surgery (ileal cycst)	1(2%)
Complacated malaria	1(2%)
Pneumonia with sepsis	5(10%)
PSGN	4(8%)
Sepsis with shock	10(20%)
Sickle disease with interstitial nephritis	3(6%)
Total	50(100%)

Our study showed overall incidence of AKI with most common etiology being sepsis accounting 15 case(30%) of total cases, of which 5 case(10 %) constituted pneumonia. Tropical acute illness was the second most common cause with 7(14%) incidence of which 6(12%) was dengue febrile illness and 1(2%) complicated malaria. Post streptococcal glomerulonephritis(8%) and ARDS(8%) being 3rd most common cause leading to AKI, interstitial nephritis (6%),renal parenchymal disease(10%), severe dehydration (6%),renal calculi (6%).



The study conducted by **Sriram krishnamurthy et al**. In southern India showed that AKI occurred in association with sepsis (55.4 %), acute glomerulonephritis (16.9 %), cardiac disease (4.8 %), envenomations (4.2 %) and hemolyticuremic syndrome (3.6 %). Pneumonia constituted 26.1 % of the sepsis. Tropical febrile illnesses (dengue, scrub typhus, enteric fever, cholera, tuberculosis, malaria and leptospirosis) constituted 15.6 % of children with AKI. Commonest etiology being sepsis and glomerulonephritis which is comparable to our study.

Al duzova,aysin et al[75] Study suggested that hypoxic/ischemic injury and sepsis were the leading causes in both age groups, with the percentage of patients with hypoxic/ischemic injury being significantly higher in newborns than in children aged > 1month (43.5 vs. 20.4%; **p**<0.001). Since we did not take children <1month in our study, hypoxic ischemic injury was not the cause of AKI in our study. Approximately 15% of patients in both groups had a low fluid intake in the absence of acute gastroenteritis. Glomerular diseases were the third most common cause of AKI (15.4%) in children aged >1 month (hemolytic uremic syndrome, **n**=18; acute post-streptococcal glomerulo-nephritis, **n**=7;membranoproliferative glomerulonephritis, (**n**=4), followed by acute gastroenteritis (11.9%).AKI secondary to drugs/exogenous toxins was seen in 9.1%.among the 41 patients with malignancy, sepsis (26.8%) and drug toxicity (26.8%) were the leading causes of AKI,followed by ischemic

injury (17.1%) and acute tumor lysis syndrome (9.8%).

These studies showed that sepsis and glomerular disease are the ommonest causes leading to AKIin children which is comparable to our study but drug induced nephrotoxicity, snake envenomations and HUS inspite of being common etiology found in reference studies, no such cases were found during our study. Our study was comparable to above mentioned two studies by **Al duzova,aysin bakkaloglu et al[75] and sriram krishnamurthy et al**[73]. As sepsis, tropical febrile illness and glomerular disease were the commonest etiology in our study also.

TABLE 5(A):STAGING OF AKI WITH AKIN.

AKIN (staging)	Frequency
Stage 1	4(8%)
Stage 2	9(18%)
Stage 3	37(74%)
Total	50(100%)

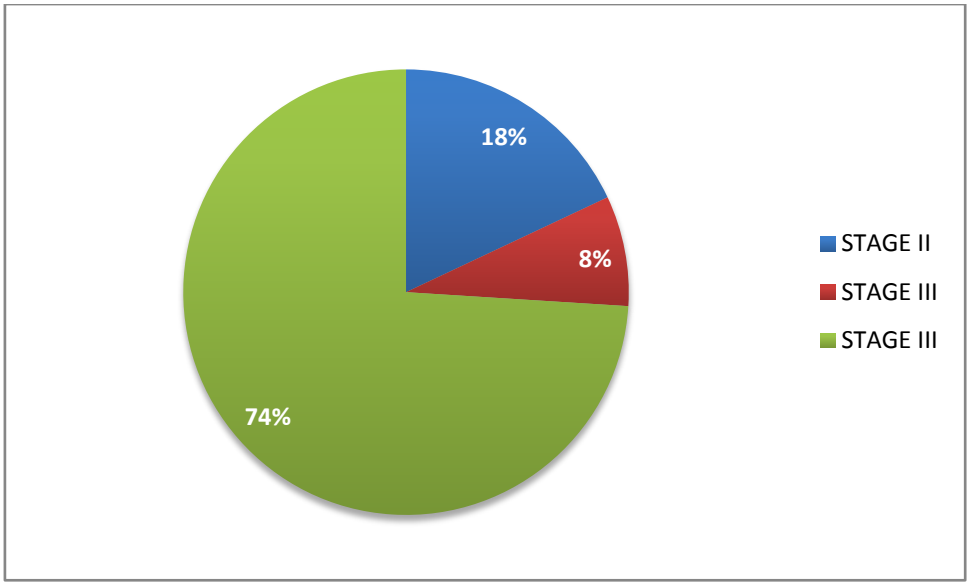


TABLE 5 (B):STAGING OF AKI WITH pRIFLE

pRIFLE STAGING	Frequency
RISK-**R**	2(4%)
INJURY-**I**	21(42%)
FAILURE-**F**	27(54%)
TOTAL	50(100%)

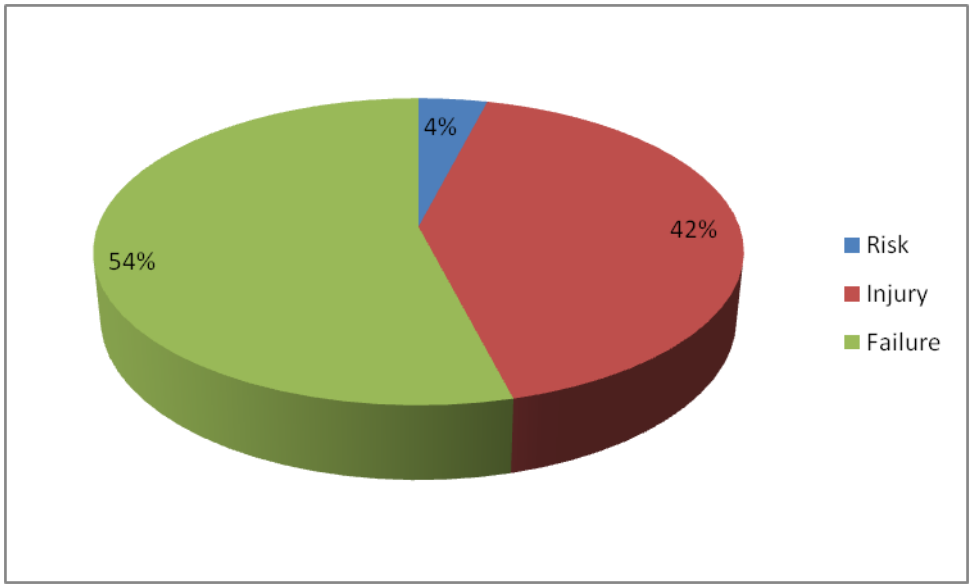


TABLE 5 (C): STAGING OF AKI WITH URINE OUTPUT CRITERIA

Urine output	Frequency
Non oliguric AKI	3(6%)
Risk	4(8%)
Injury	19(38%)
Failure	24(48%)

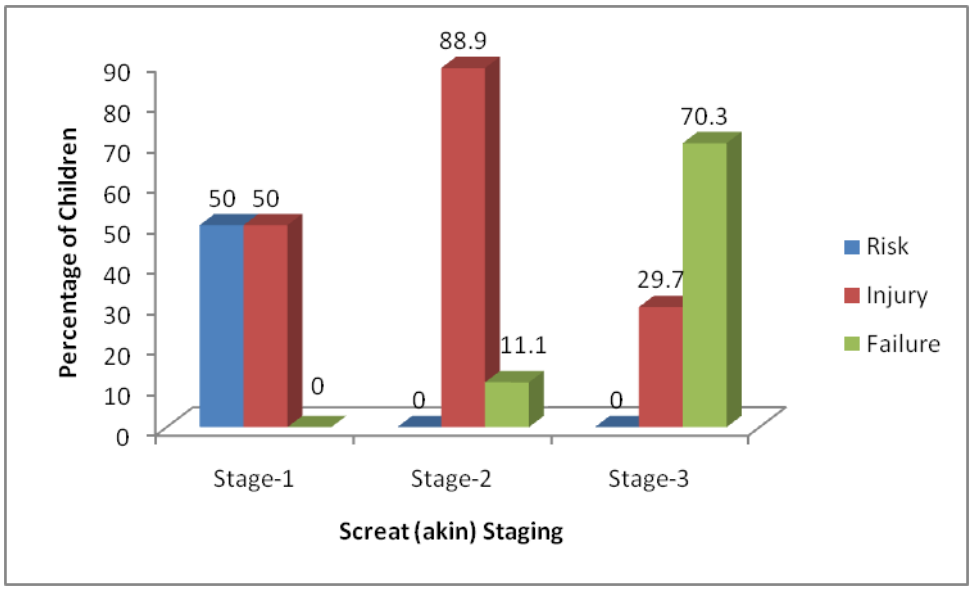
Serum creatinine and eCrCl are the two main criteria used for classification of AKIwithin 48 hours of admission based on AKIN and pRIFLE classification respectively. As per AKIN 4 (8%) patients were in Stage 1, 9(18%) were in stage 2 and 37(74%) were in stage 3 category. Where as in pRIFLE staging 2 (4%) patients were in **R** ,21(42%) in **I** and 27(54%) in **F** category. Based on this study AKIN is good in picking patients in stage 1 category where aspRIFLE is far better in picking patients in **I** category. Staging by pRIFLE is equivalent to AKIN staging where **R**is equivalent to stage 1,**I** to stage 2, and **F** to stage 3.

Urine output was also used in staging but 3 patients were excluded since they had adequate urine output hence non oliguric AKI and few patients were on dialysis and few on Lasix which made difficult to classify on the basis of urine output. Hence urine output in criteria for estimation of AKI is not reliable as per our study.

