A STUDY ON CLINICAL PROFILE AND REVERSIBLE RISK FACTORS ASSOCIATED WITH POOR OUTCOME IN ACUTE KIDNEY INJURY

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CERTIFICATE

This is to certify that the dissertation titled, ***"A Study On Clinical Profile And Reversible Risk Factors Associated With Poor Outcome In Acute Kidney Injury "*** *submitted by,* ***Dr.K.Ramdass,*** *to the Faculty of Pediatrics, The Tamilnadu Dr.M.G.R Medical University, Chennai, in partial fulfilment of the requirements for the award of M.D. Degree (Pediatrics) is a bonafide research work carried out by him under our direct supervision and guidance, during the academic year 2009-2011.*

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CONTENTS

Introduction

Acute Kidney Injury (AKI), the new term heralds a paradigm shift for our conceptualization of the syndrome which is previously called "acute renal failure." AKI retains one word, substitutes a synonym for another, but supplants "failure" with "injury." This substitution may very well redefine the epidemiology of the syndrome.

Acute renal failure (ARF), now increasingly referred to as "acute kidney injury" or AKI, which is characterized by a abrupt (ie., hours to days) impairment of kidney function. The current perception of AKI has evolved along with developments in pathology and clinical biochemistry that have permitted clinicopathologic correlations and early diagnosis[3]. Initial delineation of AKI from the early 20th century centered around specific conditions such as war nephritis[5] , crush injuries[4],and falciparum malaria[6].In 1912, Sir William Osler[7] described several recognizable causes of AKI under the heading of "acute Bright's disease," including toxins, burns, pregnancy, and sepsis. AKI is now inferred to be an increasingly common and potentially disastrous complication in hospitalized patients.

TABLE 1

COMMON CAUSES OF ACUTE KIDNEY INJURY[7a]

PRERENAL	INTRINSIC RENAL	POSTRENAL
Hemorrhage	Glomerulonephritis	Ureterovesicular junction
Sepsis	Poststreptococcal	-obstruction
Cardiac failure	Lupus erythematosus	Ureteropelvic junction
Dehydration	Henoch-schonlein purpura	-obstruction
Hypoalbuminemia	Membranoproliferative	Posterior urethral valves
	Anti-glomerular basement	
	Membrane	Ureterocele
	Hemolytic-uremic syndrome	Tumor
	Cortical necrosis	Urolithiasis
	Rhabdomyolysis	Hemorrhagic cystitis
	Acute tubular necrosis	Neurogenic bladder
	Renal vein thrombosis	
	Tumor infiltration	
	Tumor lysis syndrome	
	Acute interstinal nephritis	

Acute kidney injury is a complex disorder that occurs in a variety of settings with clinical manifestations ranging from a minimal elevation in serum creatinine to anuric renal failure. It is often under-recognized and is associated with severe consequences [8-11]. Recent epidemiological studies demonstrate the wide variation in etiologies and risk factors [8,12-14], describe the increased mortality associated with this disease (particularly when dialysis is required) [8,11,13,15,16], and suggest a relationship to the subsequent development of chronic kidney disease (CKD) and progression to dialysis dependency [8,11,15,17-19]. Emerging evidence suggests that even minor changes in serum creatinine are associated with increased inpatient mortality [20-27]. ARF has been the focus of extensive clinical and basic research efforts over the last decades. The lack of a universally recognized definition of ARF has posed a significant limitation. Despite the significant progress made in understanding the biology and mechanism of ARF in animal models, translation of this knowledge into improved management and outcomes for patients has been limited.

During the last five years, several groups have recognized these limitations and have worked to identify the knowledge gaps and define the necessary steps to correct these deficiencies. These efforts have included consensus conferences and publications from the Acute Dialysis Quality Initiative (ADQI) group [26,28-32], the American Society of Nephrology (ASN) ARF Advisory group [33], the International Society of Nephrology (ISN), and the

National Kidney Foundation (NKF) and KDIGO (Kidney Disease: Improving Global Outcomes) groups [34]. Additionally, the critical care societies have developed formal intersociety collaborations such as the International Consensus Conferences in Critical Care [35].Recognizing that future clinical and translational research in ARF will require multidisciplinary collaborative networks, the ADQI group and representatives from three nephrology societies (ASN, ISN, and NKF) and the European Society of Intensive Care Medicine met in Vicenza, Italy, in September 2004. They proposed the term acute kidney injury (AKI) to reflect the entire spectrum of ARF, recognizing that an acute decline in kidney function is often secondary to an injury that causes functional or structural changes in the kidneys. The group established the Acute Kidney Injury Network (AKIN) as an independent collaborative network comprised of experts selected by the participating societies to represent both their area of expertise and their sponsoring organization. AKIN is intended to facilitate international, interdisciplinary, and intersocietal collaborations to ensure progress in the field of AKI and obtain the best outcomes for patients with or at risk for AKI.

This report describes an interim definition and staging system for AKI and a plan for further activities of the collaborative network which were developed at the first AKIN conference held in Amsterdam, The Netherlands, in September 2005.

REVERSIBLE (MODIFIABLE) RISK FACTORS

Dehydration

Shock[35A]

Hypotension[35A]

Sepsis[35A]

CCF

Hypoalbuminemia

Nephrotoxins

(Aminoglycosides,AmphotericinB,Immunosupressents,NSAIDS,ACEI,I.