TABLE 6: COMPARISON OF AKIN STAGING VERSUS pRIFLE

		pRIFLE STAGING		
	Total (n=50)	**Risk (n=2)**	**Injury (n=21)**	**Failure (n=27)**
AKIN STAGING	1(n=4)	2	2	0
		50.0%	50.0%	.0%
	2(n=9)	0	8	1
		.0%	88.9%	11.1%
	3(n=37)	0	11	26
		.0%	29.7%	70.3%

	Kappa value	**P-value**
Measure of agreement	0.463	<0.001



Comparison of AKI with AKIN and pRIFLE staging showed statistical significance (p value<0.001). which suggests pRIFLE **I stage** helps in early detection ok AKI as compared to AKIN staging.

Poonam Mehta et al[72]Two recently proposed classifications, the RIFLE [1] and AKIN[2] criteria have been validated as diagnostic and prognostic tools in critically ill adult patients with AKI[14],[15]. Studies in critically sick children, using the RIFLE [20] or its pediatric modification, pRIFLE[29, 14] show that the incidence of AKI varies from 10% to 58%.Based on the former, **Schneider, et al**[20]

A ackran[74]stated Ninety-seven patients (81.5%) fulfilled pRIFLEcr criteria and 65 (54.6%) fulfilled pRIFLEuop criteria at some time during the study period. All patients requiring dialysis attained their pRIFLEcr max before initiation of dialysis. Patients with pRIFLEmax I or F during admission had over twice the mortality than patients with pRIFLEmax R or controls (21 vs 8%, respectively,**Po** 0.05). Patients with pRIFLEmax F also had over twice the mortality rate of the rest of the cohort (25.8% forPRIFLEmax F vs 10.9% for all others, **P.** 0.03). The current prospective study shows pRIFLE to serve well to both classify pediatric AKI epidemiology and reflect the course of AKI in children admitted to the PICU. AKI classification using pRIFLE criteria revealed that AKI is very common in critically ill pediatric patients and is associated with significant morbidity

TABLE 7: ETIOLOGY VERSUS OUTCOME OF AKI.

Etiology	Outcome				Total
	Expired	**Dama**	**Discharged**	**Referred**	
Acute fulminant hepatits	0	1	0	0	1
	.0%	100.0%	.0%	.0%	100.0%
AGE	0	0	1	0	1
	.0%	.0%	100.0%	.0%	100.0%
ARDS	1	3	0	0	4
	25.0%	75.0%	.0%	.0%	100.0%
Calculi with puj obs	0	1	2	0	3
	.0%	33.3%	66.7%	.0%	100.0%
Chd with CCF	0	0	1	0	1
	.0%	.0%	100.0%	.0%	100.0%
Dengue with complication	1	0	5	0	6
	16.7%	.0%	83.3%	.0%	100.0%
HTN	0	0	1	1	2
	.0%	.0%	50.0%	50.0%	100.0%
Malaria with complic	0	0	1	0	1
	.0%	.0%	100.0%	.0%	100.0%
Post operative abdominal surgery	0	0	1	0	1
	.0%	.0%	100.0%	.0%	100.0%
PSGN	0	0	2	2	4
	.0%	.0%	50.0%	50.0%	100.0%
Pneumonia	0	3	1	1	5
	.0%	60.0%	20.0%	20.0%	100.0%
SCD with interstitial nephritis	0	0	3	0	3
	.0%	.0%	100.0%	.0%	100.0%
Severe dehydration	0	0	2	1	3
	.0%	.0%	66.7%	33.3%	100.0%
Sepsis with shock	2	3	3	2	10
	20.0%	30.0%	30.0%	20.0%	100.0%
Renal parenchyma dis	0	1	3	1	5
	.0%	20.0%	60.0%	20.0%	100.0%
Total	4	12	26	8	50
	8.0%	24.0%	52.0%	16.0%	100.0%

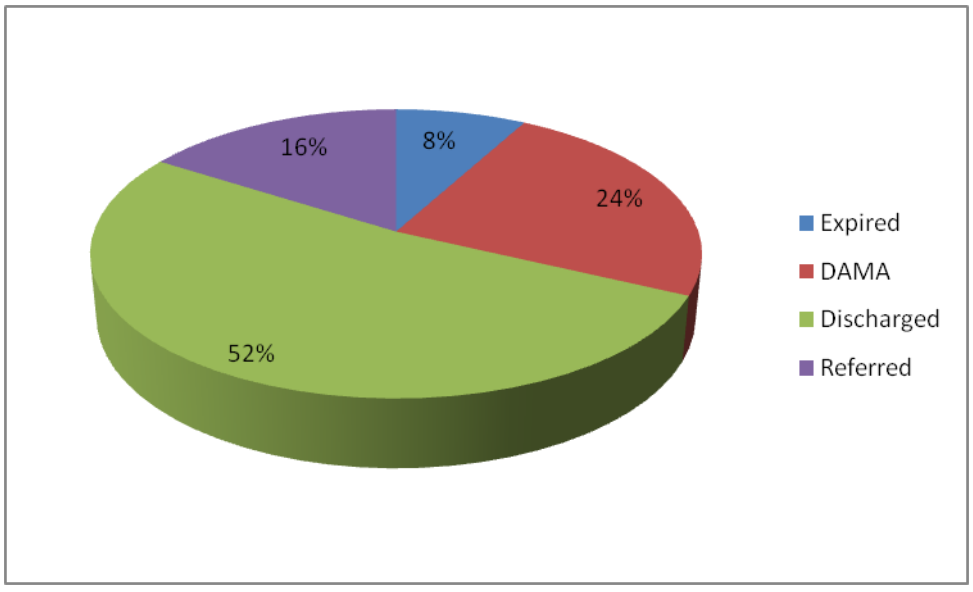
TABLE 8: ELECTROLYTE IMBALANCE WITH AKI:

Electrolyte imbalance	No. of patients
Metabolic acidosis	3
Hyperkalemia	1
hypernatremia	8

Amongst 50 patients in our study 3 had metabolic acidosis, 1 with hyperkalemia and 8 patients had hypernatremia. This was not significant possibly due to small sample size.

TABLE 9: OVERALL OUTCOME OF AKI IN STUDY POPULATION

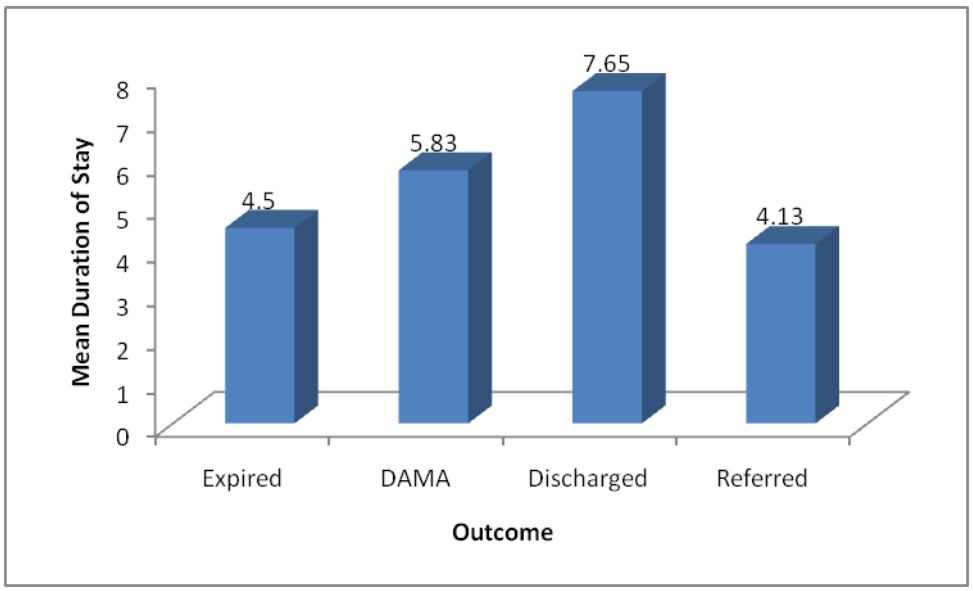
Outcome	Frequency	Percent(%)
Expired	4	8.0
Dama	12	24.0
Discharged	26	52.0
Referred	8	16.0
Total	50	100.0



of total 50 patients in our study 26(52%) were dischaged without any complication, 12(24%) were dischaged against medical advice, 8(16%) referred to higher center and 4(8%) expired.

TABLE 10: DURATION OF STAY VERSUS OUTCOME.

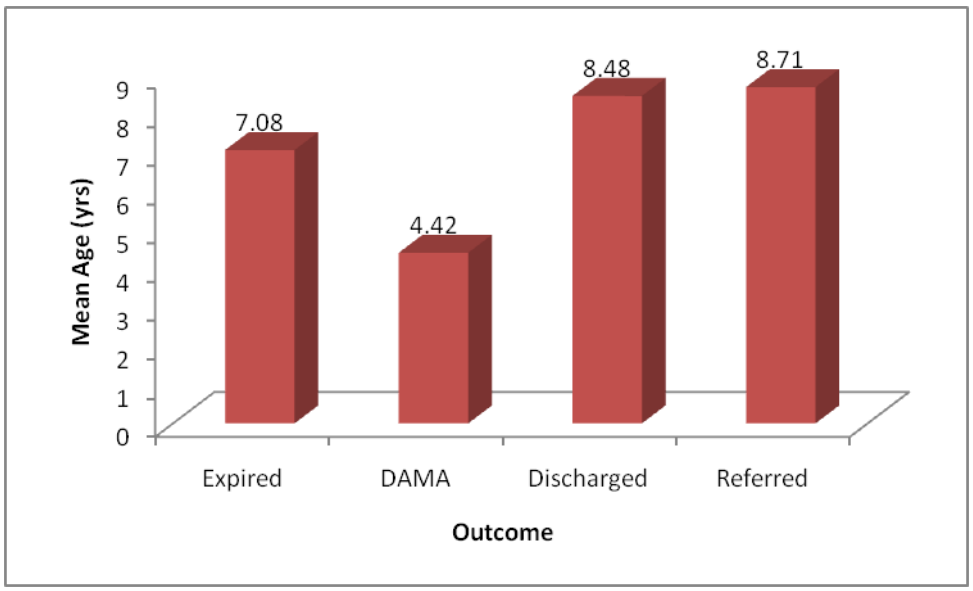
Outcome	N	Mean duration of stay	Std. Deviation	Std. Error	95% confidence interval for mean		Minimum	Maximum
					Lower bound	Upper bound		
Expired	4	4.5000	2.88675	1.44338	-.0935	9.0935	1.00	8.00
Dama	12	5.8333	5.92120	1.70930	2.0712	9.5955	1.00	21.00
Discharged	26	7.6538	3.84648	.75436	6.1002	9.2075	2.00	18.00
Referred	8	4.1250	3.22656	1.14076	1.4275	6.8225	1.00	11.00
Total	50	6.4000	4.41241	.62401	5.1460	7.6540	1.00	21.00



Mean duration of stay of discharged patients was 7.6 days,DAMA patient was 5.8 days, expired patient was 4.4 days and referred was 4.1 days.

TABLE 11: MEAN AGE OF PRESENTATION VERSUS OUTCOME.

Outcome	N	Mean age	Std. Deviation	Std. Error	95% confidence interval for mean		Minimum	Maximum
					Lower bound	Upper bound		
Expired	4	7.0833	7.28964	3.64482	-4.5161	18.6828	.33	16.00
Dama	12	4.4167	5.03096	1.45231	1.2201	7.6132	.17	15.00
Discharged	26	8.4776	5.34195	1.04764	6.3199	10.6352	.67	17.00
Referred	8	8.7083	6.17904	2.18462	3.5425	13.8741	.50	15.00
Total	50	7.4283	5.65966	.80040	5.8199	9.0368	.17	17.00



Mean age of patients discharged was 8.4 year, DAMA was 4.4 year, expired 7years, and referred 8.7 years.

TABLE 12: INTERVENTIONS IN SUPPORTIVE MANAGEMENT OF AKI VERSUS OUTCOME:

	Shock/inotrope support	**Mechanical ventilation**	**Renal replacement therapy**
Present	32	11	7
Absent	18	39	43

As per our study major interventions carried in patients with AKI were inotrope support, mechanical ventilation and renal replacement therapy.

Of total 50(100%) patients 32(64%) required inotrope support , 11(22%) were on mechanical ventilation, 7(14%) had renal replacement therapy.

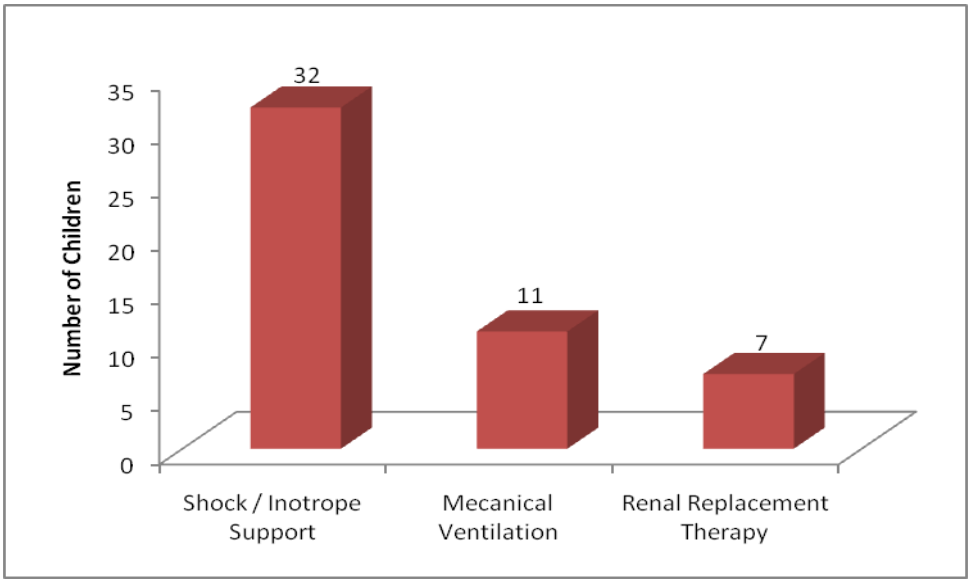
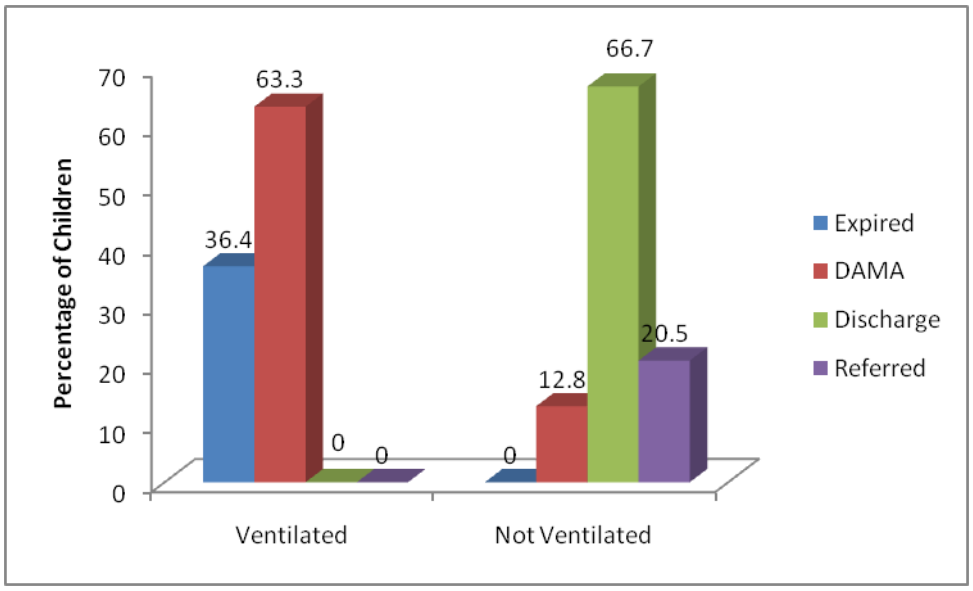


TABLE 12(A): MECHANICAL VENTILATION AND ITS OUTCOME.

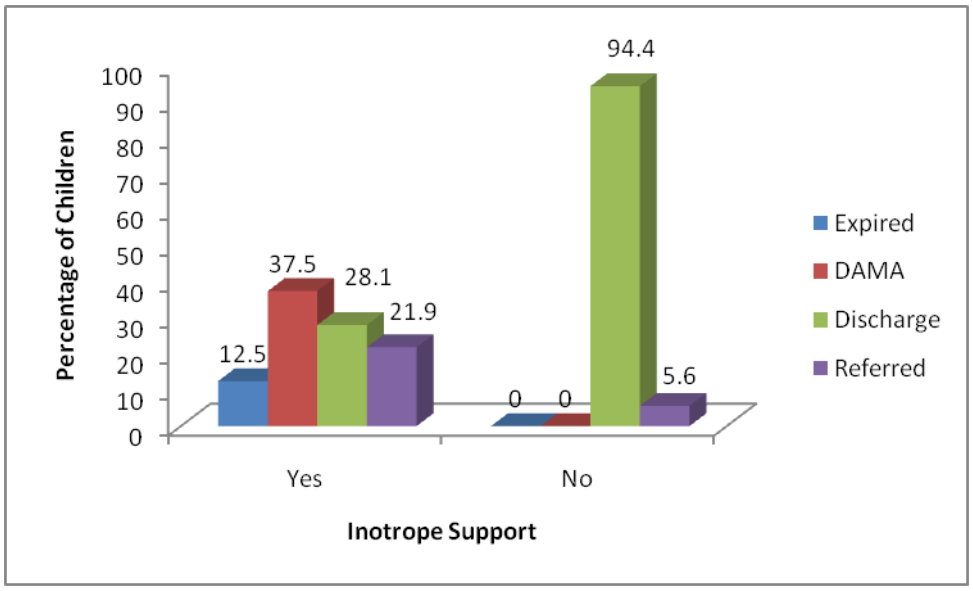
		Outcome				Total
		Expired	**Dama**	**Discharged**	**Referred**	
Ventilated	Yes	4	7	0	0	11
		36.4%	63.6%	.0%	.0%	100.0%
	No	0	5	26	8	39
		.0%	12.8%	66.7%	20.5%	100.0%
Total		4	12	26	8	50
		8.0%	24.0%	52.0%	16.0%	100.0%



Total 11(22%) patients who were ventilated, 36.4% went ecpired and rest 63.6% went DAMA due to poor prognosis and financial issues.

TABLE 12(B): INOTROPE SUPPORT AND ITS OUTCOME.