Vcontrast media)

Hypertension

NON MODIFIABLE RISK FACTORS

Age

Sex

Pre existing renal diseases

Post cardiac surgery

Burns

GLOBAL SCENARIO

ARF occurs in 2-3%of children admitted to pediatric tertiary care centres and in as many as 8%of infants in the neonatal intensive care unit.(NELSON 18th edition chapter 535)

ICH SCENARIO 2008(Institute of child health , Chennai.**)**

TOTAL ARF CASES 72

MALE 24

FEMALE 48

TOTAL DEATH 41

MALE 10

FEMALE 31

Definition and diagnostic criteria of AKI

Acute kidney injury better represents the full spectrum of acute disorders of renal function, especially in regards to reversible injury. It represents Abrupt decrease of renal function sufficient to result in retention of nitrogenous waste products, as well as loss of regulation of extracellular volume and electrolytes.

For any condition, the clinician needs to know whether the disease is present and, if so, where and when the patient falls in the natural history of the disease. The former facilitates recognition whereas the latter defines time points for intervention. Unfortunately, there has been no uniformly accepted definition of AKI. Studies describe ARF or AKI based on serum creatinine changes, absolute levels of serum creatinine, changes in blood urea nitrogen or urine output, or the

need for dialysis [8,18,27,36-43]. The wide variation in definitions has made it difficult to compare information across studies and populations [44].

Diagnostic criteria

TABLE 2

Diagnostic criteria for acute kidney injury
An abrupt (within 48 hours) reduction in kidney function currently defined as an absolute increase in serum creatinine of more than or equal to 0.3 mg/dl (≥ 26.4 μmol/l), a percentage increase in serum creatinine of more than or equal to 50% (1.5-fold from baseline), or a reduction in urine output (documented oliguria of less than 0.5 ml/kg per hour for more than six hours).

The above criteria include both an absolute and a percentage change in creatinine to accommodate variations related to age, gender, and body mass index and to reduce the need for a baseline creatinine but do require at least two creatinine values within 48 hours. The urine output criterion was included based on the predictive importance of this measure but with the awareness that urine outputs may not be measured routinely in nonintensive care unit settings. It is assumed that the diagnosis based on the urine output criterion alone will require exclusion of urinary tract obstructions that reduce urine output or of other easily reversible causes of reduced urine output. The above criteria should be used in

the context of the clinical presentation and following adequate fluid resuscitation when applicable.

Recognition of AKI requires the delineation of easily measured criteria that can be widely applied. Serum creatinine levels and changes in urine output are the most commonly applied measures of renal function; however, they are each influenced by factors other than the glomerular filtration rate (GFR) and do not provide any information about the nature or site of kidney injury.

There is accumulating evidence that small increments in serum creatinine are associated, in a variety of settings, with adverse outcomes [20-27] that are manifest in short-term morbidity and mortality and in longer-term outcomes, including 1- year mortality [22-24]. Current clinical practice does not focus much attention on small increments in serum creatinine, which are often attributed to lab variations. However, the coefficient of variation of serum creatinine with modern analyzers is relatively small and therefore increments of 0.3 mg/dl (25 μmol/l) are unlikely to be due to assay variation [45].

A time constraint of 48 hours for diagnosis was selected based on the evidence that adverse outcomes with small changes in creatinine were observed when the creatinine elevation occurred within 24 to 48 hours [22, 23] and to ensure that the process was acute and representative of events within a clinically relevant time period. In the two aforementioned

studies, there was no distinction of underlying CKD or *de novo* AKI. However, in the study by Chertow and colleagues [20], the odds ratio for mortality with a change in creatinine of 0.3 mg/dl (25 μmol/l) was 4.1 (confidence interval 3.1 to 5.5) adjusting for CKD. There is no requirement to wait 48 hours to diagnose AKI or initiate appropriate measures to treat AKI. Instead, the time period is designed to eliminate situations in which the increase in serum creatinine by 0.3 is very slow and thus is not 'acute.'

The need for including urine output as a diagnostic criterion is based on the knowledge of critically ill patients in whom this parameter often heralds renal dysfunction before serum creatinine increases.

The proposed diagnostic criteria for AKI are designed to facilitate acquisition of new knowledge. The goal of adopting these explicit diagnostic criteria is to increase the clinical awareness and diagnosis of AKI. It is recognized that there may be an increase in false-positives, so that some patients labeled with AKI will not have the condition. There was consensus that adopting the more inclusive criteria is preferable to the current situation, in which the condition is under-recognized and many people are identified late in the course of their illness and potentially miss the opportunity for prevention or application of strategies to minimize further kidney damage.

Staging/classification

The goal of a staging system is to classify the course of a disease in a reproducible manner that supports accurate identification and prognostication

and informs diagnostic or therapeutic interventions. The group recognized that a number of systems for staging and classifying AKI are currently in use or have been proposed [48]. The RIFLE (Risk, Injury, Failure Loss, and End-stage kidney disease) criteria [32] proposed by the ADQI group were developed by an interdisciplinary, international consensus process and are now being validated by different groups worldwide [43,44]. However, according to data that have emerged since then, smaller changes in serum creatinine than those considered in the RIFLE criteria might be associated with adverse outcomes [20-25]. Additionally, given the consensus definition for AKI (Table2), RIFLE criteria have been modified so that patients meeting the definition for AKI could be staged .There was a conscious decision not to include the therapy for AKI (that is, renal replacement therapy [RRT]) as a distinct stage because this constitutes an outcome of AKI.