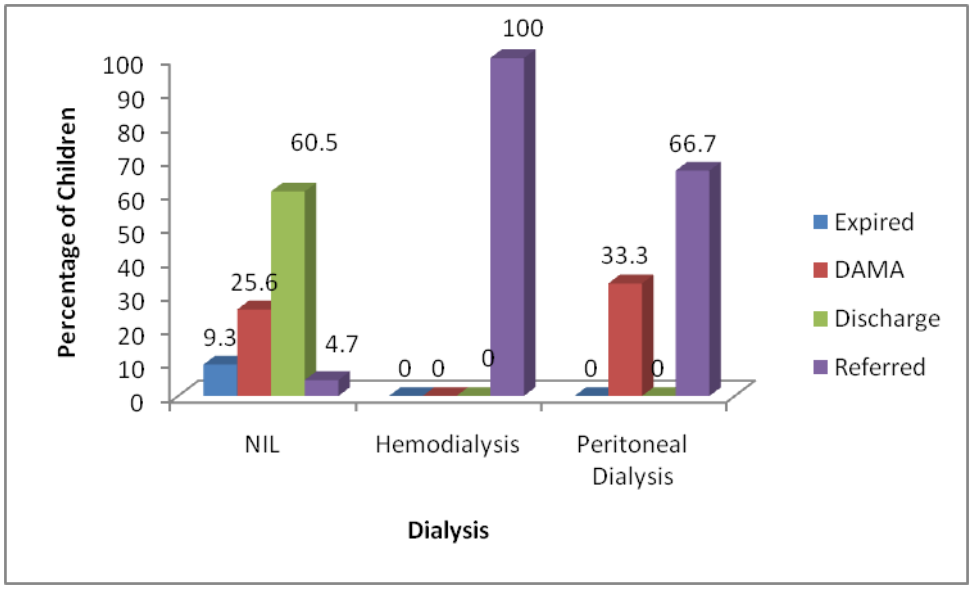
		Outcome				Total
		Expired	**Dama**	**Discharged**	**Referred**	
Inotrope support	Yes	4	12	9	7	32
		12.5%	37.5%	28.1%	21.9%	100.0%
	No	0	0	17	1	18
		.0%	.0%	94.4%	5.6%	100.0%
Total		4	12	26	8	50
		8.0%	24.0%	52.0%	16.0%	100.0%



Of 50 patients 32(64%) required inotrope, 9(28.2%) were discharged,7(21.9%) referred,4(12.5%) expired,12(37.5%) went DAMA.

TABLE 12 (C): RENAL REPLACEMENT THERAPY AND ITS OUTCOME.

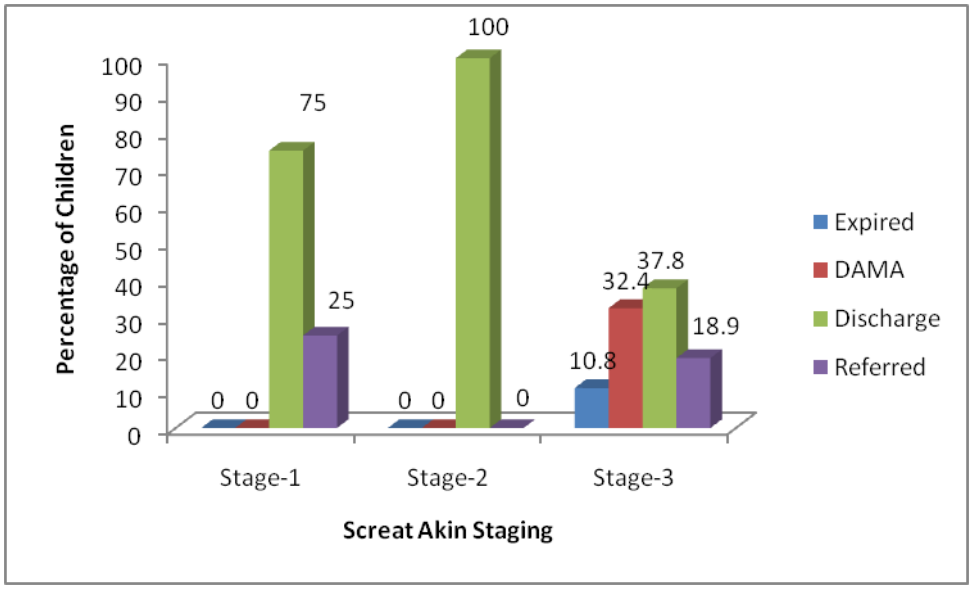
		Outcome				Total
		Expired	**DAMA**	**Discharged**	**Referred**	
Dialysis	NIL	4	11	26	2	43
		9.3%	25.6%	60.5%	4.7%	100.0%
	Hemodialysis	0	0	0	4	4
		.0%	.0%	.0%	100.0%	100.0%
	Peritoneal dialysis	0	1	0	2	3
		.0%	33.3%	.0%	66.7%	100.0%
Total		4	12	26	8	50
		8.0%	24.0%	52.0%	16.0%	100.0%



Of total 50 patients 7 (14%) patients were on renal replacement therapy of which (42.8%) were on peritoneal dialysis and (57.2%) were on hemodialysis.

TABLE 13: AKIN STAGING VERSUS OUTCOME.

		Outcome				Total
		Expired	**Dama**	**Discharged**	**Referred**	
Screat_AKIN_staging	1	0	0	3	1	4
		.0%	.0%	75.0%	25.0%	100.0%
	2	0	0	9	0	9
		.0%	.0%	100.0%	.0%	100.0%
	3	4	12	14	7	37
		10.8%	32.4%	37.8%	18.9%	100.0%
Total		4	12	26	8	50
		8.0%	24.0%	52.0%	16.0%	100.0%



	Value	**Df**	**P-value**
Pearson chi-square	13.283	6	.039

As per AKIN staging highest mortality was in stage 3. AKIN staging versus outcome was statistically not significant possibly due to small sample size.

TABLE 14: pRIFLE STAGING VERSUS OUTCOME.

		Outcome				Total
		Expired	**Dama**	**Discharged**	**Referred**	
_(pRIFLE)_staging	1	0	0	1	1	2
		.0%	.0%	50.0%	50.0%	100.0%
	2	2	1	18	0	21
		9.5%	4.8%	85.7%	.0%	100.0%
	3	2	11	7	7	27
		7.4%	40.7%	25.9%	25.9%	100.0%
Total		4	12	26	8	50
		8.0%	24.0%	52.0%	16.0%	100.0%

	Value	**Df**	**P-value**
Pearson chi-square	21.694	6	.001

As per pRIFLE staging mortality was seen in **I and F** stage. Outcome of patients was statistically significant (p value <0.001).

Of total 50 patients in our study 26(52%) patients were discharged, 4 (8%)patients expired and 12(24%) took dama. 8(16%) patients were referred to higher centre due to financial issues. Renal replacement therapy was given to 7(14%) patients of which 4 (57.2%) patients were on hemodialysis and 3(42.8%) patients on peritoneal dialysis. All patients with hemodialysis 4(100%) and 66.7% of peritoneal dialysis were referred to higher centre due to financial limitations and rest 33.3 % with peritoneal dialysis took dama. Total 11(22%) patients required mechanical ventilation and 32(60%) patients were on inotrope support. Poor outcome was associated with patients on ventilation with 36.4% mortality. Where as with inotrope support 28% patients had good recovery and 12.5% expired with 37.5 % took dama

and 21.9% referred. As the stage of AKI progesses risk of morbidity and mortality increases. In our study mortality was seen in stage 3 as per AKIN and in stage **I** and stage **F** as per pRIFLE criteria. This suggests that eCrClis more sensitive criteria in determining early acute kidney injury in pediatric age group which helps to improve outcome with early intervention. In patients of AKI with sickle cell disease and AGE recovery was 100%,and in dengue with complication recovery was 83%. Of 24% patients who went against medical advice, majority of patients had poor outcome and were explained the prognosis. In PSGN which accounted for about 8% of total patients, 50% had good outcome and rest 50% required hemodialysis. After one cycle of hemodialysis patients were referred to higher centre due to financial issues.

In gujarat, government is running IKDRC where children with AKI are offered free service and thus group of patients with financial conscience were referred to IKDRC for furtherr management.

MORTALITY VERSUS CAUSE OF DEATH

ARDS with AKI	5 days	Ventilated, inotrope support given
Sepsis with AKI	1 day	Fluid management,ventilated, inotrope support
Subacute intestinal obstruction,post operative case with sepsis	8 days	Fluid management ,higher antibiotics,ventilated, inotrope support.
Dengue with septic shock,AKI, pleural effusion	5 days	Fluid management,higher antibiotics,ventilated, inotrope support

Hakan poyrazoglu et al[75]showed that dialysis was performed in 30.3% of newborns (93.3% peritoneal dialysis, 6.7% hemodialysis), and 33.6% of children aged >1 month (59.2% peritoneal dialysis, 40.8% hemodialysis).Mechanical ventilation 28.9% in >1 month age group.

In children aged >1 month, the mortality risk increased independently 8.73-fold with MV, 5.35-fold with hypoxia, and 4.91-fold with intrinsic AKI. This is comparable to our study where 100% mosrtality was associated with mechanical ventilation and 12.5% with inotrope support. In our study majority of patients with dialysis were referred to higher centre.

Poonam mehtal et al[72]showed that younger patients and those with sepsis, shock and mechanical ventilation were at increased risk for AKI. The presence of AKI resulted in prolonged hospital stay and a four-fold higher mortality, especially among patients with AKI stages 2 and 3. The mortality in patients with AKI stage 1 (**n** =7, 14.6%)was lower compared to stage 2 (**n** =11, 84.6%) and stage 3(**n** =9, 75%) (**p**<0.001) (ijp) complications and co-morbidities included severe metabolic acidosis in 44 (26.5 %), hyponatremia in 27(16.3 %), hypernatremia in 11 (6.5 %), hyperkalemia in 24(14.5 %), hypertension in 28(16.8 %), encephalopathy in 29(17.5 %), thrombocytopenia in 28(16.9 %), mechanical ventilation in 47 (28.3 %) and shock in 61(36.7 %) children. Similar complications like metabolic acidosis, dysnatremia and hyperkalemia occurred in our study but did not have significant relation with outcome.

In our study all the three major criteria serum creatinine, urine output and estimated creatinine clearance (glomerular filteration rate) are used for staging of acute kidney injury. But urine output was not much useful in staging of AKI.

SUMMARY

Study was conducted in pediatric intensive care unit of department of Pediatric SBKS MI&RC. Pipariya, Vadodara Gujarat from 31/12/2015 to 30/6/2017.

Total 393 patients, critically ill were admitted in PICU during study period, aged >1month <18 years at Dhiraj hospital were screened and 50 patients who met inclusion criteria were included in the study.

Serum creatinine, estimated creatinine clearance and urine output were monitored during study period and were classified through pRIFLE using serum creatinine and estimated creatinine clearance in reference to baseline serum creatinine and glomerula filtration rate.

- Of 50 patients included in the study 33(66%) male, 17(34%) female with maximum age of presentation 3.1 to 8 years and mean duration of stay 6.4 days.
- Staging of AKI was done with pRIFLE and AKIN staging suggesting PRIFLE is more rapid in picking patients of AKI in **I** category in comparison to stage 2 of AKIN staging.
- Mortality risk increased with progression of staging of AKI.
- Urine output was not useful in staging of AKI in our study.
- The commonest etiology for patients with AKI was sepsis 15(30%) of total patients of which 5(33.3%) patients had pneumonia.

- 2nd most common etiology resulting in AKI was acute tropical illness accounting total 7(14%) of which (85.7%) were of dengue febrile illness and 14.3%(1) with complicated malaria.

- 3(6%) patients had metabolic acidosis, 1(2%) with hyperkalemia, 8(16%)with hypernatremia.

- 32(64%) patients who required inotrope support, 11(22%) were on mechanical ventilation,7(14%) were given renal replacement therapy. Pts on mechanical ventilation had poor outcome with 36.4% expired and 63.6% took dama.

- Of patients who were on renal replacement therapy (42.8%) were on peritoneal dialysis, (57.2%) were on hemodialysis.

- Overall Outcome of patients was 26(52%) discharged,12(24%) tookDAMA, 8(16%) referred,4(8%) expired.

- Of 4 patient expired following were the diagnosis, ARDS with AKI, sepsis with AKI, Subacute intestinal obstruction post op with sepsis,dengue with septic shock with AKI. Of 4 patients 2 were in **I** and 2 were in **F** category as per pRIFLE where as AKIN staging showed stage 100% mortality in stage 3.

- This Suggests ecrcl being more sensitive marker in picking patients with AKI in relation to serum creatinine which helps in early detection of AKI and improving outcome.

CONCLUSION

There is paucity of AKI related literature and studies in Indian scenario.

The terminology ARF is replaced by AKI. More studies are required to prove benefits of pRIFLE or AKIN staging .

But the common cause for AKI was sepsis, next to it was acute tropical illness.Perhaps good control on vector borne disease may significantly reduce burden of AKI.

We had referred many patients to higher centre because in Gujarat IKDRC is giving free service to pediatric population with kidney injury, hence we conclude that India needs many such centers to serve patients with AKI.

LIMITATIONS

Potential limitation of our study include relatively small sample size, however there are very few studies describing AKI in critically ill patients in pediatric age group.

Another potential concern was non availability of actual base line serum creatinine values of our patients. Because base line creatinine levels are the levels measure during the healthy state of patients admitted.

Because of dependency of serum creatinine levels on muscle mass, it is extremely difficult to interpret in patients with severely reduced muscle mass.

Accurate Urine output measurement was again a limitation. The introduction of urine output criterion into pRIFLE was thought to increase sensitivity but the reliability of these paramters were raised due to its dependency on extra renal factors such as volume status or release of antidiuretic hormones

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ABBREVIATIONS

ACE: ANGIOTENSIN CONVERTING ENZYME

ADQI: ACUTE DIALYSIS QUALITY INITIATIVE

AGE: ACUTE GASTROENTERITIS

AKI: ACUTE KIDNEY INJURY

AKIN: ACUTE KIDNEY INJURY NETWORK

ARDS: ACUTE RESPIRATORY DISTRESS SYNDROME

ARF: ACUTE RENAL FAILURE

ATN: ACUTE TUBULAR NECROSIS

CHD: CONGENITAL HEART DISEASE

CRRT: CHRONIC RENAL REPLACEMENT THERAPY

eCrcl: ESTMATED CREATININE CLEARANCE

DAMA: DISCHARGE AGAINST MEDICAL ADVICE

GFR: GLOMERULAR FILTERATION RATE

HTN: HYPERTENSION

HUS: HEMOLYTIC UREMIC SYNDROME

IHD: INTERMITTENT HEMODIALYSIS

IKDRC: INSTITUE OF KIDNEY DISEASE AND RESEARCH CENTER

KIDGO:KIDNEY DISEASE IMPROVING GLOBAL OUTCOME

PICU: PEDIATRIC INTENSIVE CARE UNIT

pRIFLE: PEDIATRIC RISK INJURY FAILURE LOSS OF FUNCTION AND END STAGE DISEASE.

PSGN: POST STREPTOCOCCAL GLOMERULONEPHRITIS

PUJ: PELVIC URETRIC JUNCTION

MDRD: MODIFICATION OF DIET IN RENAL DISEASE

MV: MECHANICAL VENTILATION

SCD: SICKLE CELL DISEASE.

PROFORMA

Name : Age: Sex:

Address:

Contact No.

Complains of:

Fever

Cough

Pain in abdomen

Decreased frequency of micturation

Other complains

Family history:

Immunization :

Physical examination

Weight(kg):

Nutritional status(PEM grade) :

Height In cms:

Arm span:

Upper segment/lower segment:

BMI :

Pallor ( )Icterus ( )Clubbing( )Cyanosis ( )Edema ( )Lymph nodes:

Vitals:

ABDOMINAL EXAMINATION

Inspection:

Palpation:

Liver Enlarged cms below costal margin

1.yes

2.no

Spleen

Kidney

Percussion:

Auscultation:

OTHER SYSTEM:

Investigations:

Hemoglobin:

Total leukocyte count :

Differential leukocyte count: N L M E B

(absolute eosinophil count:)

Platelet:

ESR :

CRP:

PT

APTT

AST : **ALT:**

urea	**admission**	**24 hours**	**48 hours**	**Day 3**	**Day 4**	**Day 5 …**
s. creatinine						
Urine output						

Serum Electrolytes (Na, K, Cl, Ca, Mg, PO_4)

Abg analysis

Urinary Electrolytes

Urinary creatinine

Creatinine Clearance

Fractional excretion of Sodium

X-RAY film of chest:

USG Abdomen

Renal Doppler

Renal Biopsy

CT IVP

PATIENT INFORMATION SHEET AND CONSENT FORM

PATIENT INFORMATION SHEET

Study Title:

TO STUDY THE OUTCOME AND PROGNOSIS OF ACUTE KIDNEY INJURY IN PEDIATRICS PATIENTS

1. Introduction

   To study etiological factors for acute kidney injury and asses acute kidney injury as prognostic factor

2. What is the purpose of this study?

   Its only observational study

3. Why have I been chosen?

   My condition is suitable for this study.

4 Do I have to take part?

   It is only an observational study

5. How long will the study last?

   The study last for around 1 to 1.5 years time period

6. What will happen to me if I take part?

   - Screening Period: observational study
   - Treatment Period: observational study
   - Allocation of investigational product: basic routine investigation (CBC, RFT, SERUM ELECTROLYTE)
   - Follow-up period: not required ,only observational study

7. What do I have to do?

   Only observational study ,no extra burden will be given

8. What is the drug being tested?

   No drug being tested only observational study

9. What are the benefits of the study?

   Patient will be acknowledged about undergoing disease

10. What are the alternatives for treatment?

    Only observational study no alternatives

11. What are the side effects of the treatment received during the study?

    No side effect

12. What if new information becomes available?

    Information will be shared with you

13. What happen when the study stops?

    Where ever study stops you will be notified

14. Will my taking part be kept confidential?

    Yes

15. What else should I know?

    There is no information study right now as soon as available you will be notified

16. Additional Precautions

    Not available

17. Who to call with questions?

    Dr CHARMEE JOSHI

    PH NO: 9898997168

Protocol No.: XXXXXXXX

INFORM CONSENT

Study Title

TO STUDY THE OUTCOME AND PROGNOSIS OF ACUTE KIDNEY INJURY IN PEDIATRICS PATIENTS

StudyNumber:

Subject's Initials:

Subject's Name:

Date of Birth /

Age:

Address of the Subject: _

Qualification:

Occupation: Student/ Self-Employed/ Service/ House-wife/Others:

(Please tick as appropriate)

Annual Income of the subject:

Details of Nominee (s):

Name of Nominee:

Address of Nominee:

Relation to Subject:

Please initial box (Subject)

1. I confirm that I have read and understood the information sheet dated …….....….for the above study and have had the opportunity to ask questions. [ ]

(ii) I understand that my participation in the study is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected. [ ]

(iii) I understand that the Sponsor of the clinical trial, others working on the Sponsor's behalf, the Ethics Committee and the regulatory authorities will not need my permission to look at my health records both in respect of the current study and any further research that may be conducted in relation to it, even if I withdraw from the trial. I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published. [ ]

(iv) I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s) [ ]

(v) I agree to take part in the above study. [ ]

Signature Thumb impression) of the (or Subject/LAR:

Date: / /

Signature of parents Name:

Signature of the Investigator:

Date: / /

Study Investigator's Name:

Signature of the Witness _____

Date: / _/

Name of the Witness:

_____

Copy of the Patient Information Sheet and duly filled Informed Consent Form shall be handed over to the subject or his/her attendant.

SumandeepVidyapeeth University

Piparia, Ta. Waghodia, Dist. Vadodara, Pin 391760

Informed Consent Form (ICF) for Participants in Research Programmes involving studies on human beings

- Study Title:to study outcome and prognosis of acute kidney injury in pediatric patients.

Study Number: SVU/SBKS/ /2013-____

Participants Initials: _____ Participants Name: _____

Date of Birth / Age _____ (Years)

1. I confirm that I have read and understood the information sheet dated _____ for the above study and have had the opportunity to ask questions. [ ]
2. I understand that my participation in the study is voluntaryand that I am free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected. [ ]
3. I understand that the investigator of this study, others working on the investigator's behalf, the Ethics Committee and the regulatory authorities will not need my permission to look at my health records, both in respect of the current study and any further research that may be conducted in relation to it, even if I withdraw from the study. I agree to this access. However, I understand that my identity will not be revealed in any information related to third party or published. [ ]
4. I agree not to restrict the use of any data or results that arise [ ]
5. from this study provided such a use is only for scientific purpose(s).[ ]
6. I agree to take part in the above study. [ ]

Signature (or thumb impression) of the parents/guardians _____ legally acceptable representative
Signatory's Name _____ Date _____
Signature of the investigator _____ Date _____
Study Investigator's Name _____
Signature of the impartial witness _____ Date _____
Name of the witness _____

દર્દી માહિતી પત્રક અને સંમતિ પત્રક

દર્દી માહિતી પત્રક

અભ્યાસનું શીર્ષક : બાળ દર્દીઓમાં નિષ્કર્ષ અને સખત કિડની ઈજાના પૂર્વ નિદાનનો અભ્યાસ કરવો.