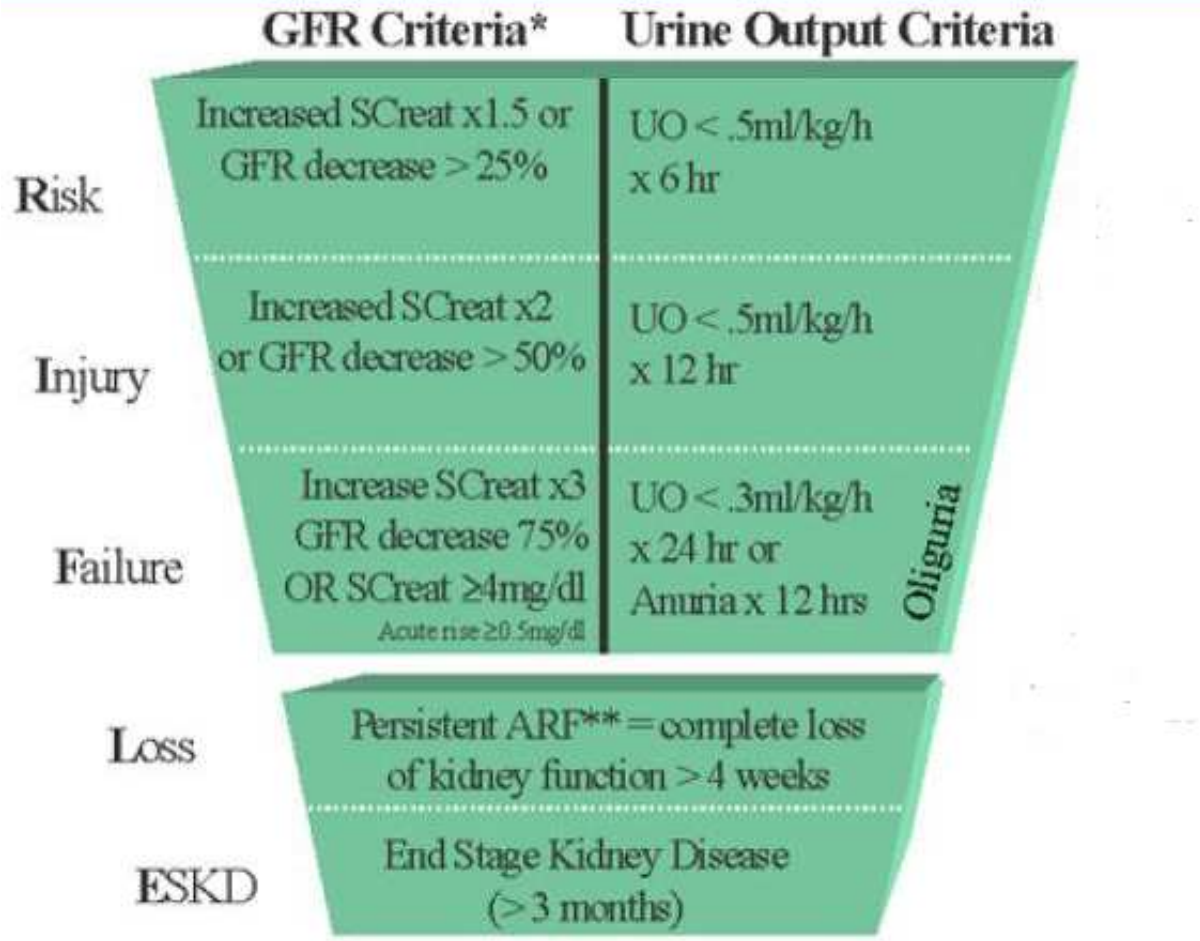
Table 3

Classification/staging system for acute kidney injury

Stage	Serum creatinine criteria	Urine output criteria
1	Increase in serum creatinine of more than or equal to 0.3 mg/dl (≥ 26.4 μmol/l) or increase to more than or equal to 150% to 200% (1.5- to 2fold) from baseline	Less than 0.5 ml/kg per hour for more than 6 hours

2	Increase in serum creatinine to more than 200% to 300% (> 2- to 3 fold)from baseline	Less than 0.5 ml/kg per hour for more than 12 hours
3	Increase in serum creatinine to more than 300% (> 3-fold) from baseline (or serum creatinine of more than or equal to 4.0 mg/dl [≥ 354 μmol/l] with an acute increase of at least 0.5 mg/dl [44 μmol/l])	Less than 0.3 ml/kg per hour for 24 hours or anuria for 12 hours

RIFLE CRITERIA



ARF and oliguria which were considered synonymous, is not the routine in present day knowledge. Children with ARF following aminoglycoside therapy and septicaemia can present with non oligurie ARF. If anuria or oliguria alone is considered essential as the pointer for the diagnosis of ARF, clinically these children will be missed. So a strong suspicion on the back ground of such situation for nonoliguric ARF is essential to make the clinician to do biochemical evaluation periodically and for early detection of nonoliguric or high output ARF.

Definition and Values in children.

Normal urine output is more than 3 ml/kg/hr.Oliguria is urine output less than 1 ml/kg/hr. Anuria is urine output less than 0.5 ml/kg/hr

With the present available knowledge it is clearly understood that to monitor renal function, it is important to use biochemical studies as well as measurement of urine volume which will help us not to miss non oliguric renal failure.