૧. પ્રસ્તાવના :

તીવ્ર કિડની ઈજા તેમજ અતિતીવ્ર કિડની ઈજાને પૂર્વનિદાન તરીકે હેતુ વિષયક પરિબળોનો અભ્યાસ કરવો.

૨. આ અભ્યાસનો હેતુ શો છે?

આ માત્ર અવલોકનાત્મક અભ્યાસ છે.

૩. મને શા માટે પસંદ કરવામાં આવ્યો છે?

મારી સ્થિતિ આ અભ્યાસ માટે ઉચિત છે.

૪. શું મારે ભાગ લેવાનો છે?

તે માત્ર એક અવલોકનાત્મક અભ્યાસ છે.

૫. આ અભ્યાસ કેટલો લાંબો સમય ચાલશે?

આ અભ્યાસ ૧ થી ૧.૫ વર્ષના સમયગાળા સુધી ચાલશે.

૬. જો હું ભાગ લઉં તો મને શું થશે?

તપાસનો સમયગાળો : અવલોકનાત્મક અભ્યાસ ચિકિત્સા સમયગાળો : અવલોકનાત્મક અભ્યાસ અન્વેષણાત્મક ઉત્પાદનની ફાળવણી : પાયાની નિયમિત તપાસ (CBC, RFT, SERUM, ELECTROLYTE) ફોલોઅપ સમયગાળો : જરૂર નથી, માત્ર અવલોકનાત્મક અભ્યાસ છે.

૭. મારે શું કરવાનું રહેશે?

માત્ર અવલોકનાત્મક અભ્યાસ, કોઈ વધારાનું ભારણ આવશે નહીં.

૮. કયા ઔષધનું પરીક્ષણ કરવામાં આવશે?

કોઈ ઔષધનું પરીક્ષણ થનાર નથી, માત્ર અવલોકનાત્મક અભ્યાસ

૯. આ અભ્યાસના લાભ કયા છે?
દર્દીને વિકસીત રહેલા રોગ વિશે જાણવવામાં આવશે.

૧૦. આ ચિકિત્સાના વિકલ્પો કયા છે?
માત્ર અવલોકનાત્મક છે. કોઇ વિકલ્પો નથી.

૧૧. આ અભ્યાસ દરમિયાન મળતી ચિકિત્સાની કઇ આડઅસર અસરો છે?
કોઇ આડ અસર નથી.

૧૨. જો કોઇ નવી માહિતી મળે તો શું?
માહિતી તમારી સાથ વહેંચવામાં આવશે.

૧૩. જો અભ્યાસ અટકી જાય તો?
જ્યારે અભ્યાસ અટકી જાય ત્યારે તમને જાણ કરવામાં આવશે.

૧૪. જો કંઇ ખોટું થાય તો?
તમામ જવાબદારી દર્દીની માહિતી આપનારની રહેશે. કૃપા કરી તમને (ડૉ. ચાર્મી જોષી મો. ૯૮૯૮૯૯૭૧૬૮) નો સપર્ક કરી શકો છો.

૧૫. શું મારી સહયોગીતાને ગુપ્ત રાખવામાં આવશે?
હા

૧૬. મારે બીજું શું જાણવું જોઇએ?
હાલમાં કોઇ માહિતી અભ્યાસ નથી જ્યારે મળશે કે તરત જ તમને જાણ કરવામાં આવશે.

૧૭. વધારાની સાવધાની?
નથી

૧૮. પ્રશ્ન / મુંઝવણ માટે કોને ફોન કરવો?
ડૉ. ચાર્મી જોષી
ફોન નં. ૯૮૯૮૯૯૭૧૬૮

સંમતિપત્રક

અભ્યાસનું શીર્ષક : બાળ દર્દીઓમાં નિષ્કર્ષ અને સખત કિડની ઈજાના પૂર્વ નિદાનનો અભ્યાસ કરવો.

અભ્યાસ નં. :

અભ્યાસપાત્રનું ટુકુ નામ : ..... અભ્યાસ પાત્રનું નામ : .....

જન્મ તારીખ / ઉંમર .....

અભ્યાસ પાત્રનું સરનામું : .....

લાયકાત : .....

વ્યવસાય : વિદ્યાર્થી / સ્વરોજગારી / નોકરી / ગૃહિણી / અન્ય (યોગ્ય હોય તેના પર નિશાન કરો)

અભ્યાસપાત્રની વાર્ષિક આવક : .....

વારસદાર(રો)ની વિગતો : .....

વારસારોનું નામ : .....

વારસદારોનું સરનામું: .....

.....

અભ્યાસપાત્ર સાથે સંબંધ : .....

(બોક્સમાં ટૂંકી સહી કરો)

અભ્યાસપાત્ર

૧. હું ખાતરી આપું છું કે આ અભ્યાસનું માહિતી પત્રક મેં તા. ..... ના રોજ વાંચ્યું છે સમજ્યું છે અને મને પ્રશ્નો પૂછવાની તક મળેલ છે. [ ]

૨. હું સમજું છું કે આ અભ્યાસમાં મારી સહયોગિતા સ્વૈચ્છિક છે અને હું કોઇ પણ સમયે કારણ દર્શાવ્યા વિના જ મુક્ત થઇ શકુ છું તેથી મારી તબીબી ચિકિત્સા કે કાયદાકીય હક્કો પર પ્રભાવ પડશે નહીં. [ ]

૩. હું સમજું છું કે અભ્યાસના અન્વેષક, તેઓના વતી કાર્ય કરનાર, હિતરક્ષક સમિતિ અને નિયામક સતાઓને મારા વર્તમાન કે ભાવિ સંશોધન જે મારી સહભાગિતા પાછી ખેંચી લીધા પછી પણ થાય તો બંનેકિસ્સામાં મારી સ્વાસ્થ્ય માહિતી જોવા મારી પરવાનગીની આવશ્યકતા નથી. હું તેના ઉપયોગમાં લેવામાં સહમત છું જો કે હું સમજું છુંકે ત્રાહિત વ્યક્તિ કે પ્રકાશન દ્વારા મારી ઓળખ રજુ કરવામાં આવશે નહીં. [ ]

૪. હું કોઇ પણ માહિતી કે પરિણામ જે માત્ર આવા વૈજ્ઞાનિક હેતુસર ઉપલબ્ધ કરાવાય છે તેના ઉપયોગમાં અવરોધ ન કરવા માટે સંમત છું. [ ]

૫. હું ઉપરોક્ત અભ્યાસમાં સહભાગી થવા સંમત છું. [ ]

અભ્યાસની સહી કે અંગુઠાનું નિશાન .....તારીખ .....

માતાપિતાની સહી નામ .....તારીખ .....

અભ્યાસ અન્વેષકની સહી .....તારીખ .....

સાક્ષીની સહી .....તારીખ .....

સાક્ષીનું નામ .....

દર્દી માહિતી પત્રક અને યોગ્ય રીતે ભરેલું સંમતિ પત્રકની નકલ અભ્યાસપાત્ર કે તેની સાથેની વ્યક્તિને આપવું.

અભ્યાસમાંભાગલેવામાટેસમજીવિચારીનેઆપેલીપરવાનગીનુસમ્મતિ-પત્રક

અભ્યાસનુનામ: બાળ દર્દીઓમાં નિષ્કર્ષ અને સખત કિડની ઈજાના પૂર્વ નિદાનનો અભ્યાસ કરવો.

અભ્યાસક્રમાંક:_____ તારીખ:_____

સહભાગીનુપુરુંનામ:_____

સહભાગીનુકુટુંબનામ:_____

સહભાગીનીજન્મતારીખ / ઉંમર: _____

૧. હુંખાત્રીઆપુંછુંકે મારા બાળક માટે ઉપરોક્તઅભ્યાસની (તા: / /) માહિતીવાંચીછે અનેસમજીછેઅનેતેઅંગેનામુંઝવતાપ્રશ્નોપુછવાની અમને તક આપવામાઆવીછે.

૨. હુંજાણુંછુંકે મારા બાળકના આ અભ્યાસમાંભાગ લેવો મરજીયાત છે અને, કોઇપણજાતનુકારણઆપ્યવગર, તેમાથીગમેત્યારખસીજવાની છૂટછે, અને આમકરવાથી મારા બાળકનાતબીબીસારવારકેકાયદેસરનાહક્કોનેકોઇઅસરનહીંથાય.

૩. હુંજાણુછુંકેઆઅભ્યાસનાતપાસકર્તા, તેમનામદદનીશો, એથિકલટીમઅનેતેનાઉપરદેખરેખરાખતાઅધિકારીઓને મારા બાળકના

સ્વાસ્થ્યનીકોઈપણજાતનીમાહતી, સદરઅભ્યાસનેલગતીકેતેસિવાયની, મેળવવામાટે મારા બાળકનાપરવાનગીનીજરૂરરહેશેનહીં, ભલેપછી અભ્યાસમાંથી ખસીજાય. હુંજાણુંછુંકે મારા બાળકના આપ્રકારનીમાહિતીઅન્યકોઇનેજાણકેપ્રસિધ્ધનહીંકરવામાંઆવે.

૪.આઅભ્યાસદરમ્યાન, અથવાતેનાઅંતેપ્રાપ્તથતીમાહિતી, કોઈપણજાતનીવૈજ્ઞાનિકશોધમાટેઉપયોગકરવામાટે હુ

સ્વૈચ્છિક રીતેછુટઆપુંછું.

૫. હુ મારા બાળકના અભ્યાસમાંભાગલેવા / જોડાવામાટેસહમતિઆપુંછું.

અભ્યાસમાંભાગલેનારનીસહિઅથવાઅંગુઠાનુનિશાન:_____તારીખ:_____

કાયદેસરનાસ્વિકૃતતપાસકર્તાનીસહી: _____તારીખ:_____

તપાસકર્તાનુનામ: ડો._____

તટસ્થસાહેદ / ગવાહનીસહી: _____તારીખ:_____

તટસ્થસાહેદ / ગવાહનુનામ: _____

No.	age	sex	duration of stayin days	c/o	wt (kg)	ht (cm)	systemic examination	hb	tc	dc	plt(lakh)	esr	crp	serum (na/k/cl)	urinary electrolytes	urea	creatinine
1	10yr	male	2	fever ,not passing urine 2 days	18.8kg	123cm	p/a tender , pansystolic murmur	14.6	11,600	48/45/3/4	3.6	18	5	140/4.8/100		124	4.2
2	2mnth	female	4	fever and vomiting	3.36kg	55cm	umbilical hernia	9.5	35384	39/52/4/5	1 lakh	10	7.4	172/4.5/131		173	2.9
3	2year	female	5	loose stools	5.6kg	73cm	hepatomegaly	10.2	12,100	67/25/4/4	1.8	24		131/2.1/104		131	2.3
4	10year	female	4	fever/swelling over face	22.3	130	hepatospleenomegaly	11	45000	61/30/4/5	20,000	10	20.2	140/3.1/102		86	1.4
5	14yr	female	6	swelling over face and eyelids with decreased frequency of mictuirition	48	151	hepatospleenomegaly	6.2	7600	69/27/4/5	1.9	18	24	135/4.1/104	64/21/48	120	14.4
6	8year	male	2	fever and dribbling of urine	21	120	perabdomen tense	7.1	9300	78/134/5	1.5	12	2	140/4.3/113	80/13/72	182	8.8
7	8month	male	1	not passed urine 4 days	8kg	74	mild icr scr	8.5	21,800	59/36/2/3	2.32	14	5	130/5.5/95		159	5.5
8	6month	male	1	vomiting ,loose stools	5.5	58	spleenomegaly	9.4	25000	71/20/4/5	1.2	12	44	111/2.5/91		165	5.2
9	15 year	female	5	vomiting and convulsions	25.6	146	hepatospleenomegaly	7.3	11,000	77/16/3/4	1.88	102	6	125/5/94	97/17/80	185	12.4
10	9mnth	female	3	vomiting rds	7	60	hepatomegaly	15	2500	78/15/3/4	2	15	6	140/4.3/113		50	2
11	1yr	male	4	fever	8kg	70 cm	delayed skin turgor ,suken eyeballs	12.8	28,000	70/28/1/1/	1.5	38	90	156/4.2/98		85	1.5
12	16year	female	8	abdominal pain	50	154	abdominal pain	9.2	3000	30/65/4/1	80,000	24	90	154/4.4/100		120	2.5
13	12year	male	3	abd pain with dribbling of urine	35kg	130 cm	abdominal tenderness	10.6	20,000	60/35/4/1	2	20	20	142/5.4/90		24	1
14	11year	male	4	fever with rash	30	130 cm	fever	9.2	10,000	50/40/7/3	80,000	10	2	138/4/92		100	1.2
15	14yr	male	7	abd pain with dribbling of urine	28	135	abdominal pain	10.2	12,000	50/42/5/3	2	18	2	134/3.5/100		52	1.5
16	10year	female	3	dyspnea at rest	25	130 cm	hepatomegaly	15	8000	30/76/3/1	2.5	10	2	134/3.5/100		58	2.7
17	8month	female	2	fever	7	70 cm	hepatomegaly	8	28000	66/30/3/1	2	10	2	130/5.5/95		159	5.5
18	14yr	male	2	swelling over face with decreased mictuiriton	42	152	periorbital puffiness	6	7600	69/27/4/5	2.5	18	2	135/5/106	64/21/48	220	14.3
19	15y	male	8	swelling over eyes a/w vomiting and loose stools	41	161	hepatomegaly	10.2	16,000	48/44/4/4	5	11	0.5	140/4/90		25	1.2
20	8year	female	6	abdominal pain with red color urine	20.5	141	pallor	10.8	26,000	60/45/4/1	5	17	2	138/4.8/97		60	1.2
21	7y	male	6	vominting and abdominal pain	14.34	110	pallor	8	7000	60/31/4/5	2.24	10	2	140/3.8/100		42	1.5
22	5y	male	13	facial puffiness with head ache	26	143	pallor	10	12,600	79/13/4/4	4	10	2	140/5/100		44	1.5
23	15y	male	2	abdominal pain vomiting	40	156	pallor	8.9	9,800	77/16/3/4	3	62	2.2	134/4.5/95		166	6.6
24	3.5year	male	11	loose stool vomiting	6.5	64	delayed skin turgor , sunken eyeball , respiratory distress	10.7	16,800	55/36/4/5	2.36	20	9	160/3.6/98		63	5
25	10year	female	9	swelling over face and abdomen	32	135	abdominal distension bladder palpable	9.6	10,100	61/22/3/4	1.3	10	2	135/3.6/98		240	17.8
26	7years	male	6	fever	15.8	114.5	hepatosplenomegaly	8.8	13000	55/36/4/5	4.5	5	23	140/4.4/98		84	1.5
27	7years	female	8	fever	14.43	108	abdominal tenderness	12	3100	45/46/4/5	80,000	10	2	150/4.5/90		70	1.6
28	14years	male	11	abdominal pain,vomitting	42.66	152	sunken eyeball	9.4	11000	90/5/2/3	2.22	8	1	135/4/90		65	1.8
29	1year	female	7	pallor,fever,breathlessness,loose stool	5.74	69	pallor	6.5	6692	20/7/4/5	2.42	10	1	133/4.5/101		88	1.9
30	15years	female	7	pain all over the body	31.94	148	hepatosplenomegaly	7.8	6300	61/30/4/5	1.71	10	11.69	137/4/100		74	1.5
31	4years	male	7	dehydration	6.73	66	sunken eyeball	8.3	42300	66/25/4/5	3.18	34	197.02	156/4/102		102	1.6
32	5years	male	18	nvoluntary movements	12.3	98	pallor	8.1	13300	50/41/4/5	4.5	10	2	139/3.7/109		44	1
33	2years	female	4	vomitting ,fever,altered sensorium	7	77	pallor	11.8	11400	60/32/4/4	0.2	10	2.4	157/2.5/106		78	1.5
34	13years	female	14	pain in upper limb lower limb ,pain in abdomen,difficulty in walking	25	135	hepatosplenomegaly	6.2	7200	76/17/3/4	2.16	34	203	140/4.4/98		45	1.6
35	3 months	male	5	fast breathing,cough,fever	2.8kg	63	pallor	11.1	20800	90/7/1/2	1.2	40	171	153/4/127		64	1.8
36	16years	male	7	fever, vomitting	41	162	abdominal tenderness	11.3	6000	51/40/4/5	80000	12	12	140/3.5/98		88	1.6
37	8years	male	7	fever, cough,cold, vomitting	21	131	abdominal tenderness	8.9	5300	52/40/4/3	1.6	10	12	143/3.1/107		98	1.1
38	4years	male	12	generalised weakness	13	101	areflexia	10	4500	54/40/4/3	2	10	0.5	135/3.5/95		30	1.8
39	7years	male	6	vomiting ,loose stools,fever	22kg	120	s/o severe dehydration	10.2	20,300	60/45/4/1	2	15	6	127/5/94		65	1.1
40	2year	male	7	vomiting ,loose stools	12kg	85	s/o severe dehydration	11	10,300	56/40/3/1	2.2	10	2.2	153/3.5/95		66	1
41	8 month	male	12	cough, respiratory distress	8kg	69	s/o respiratory distress	8.8	23,500	70/28/1/1/	1.2	35	25	130/4.5/90		89	1
42	15months	male	11	fever,cough , fast breathing	9	75	s/o respiratory distress	7.5	20,000	67/26/4/3	80,000	20	34	132/4.0/92		40	1.5
43	4months	male	1	fever,respiratory distress	5	65	s/o shock	8	21000	71/25/2/2	20,000	12	102	150/5.5/102		102	2.5
44	10year	male	21	fever, respiratory distress,shock	30	134	s/o respiratory distress with shock	9	23,000	70/28/1/1/	30,000	20	54	152/5.0/98		100	3
45	4years	male	2	abdominal pain with burning mictuirition	14	100	pallor, abdominal distension	9.5	10,300	68/25/4/3	2.5	10	2	134/3.5/100		68	1.9
46	9 months	female	13	cough fever	7.5	73	tachypnea	9	10,000	65/29/3/3	3	10	2	139/4.2/100		45	0.8
47	14 years	male	3	fever with rash	45	163	petechia	11.5	4000	40/52/4/4	2	2	80,000	140/3.5/95		145	1.1
48	17 years	male	5	fever with chills	54	165	fever	10	4500	44/54/1/1	20	2.2	85,000	135/4.4/90		135	1.9
49	14 years	male	2	abdominal pain	60	160	vomiting , loose stool	10.5	10,200	50/45/3/2	10	2	1.5	145/4.9/90		66	1.2
50	11 MONTH	MALE	3	fever	8.5	73cm	fever	8	16,300	56/27/3/4	5	10	4	148/5/95	100	100	1.4