The causes of ARF are many, pre renal failure is essentially due to reduction in perfusion of kidney resulting in renal function. Intrinsic renal failure results from diseases of kidney, while post renal failure is essentially due to obstruction disorders.

In prerenal ARF, resulting from decreased renal perfusion, the main pathogenetic event is a reduction in total or effective circulating blood volume. Evidences of kidney damage is absent. Reduction in intravascular volume causes reduction in cardiac output and hence reduction in renal cortical blood flow and hence reduction G.F.R. If conditions leading on to this reduction of perfusion of kidney is corrected within a stipulated time, renal function become normal. On the contrary, if defective perfusion persists, incipient renal failure sets in , which is one step higher than prerenal failure but one step lower than intrinsic renal failure.

In Intrinsic renal failure, glomeruli, tubules, interstitium and blood vessels can be affected either individually or collectively. Acute glomerulonephritis usually the common cause of A.R.F in older children, whereas HUS is a common cause of A.R.F in toddlers[49]. Acute tubular necrosis (ATN) is a syndrome of A.R.F in the absence of arterial or glomerular lesion. The main mechanism is necrosis of tubular cells. Alteration in intrarenal hemodynamics, tubular obstruction and passive back flow of glomerular filtrate across injured tubular cells into the peritubular capillaries are the proposed events in the pathogenesis of A.T.N other than necrosis of tubular cells.

Acute interstitial nephritis results from hypersensitivity reaction to a therapeutic agent or an infective agent. Tumors essentially produces A.R.F by infiltration or by intratubular obstruction due to uric acid crystals. Acute renal failure due to developmental anomaly and hereditary nephiritis are essentially due to intravascular volume contraction following vomiting , diarrhoea etc.,

Obstruction causing post renal failure needs energetic measures in the era of sonology. This cause should be eliminated in every child with ARF. One should know that the dialation of upper collecting system will manifest usually after several days of acute uretheral obstruction.

The precipitating diseases form the major presenting feature. Renal failure itself can manifest with oliguria, pallor, edema, anaemia, hypertension, vomiting and lethargy. Acute hypertension in A.R.F. can manifest as

hypertensive encephalopathy,congenstive cardiac failure, pulomonary edema, arrhythmias, seizures, coma and G.I bleeding are the manifestations of complications of A.R.F.

In a child with renal failure, diagnosis is often made with history. With vomiting, diarrhoea, fever, dehydration and renal failure , one can doubt the existence of prerenal azotemia. But this background history may point towards , H.U.S and renal vein thrombosis RVT also. Recent , past history of infection denotes post infective glomerulonephritis P.I.G.N. Features of recurrent abdominal pain, arthiritis and arthralgia, jaundice , wheeze and skin rashes are suggestive of vasculitis.Triad of features with abdominal pain, hematuria and renal mass will give a clue towards R.V.T. Features of septicaemia and administration of aminoglycosides should arouse an index of suspicion of nonoliguric A.R.F.

In nonoliguric A.R.F it is only the index of suspicion that will allow the clinician to diagnose the condition. If urine volume alone is taken in to account, we will miss this condition. On the background of septicemia and aminoglycoside therapy, if a child is not showing improvement generally and if the child develops refractory metabolic acidosis or seizures, attention to diagnose ARF by biochemical monitoring should be done.

In oliguric A.R.F., the following approach is made. All these children had the following investigations done. Urine analysis , blood Urea, s creatinine,

s.electrolytes, s.total protein, s.albumin, s.globulin, chest X-ray and ultrasonogram, urine should be sent for sodium and fractional excretion of sodium FeNa and renal failure index (RFI) are calculated with urine sodium and serum creatinine. The formulae that are used for calculating FeNa and RFI are as follows.

$$\text{FeNa+} = \frac{\text{Urine sodium/ Serum sodium}}{\text{Urine craetinine/serum creatinine}} \times 100$$

$$\text{R.F.I} = \frac{\text{Urine sodium}}{\text{Urine Creatinine/Plasma creatinine}}$$

A child with oliguric A.R.F presenting with low urinary sodium indicates that kidneys are acting normally and there is maximum proximal renal reabsorbtion of sodium and water on the background of ECF contraction. It is popularly said, that kidneys are acting super normally in this situation. The resultant decrease of urine sodium less than 20mEq/litre, signifies the event. Similarly FeNa and R.F.I less than 1 reflects normal functioning of the kidney.

In intrinsic renal failure, because of the intrinsic renal defects and tubular damage, the conservation of sodium is defective resulting in increased urinary

sodium more than 40mEq/lit and FeNa and R.F.I are more than 1, which reflects the diseased kidney.

If prerenal failure is diagnosed by history, Clinical methods and urine sodium, every attempts should be made to improve the perfusion of the kidney. Perfusion of the kidney is improved by adequate fluid therapy. If haemorrhage is the main event, colloids are highly useful. If pump failure like C.C.F. including the use of inotropic agents correct the prerenal failure. By this time investigations like ultrasonogram, chest X-ray, will give further clues. A dilated heart with A.R.F. will give the indication that further fluid therapy is hazardous. An obstructed collecting system in U.S.G will point towards post renal cause. Bilateral contracted kidneys will indicate that it is a situation of acute on chronic renal failure. Biochemical investigations like blood urea, serum creatinine, serum electrolyte will give the assessment of the renal failure. Rapidly increasing blood urea and serum creatinine, and increasing serum potassium and decreasing bicarbonates may indicate hypercatabolic state, which usually occurs in situations like septicaemia and post operative situations. Coagulation profile, liver function tests, investigations for leptospirosis are the other investigations done in concerned situation. Biopsy in A.R.F is indicated only when the natural course of A.R.F is not followed.