No.	akin staging			baseline creatinine range	u/o (ml/kg/hr)over 12 hours	NO AKI	risk	injury	failure	gfr	prifle			akin staging	diagnosis	treatment	outcome	dialysis	ventilated	inotrope support
	stage 1	stage 2	stage 3								risk	injury	failure							
1			✔	0.64	<0.3 >12HRS				✔	16.1			✔	3	B/L renal hydronephrosis with oliguric acute kidney injury chd, renal parenchymal disease	ivf, ivab ,inotrope	dama		no	yes
2			✔	0.35	<0.3 >12HRS				✔	8.3			✔	3	septic shock with aki/pneumonia /metabolic acidosis/hypernatremia.	inj ceftriaxone/inj dobutamine/ivf	dama		yes	yes
3			✔	0.35	<0.3 >12HRS				✔	13.8			✔	3	ards/pneumonia/ aki/AGE/severe dehydration/sam/hypokalemia	inj ceftriaxone,inj dopamine,ivf,inj pipzo,ventilated	expired		yes	yes
4			✔	0.64	>1ml/kg/hr /day	✔				51		✔		2	dengue shock syndrome stage 3/septic shock/aki/severe thrombocytopenia/dic/hypokalemia/b/l pleural effusion /moderate ascites	inj falcigo, inj cefotaxime inj pantop ,inj meropenem	expired		yes	yes
5			✔	0.81	<0.3 >12HRS				✔	5.7			✔	3	acute kidney injury/psgn/anemia/hypokalemia	ivf , lasix, inj ceftriaxone,t nefidipine ,hemodialysis	referred	hemodialysis	no	yes
6			✔	0.55	<0.3 >12HRS				✔	7.5			✔	3	left sided forearmabscess /b/l uretric calculi /AKI	ivf inj ceftriaxone	dama		no	YES
7			✔	0.35	<0.3 >12HRS				✔	5.9			✔	3	pneumonia / acute kidney injury/hyperkalemia	peritoneal dialysis	dama	peritoneal dialysis	no	yes
8			✔	0.35	<0.3 >12HRS				✔	4.9			✔	3	septic shock/pneumonia/acute kidney injury/anemia/hypokalemia	inj linezolid inj cefoperazone ,inj dopamine	referred		no	yes
9			✔	0.81	<0.5ml/kg/hr>6hrs		✔			21.4			✔	3	acute kidney injury in renal parenchymal disease/thrombocytopenia/generalised tonic seizure	inj ceftriaxone,inj eptoin,t nefidipine 2 cycles of hemodialysis	referred	hemodialysis	no	yes
10			✔	0.35	<0.3 >12HRS				✔	13.2			✔	3	pneumonia/severe metabolic acidosis/non oliguric AKI	ventilated ,inj ceftriaxone/inj dopamine	dama		yes	yes
11			✔	0.35	<0.3 >12HRS				✔	20.5			✔	3	sepsis in AGE/SEVERE HYPERNATREIMC DEHYDRATION/AKI	ivf iv ab	discharge		no	YES
12			✔	1.3	>1ml/kg/hr /day	✔				33.88		✔		2	subacute intestinal obstruction with sepsis with aki WITH HYPERNATREMIA hypotension with colostomy done	ivfiv ab ventilation	expired		yes	yes
13	✔			0.71	<0.5ml/kg/hr>6hrs		✔			71.5	✔			1	uti with hydronephrosis/RENAL PARENCHYMAL DISEAS/AKI	ivf, iv ab	referred		no	no
14		✔		0.64	<0.5ml/kg/hr>6hrs		✔			59.5		✔		2	dengue fever/AKI	ivf	discharge		no	no
15			✔	0.81	<0.5ml/kg/hr>12hrs			✔		49		✔		2	left puj obs with rt kidney stone/AKI	ivf iv ab dj stent	discharge		no	no
16			✔	0.64	>1ml/kg/hr /day	✔				26.48			✔	3	malignant hypertension with left ventricular hypertrophy with hypertension retinopathy grade 4/AKI	ivf iv ab anti hypertensive	referred	hemodialysis	no	yes
17			✔	0.35	<0.3 >12HRS				✔	5.72			✔	3	pneumonia/ septic shock / aki	ivf ab lasix	referred	peritoneal dialysis	no	yes
18			✔	0.81	<0.3 >12HRS				✔	5.8			✔	3	psgn /AKI/ hypertension with anemia	ivf ab lasix	referred	hemodialysis	no	yes
19	✔			0.81	<0.5ml/kg/hr>6hrs		✔			73	✔			1	hypertension with idiopathic renal disease with subacute appendicits/AKI	ivf ; iv ab antihtn	discharge		no	no
20		✔		0.55	<0.5ml/kg/hr>12hrs			✔		64.6		✔		2	psgn/WITH HEMATEMESIS/AKI	ivf, iv ab	discharge		no	no
21			✔	0.48	<0.5ml/kg/hr>12hrs			✔		40.3		✔		2	right kidney hydronephrosis with dilated ureter with aki secondary to partial obstruction rt vuj/aki	ivf; iv ab ;	discharge		no	no
22			✔	0.42	<0.5ml/kg/hr>12hrs			✔		52.43			✔	3	psgn with uti with hypertension	ivf;ivab	discharge		no	no
23			✔	0.81	<0.3 >12HRS				✔	13			✔		pneumonia/sepsis/aki	ivf iv ab	dama		no	yes
24			✔	0.42	<0.3 >12HRS				✔	7			✔	3	AGE / severe dehydration with aki/hypernatremia.	ivf iv ab	referred	peritoneal dialysis	no	yes
25			✔	0.64	<0.3 >12HRS				✔	4.3			✔	2	acute retention of urine due to uretric cyst with bladder neck obstruction with secondary renal damage due to obstructive uropathy/aki	ivf ab dj stent	discharge		no	no
26		✔		0.55	<0.5ml/kg/hr>12hrs			✔		41.9		✔			vasoocclusive crisis in sickle cell disease with pneumonia with undernutrition/WITH INTERSTITIAL NEPHRITIS/aki	ivf, ivab,	discharge		no	no
27			✔	0.55	<0.5ml/kg/hr>12hrs			✔		37.12		✔		2	dengue fever with warning signs with thrombocytopenia/aki	ivf,ivab,	discharge		no	yes
28			✔	0.81	<0.5ml/kg/hr>12hrs			✔		46.4		✔			ileal duplication cyst/POST OP/aki	ivf iv ab	discharge		no	no
29			✔	0.35	<0.3 >12HRS				✔	16			✔	3	AGE/severe dehydration with severe acute malnutrition/aki	ivf iv ab	discharge		no	no
30		✔		0.81	<0.5ml/kg/hr>12hrs			✔		54		✔		2	vaso occlusive crisis in k/c/o sickle cell disease/aki	ivf iv ab	discharge		no	yes
31			✔	0.42	<0.3 >12HRS				✔	22.68			✔	3	septic shock/hypernatremic dehyration/aki	ivf iv ab	discharge		no	yes
32		✔		0.48	<0.3 >12HRS				✔	44.1			✔	3	b/l hydroureter with hydronephrosis due to bladder calculi with sickle cell trait	ivf iv ab	discharge		no	no
33			✔	0.35	<0.3 >12HRS				✔	28			✔	3	septic shock with metabolic acidosis with sam/hypernatremia/aki	ivf iv ab	dama		yes	yes
34			✔	0.81	<0.5ml/kg/hr>12hrs			✔		46.4		✔		2	vaso occlusive crisis in k/c/o sickle cell disease with severe anaemia/INTERSTITIAL NEPHRITIS/aki	ivf iv ab	dor		no	no
35			✔	0.35	<0.3 >12HRS				✔	15.4			✔	3	septic shock/ respiratory failure with uti with b/l hydroureter with hydronephrosis with thrombocytopenia with dic with hyponatremia/aki	ivf iv ab	dama		yes	yes
36	✔			1.3	<0.5ml/kg/hr>12hrs			✔		55.6		✔		2	dengue fever with warning signs with secondary hypertension/aki	ivf iv ab	discharge		no	yes
37		✔		0.55	<0.5ml/kg/hr>12hrs			✔		65.5		✔		2	dengue fever with warning signs with p. vivax malaria/aki	ivf iv ab	discharge		no	no
38			✔	0.42	<0.3 >12HRS				✔	30.86			✔	3	guillian bairre syndrome/ARDS/aki	ivf, ivig ,corticosteroids	dama		yes	yes
39		✔		0.55	<0.5ml/kg/hr>12hrs			✔		60		✔		2	sepsis with severe hypernatremic dehydration/aki	ivf,ivantibiotic,inotrope support	discharge		no	no
40		✔		0.35	<0.5ml/kg/hr>12hrs			✔		46.75		✔		2	hypernatremic dehydration with shock/aki	ivf,iv ab, inotrope support	discharge		no	yes
41			✔	0.35	<0.5ml/kg/hr>12hrs			✔		38		✔		2	pneumonia with septic shock/aki	ivf,iv ab , inotrope support	discharge		no	yes
42			✔	0.35	<0.5ml/kg/hr>12hrs			✔		27.5		✔		2	ards/left sided lung collapse/aki	ivf, iv ab ,inotrope support,ventilated	dama		yes	yes
43			✔	0.35	<0.3 >12HRS				✔	11.44			✔	3	septic shock with aki	ivf,iv ab,inotrope , ventilated	expired		yes	yes
44			✔	0.64	<0.3 >12HRS				✔	24.54			✔	3	ARDS/AKI/ severe pneumonia/shock	ivf,iv ab , inotrope support, ventilated	dama		yes	yes
45			✔	0.42	<0.3 >12HRS				✔	28.94			✔	3	grade 4 hydronephrosis left kidney /multiple renal calculi/AKI	ivf , surgical intervention	discharge		no	no
46		✔		0.35	<0.5ml/kg/hr>12hrs			✔		40.15		✔		2	pneumonia in large restrictive pda/CHD/CCF/AKI	ivf, iv ab	discharge		no	YES
47			✔	0.81	<0.5ml/kg/hr>12hrs			✔		81.5		✔		2	dengue fever with warning signs/AKI	ivf , iv ab ,	discharge		no	no
48	✔			1.3	<0.5ml/kg/hr>12hrs			✔		47.7		✔		2	P VIVAX MALARIA/AKI	ivf antimalarial ab	discharge		no	YES
49	✔			0.81	<0.5ml/kg/hr>12hrs			✔		73		✔		2	AGE with some dehydration/AKI	ivf , iv ab ,	discharge		no	no
50			✔	0.35	<0.3 >12HRS				✔	23.46			✔	3	acute FULMINANT hepatits with aki	ivf iv ab inotrope support	dama		no	yes