Treatment is essentially decided by the type of renal failure. In prerenal situation, fluid therapy, blood or plasma transfusion, antibiotics, are needed.

Treatment of pump failure with inotropic agent etc., prevent the child from going in for intrinsic renal failure.

In intrinsic renal failure, the treatment is decided by the type of disease, presentation and complication. In acute interstitial nephritis, basically the elimination of the offending agent, treatment of hypertension if present. Fluid modulation, and dialytic therapy may be needed. Acute tubular necrosis of nonoliguric type is effectively treated with conservative line of management with fluid modulation, dietary adjustment, antibiotics, antihypertensives on need and periodic bio chemical monitoring. In oliguric A.R.F, antibiotics, antihypertensives on need, dietary modulation and fluid adjustment are the main consideration. Majority of them benefit from conservative line of the treatment but a significant number of children need dialytic therapy. Dialysis in ARF is not decided by a single factor. Edema, hypertension, congestive heart failure and acidosis, which are refractory, need dialysis. Increasing urea and creatinine, hyperkalemia are definite indication of dialysis. Dyselectrolytemia which cannot be corrected by fluid therapy gets the benefit with dialysis. Majority of them are better treated with intermittent peritonieal dialysis. Hemodialysis is indicated for ARF in children, only when the child is having hypercatabolic ARF and when peritoneal dialysis procedure is not possible like very recent abdominal surgery or severe abdominal injury.

Phospate binders like aluminium hydroxide get to reduce hyperphosphatemia, calcium supplementation especially when on dialytic therapy are indicated in the management of ARF. Meticulous control of infections with adequate and appropriate antibiotic therapy and aggressive prevention of infection is mandatory. Treatment of complications like pulmonary edema with furosemide and dialytic therapy, hypertensive encephalopathy with sublingual nifedipine and oral antihypertensives along with I.V furosemide are essential. Hyperkalemia is treated with I.V. calcium gluconate , I.V sodium bicarbonate, I.V dextrose with insulin and dialytic therapy.

Children with ARF are given a diet with daily recommended allowance of protein and calories. No need to restrict protein in ARF, as children are in need of protein, irrespective of being on dialytic or non dialytic therapy. But during peritoneal dialysis 3-5 gms/kg of protein has to be given since there is loss of essential aminoacids through P.D. fluid. Salt and water are initially reastricted, and once edema resolves, hypertension is controlled and dieresis occurs, salt and water are gradually modified towards normal intake.

DIALYSIS

Peritonial dialysis is a form of renal substitution therapy. Hemodialysis is the other mode. Peritonial dialysis almost certainly represents solute and fluid exchange between peritoneal capillary blood and dialysis solution in the peritoneal cavity across the peritoneal membrane which consists of a vascular wall, the interstitium, the mesothelium, and adjacent fluid films, solute movement follows the physical laws of diffusion and convective transport, whilst fluid shifts relate to osmosis. The crucial components of the peritoneal dialysis system are, therefore peritoneal blood flow, the membrane and compensation and flow rate of peritoneal dialysis fluids. It is simple, safe and useful procedure, which can be started even at a peripheral center with very little facilities. The convenience, simplicity, and relative safety of P.D. has allowed the paediatricians to begin dialysis in children as soon as it is needed.

Two factors may make peritoneal dialysis more suitable than hemodialysis for critically ill pediatric patients. One is the ready across to the peritoneum versus typically more difficult vascular access, and the other is the better tolerance of peritoneal dialysis by hemodynamically unstable children.

Complications of peritoneal dialysis are many like block in catheter, hemorrhagic effluent, peritonitis, electrolyte imbalance, injury to internal viscera, etc.,

PHYSIOLOGY OF PERITONEAL DIALYSIS :

Essential aspects involved in PD include diffusion , osmosis, solvent drag and siphon effect. Active transport is not a major feature of peritoneal membrane[52]. The peritoneal membrane plays a major role in this respect. Uremic toxins from a point of higher concentration in the blood diffuses through the membrane into the dialysis compartment, which is the peritoneal cavity filled with the dialysis fluid. Urea, Creatinine, Uric acid, sodium, potassium, calcium diffuses in this way. By maintaining the osmolity of dialysate fluid in the dialysis compartment creates an osmotic effect. This osmotic effect is an essential aspect of ultrafiltration.

The peritoneal surface area in children which is roughly for an infant around 450 sq.cm per kilogram body weight, which is roughly twice that of the adults. The movement of the solutes between peritoneal capillaries and dialysis fluid in across a number of resistance sites, the capillary wall is one of the major resistance sites to dialysis exchange. The peritoneal interstitium is another resistance site. Dehydration of the peritoneal gel by hypertonic fluid increases resistance where as peritoneal mesothelium produces little resistance only.

By increasing the dwell time i.e., the time allowed for all physiological process to occur , the time of contact between blood and dialysis compartment,

the solute clearance is made better. By decreasing the dwell time, its aids in increasing fluid removal. As a standard procedure, by keeping dwell time at 60 min, the solute and solvent clearance are made optimal. The PD fluid put in to the peritoneal cavity drains out by siphon effect.

Peritioneal dialysis is useful when long dwell time is given because molecules are given time to equilibrate between plasma and dialysate, middle molecules (molecular weight of 500 to 5000) are removed better by long dwell time dialysis.

Peritonitis is not a contraindication for dialysis and in fact in acute peritonitis clearance is better whereas in chronic peritonitis due to resultant adhesions, clearance is poor.

Factors that may increase the peritoneal clearance includes[51,53,54]

a. Increase in flow rate
b. Increase in temperature of PD fluid from 20˚ C to 37˚ C increases the solute clearance by 30-35% [51]
c. Increase in dextrose concentration.
d. Use of vasodialator agents.[54]
e. Alternating hypertonic with isotonic dialysis solution

Increasing the temperature of PD fluid from 20 degree C to 37 degree C increase the solute clearance by 30-35%. Use of vasoactive substances like

sodium nitroprusside, furosemide, isoproterenol intraperitoneally and dopamine systemically can increase clearance. Joy M-peter et al., demonstrated statistically significant increase in the clearance of urea and uric acid when dopamine was used systemically in the dose of 4µg/kg/min to achieve splanchnic vasodilatation[54].

Factors decreasing the peritoneal clearance includes[50]

a. Use of dialysate at room temperature
b. Use of vasoconstrictor agents
c. Presence of vascular disease
d. Systemic arterial hypertension
e. Paralytic ileus

Types of Peritoneal dialysis (IPD)

Intermittent peritoneal dialysis (IPD)

Originally this technique was called periodic peritoneal dialysis. (Bone et al., 1962). It was the one commonly practiced in India. The basic technique of P.D rests on the intermittent irrigation of the peritoneal cavity with dialysis fluid through a single peritoneal catheter. The cycle includes inflow of the fluid into the peritoneal cavity, a dwell phase of variable duration and drain phase. The cycle is repeated as many times as is needed. The duration of one dialyisis varies from 12 hours to 96 hours which depend on the clinical and bio-chemical

parameters. A standard protocol used exchange cycles with 5-10 minutes infusion time, 30-40 minutes dwell time and 10-20 minutes draining time[52]. For each cycle 25-50ml/kg of dialysate fluid is used which is decided on the age, weight and severity of respiratory distress.

Continuous ambulatory peritoneal dialysis (CAPD)

The basic principle is permanent bathing of peritoneal cavity by dialysate solution which are exchanged every 4 hours in the day time and once at night. Catheter used is tencknof catheter. The long dwell time is used in CAPD to allow for complete equilibrium of solutes between plasma and dialysate.[55]

Continuous cyclic peritoneal dialysis (CCPD)

CCPD involves three or four automatic exchanges of fluid at night while the patient sleeps and prolonged retention of fluid during the day. The solute clearance are similar to CAPD . CCPD is usually chosen over CAPD because of reduced parental tasks[56]. In adolescents, CAPD or CCPD is chosen over HD because of the ability of the patient to perform the procedure.

Nightly intermittent peritoneal dialysis (NIPD)

Nightly peritoneal dialysis is a regimen performed every night without a long dwell day time exchange(CCPD without diurnal exchange). It is used in patients with recurrent abdominal hernias, prolapsed bladder, rapid glucose

absorption resulting in poor ultrafiltration, abdominal discomfort and chronic hypertension.

Tidal peritoneal dialysis

This is a technique where after the fill of peritoneal cavity, only a portion of dialysate is drained and is replaced by fresh dialysis fluid with each cycle leaving the majority of dialysate in constant contact with peritoneal membrane until the end of dialysis session when the fluid is drained as completely are possible. It is 20% more efficient than nightly peritoneal dialysis[56].

INDICATIONS OF IPD

1. **Acute renal failure.**[50]

   a. Anuria 48hrs , oliguria 72hrs.

   b. Hyperkalemia : Serum potassium > 7.0 mEq/L

   c. Intractable acidosis : serum bicarbonate < 12 mEq/L

   d. sever azotemia : Blood urea nitrogen >= 150 mg/dl.

   e. Fluid overload : Usually with hypertension, CCF or acute pulmonary edema.

   f. Symptoms of uremia : Encephalopathy, pericarditis, intractable vomiting, hemorrhage

   g. Hyponatremia, hypocalemia, hyperphosphatemia

h. Removal of fluid for optimal nutrition, transfusion

2. Chronic renal failure

a. Awaiting hemodialysis or transplant.[52]

b. Patients with unstable cardiovascular system.

c. Acutre on chronic renal failure

3.Removal of Toxins

1. a. **Antimicrobial.**

Gentamicin

Amikacin

Kanamycin

Cephalexin

Colistin

Isoniazid

Ethambutol

b. **Analgesics and sedatives.**

Aspirin.

Barbiturates.

c. **Cardio vascular drugs**

Quinine

Methyldopa

Nitroprusside

2. Neem oil poisoning

3. Copper sulphate and salicylate poisoning.

4. Interactable congestive cardiac failure[55]

5. Interacteble edema in nephritic syndrome.

6. Inborn errors of metabolism

a. Congenital urea cycle enzymopathies with resultant hyperammonemic coma.

b. Maple syrup urine disease

c. Propionic acidemia

d. Congenital organic academia.

Mathews et al., in 1990 demonstrated that peritoneal dialysis is effective in new born period in the treatment of inborn errors of metabolism and renal failure.

CONTRAINDICATIONS FOR PERITONEAL DIALYSIS.

The lack of an adequate or intact “peritoneal cavity” is mainly the contraindication for peritoneal dialysis.

1. Neonates with diaphragmatic hernia, omphalocele or gastroschisis.

2. Recent abdominal surgery with a draining abdominal wound.

3. Children with bleeding disorder without giving correction factor

4. Severely infected anterior abdominal wall

5. Severe bony deformities of the back preventing the child from having a correct posture

6. Hypercatabolic states where in peritoneal dialysis becomes ineffective

7. Ventriculo peritoneal shunts in hyperocephalus.

8. Colostomy and fecal fistulas.

9. Intestinal obstruction with gross abdominal distension.

10. Extensive intra abdominal adhesions.

PROCEDURE FOR INTERMITTENT PERITONEAL DIALYSIS.[57]

Under strict precautions, the procedure is to be done. The abdomen is palpated for any organomegaly. One should make sure that bladder is empty. Infiltrate 2.5 ml of 1% xylocaine anywhere 1 cm away from and around umbilicus, preferably infraumblical region (i.e.,) at the level of linea alba since it is avascular. In new born, prefer to take supraumblical region, since the distance between the entry of catheter and right iliac fossa is short. Peritoneal cavity should be distended with the calculated peritoneal fluid(i.e., 30-50ml/kg) through 18 gauge needle. This step is necessary to distend the abdomen just before introduction of the catheter, so that the intestine will move away from the midline . In this case of massive ascites, let out 100-200 ml of ascitic fluid and then start input of P.D. fluid. Slowly increase upto 30ml/kg. Head should be raised so that abdominal wall is taut. A small incision is made with 11 size blade. Through this wound pass the catheter by applying little pressure with a twisting motion. Entrance of the trip into the peritoneal cavity is indicated by a sudden decrease in resistance and by the appearance of dialysis fluid in the catheter. The stylet is removed slowly and catheter is advanced as far as possible into the right (preferably or left iliac fossa. The catheter is connected into the administration through small connection tube and dialysis fluid is flown.

The block that can occur is due to proteinaceous materials or blood clot, Heparin is added even if the peritoneal fluid is hemorrhagic, since it has a local action and no systemic action[58]. Potassium chloride is added to P.D. fluid according to the need of the child. If hyperkalamic, skip this step. Then after 10^{th} exchange do serum electrolytes and add KCI according to the need. Initially exchange are for small dwell time. The duration of dialysis range from 24 hours to 96 hours depending upon the clinical and biochemical parameters. Dialysis for 24 hours is the ideal choice. 48 hours is used for some children needing longer dialysis, 72-96 hours dialysis is used only in extreme situations like persistent uremia symptoms and poor cardiac status preventing hemodialysis. The longer the duration , more is the chance of infection.

The catheter so placed is secured in its position by silk purse string sutures and wound is closed after applying anti-septic cream. Every attempt should be made to keep the system clean and sterile. Unnecessary handling of the system will increase the chance of infection.

1. PRESCRIPTION

1. 30-50 cc/kg fluid is used for every exchange. Reduction in fluid should be done only in children with severe respiratory distress.

2. Usually isotonic fluid is used (even though 1.7% glucose containing PD solution produce 355 milliosmoles/litre which is necessary for fluid

removal, for all other practical purposes 1.7 % solution is considered isotonic), Hypertonic solution (4% and 7%) is only for fluid removal in interactable edema, pulmonary edema and congestive cardiac failure. Always it should be used with caution because of the chances of infection. Altering hypertonic and isotonic exchanges are ideal than using hypertonic solution alone.

3. Conditions wherein hypokalemia predominate, dialysis is done using 4mEq/L of potassium (2ml KCI/"L" of peritoneal dialysis fluid). Conditions where hyperkalemia is present , nil potassium exchanges are given and potassium is added only when serum potassium is less than 4.5mEq/L. In this conditions where serum potassium estimation is not possible or if dialysis is started awaiting serum potassium results, 2 mEq potassium per litre(1ml KCL/litre) from 11^{th} exchange is added always labelling the bottle about the added potassium is a must for avoiding iatrogenic hyper or hypokalemia.

4. Heparin 125 units/litre is added to keep the system free from clotting. Fortunately, there is no systemic heparinization following peritoneal dialysis unlike hemodialysis patients can undergo surgery or renal biopsy immediately.

5. The standard schedule of dwell time is 60 minutes. But while treating pulmonary edema and congestive cardiac failure it can be reduced to 30 minutes. Children with renal failure following chronic tubulointerstitial disease, presenting with both renal failure and dehydration, dwell time is kept for 120 minutes.

6. Addition of antibiotics (ampicillin 250mg/ litre or Gentamycin 8mg/litre of PD fluid) is done only in situations of peritonitis.

Outcome of A.K.I depends on the etiology, associated complications, and the appropriates medical interventions. Delay in seeking medical care, will definitely play a major role in deciding morbidity and mortality.

Hence energetic measures are needed in managing a child with A.K.I.

REVIEW OF LITERATURE

The epidemiology of acute renal failure in children is influenced by many factors, the principle one being the standard of living and health care in the community. The incidence and etiology of A.R.F. are variable depending upon the age of the subjects and the diverse ecological factors. The incidence is extremely high in all poorly developed countries mainly because of increased incidence of skin infections and untreated infectious diarrhoeas.

1.**Arora.P et al.,**[55] In the study by P.Arora et al., from the department of nephrology, Sanjay Gandhi Postgraduate Institute of Medical Sciences, Lucknow, out of 52 children with A.R.F boys predominated over girls. In this study H.U.S was the commonest cause of A.R.F (30.8%) followed by A.T.N (28.84%) and A.G.N (13.23%). Oliguria was seen in 46.6% and anuria in 53.6%, dialysis was started in 35 out of 52 children. The mortality was 34.6% among the children who died, 7 children had H.U.S, 4 children of post surgical state, 3b children of A.T.N, 3 children of A.G.N and 1 of obstructive uropathy died, C.N.S and respiratory complications were bad prognostic factors.

2.James C.M Chan et.al.,[59] In the study of James C.M Chan, MD from the department of Nephrology, Children's Hospital, Washington, 20 children were analysed to study the mortality in peritoneal dialysis. The mortality was concentrated primarily in control and correction of peritoneal factors , adequate control of post renal factors and with efficient treatment of intrinsic renal

failure, the overall mortality and morbidity of A.R.F in children can be reduced to vary minimal percentage. The pleasure of seeing A.R.F children recovering is definitely one of the important satisfying event in the career of every paediatrician and pediatric nephrologists.

3.Patrick Niaudet et al.,[60]In a study conducted by Patrick Niaudet et al., from the department of nephrology, pediatric hospital, France. Out of 125 children, H.U.S was the leading cause of A.R.F and was seen in 67 children. Next in the list was acute nephritic syndrome, accounting to 18 cases. Among the 67 children with H.U.S 35 recovered. The modality of treatment was mainly dialysis. 123 children were started with dialysis. Among the 67 children with H.U.S, 9 children died. 1 child died out of 4 children with renal vein thrombosis (R.V.T). 3 children out of the 30 children with ATN died.

4.Kandoth P.W et, al.,[58]In the study conducted by P.W Kandoth et al from department of paediatrics, T.N medical college and B.Y.L Nair Hospital, Mumbai, ninety eight children requiring dialysis therapy were analysed. Renal causes were predominant accounting to 65%. In them, the preranal and post renal causes responsible for A.R.F were 19% and 16% respectively. A.G.N was seen in 13 children, A.W.D with hypovolemia in 4 and A.T.N in 6 Five causes were due to H.U.S .All these children had oligo - anuria, hypertension in 23% infection in 47%, and neuro psychiatric manifestations in 20%. Peritoneal dialysis , was carried out in 95%. The factors associated with poor prognosis

included female sex, age less than one year, neurological manifestations and hypotension. Teh overall mortality was 41.5%. There is a male preponderance in this study, in the ratio of 1.8:1.

5.Shah B.V et al.,[61] 66 children with A.R.F were analysed by B.V.Shah et al.,from the department of medicine and nephrology K.E.M Hospital ,Parel, Mumbai. Among them 62% had mortality. Four major factors that had been identified as having adverse influence on the prognosis were age , etiology, duration of oligo-anuria and uremic complications like infections, gastrointestinal and neurological problems. Survival was poor in less than one year of age accounting to 22.2%. The major causes leanding to A.R.F were gastroenteritis, H.U.F in younger children and in order children due to A.G.N. When the duration of oliguria was short, outcome was favourable gastrointestinal neurological and infectious complications correlated with a poor outcome. Out of 22 children who had convulsions, only 5 children survived. The infectious complications in the form of respiratory infection was seen in 20 children. U.T.I was seen in 8 children , peritonitis in 3 children and pyogenic meningitis in 4. Overall, the infections complications were seen in 24 children (36.4%) and among them only 4 (16.6%) survived.

6.N.Gallego et al.,[62] From the Department of Nephrology Hospital, Madrid, Spain analyse 138 cases of A.R.F. The overall mortality was 48.3%. There was no difference in mortality with reference to sex. The age group was

similar among survivors and non survivors. But he observed that the mortality was higher in newborns (83.3%). The mortality was higher in patients who underwent some surgical procedure prior to A.R.F.

7.Mahakur A.C et al.,[63] 175 children with A.R.F who underwent dialysis were analysed by A.C Mahakur et al., from the department of Nephrology , S.C.B. Medical College Cuttack. Acute diarrhoeal disease, acute glomerulonephritis and viperidine snake bites were the common causes of A.R.F in Orissa.

8.Alan S.Jones et al.,[64] Department of child Health and Pathology, Missouri reported 20 newborn babies out of total , had acute tubular necrosis as a result of perinatal asphyxia, hypoxemia or shock secondary to hyaline membrane disease, bacterial infection or necrotising enterocolitis. Seven infants of the study group were treated with peritoneal dialysis.

9.Arora et al.,[65] Department of Nephrology, SGPGIMS, Lucknow study showed 35 patients out of 52 children with A.R.F required dialysis support for a mean duration of 18 days (2-90)days. The mortality was 34.6%.

10.Shah et al.,[61] Department of Medicine and Nephrology, KEM Hospital, parel Mumbai and reported infection an the most serious complication of ARF, constituting a leading cause of death. Gastrointestinal

25.6% and neurological complications 22 % were also noted, which adversely affected the outcome.

Shah et al., KEM hospital, Mumbai reported that infants comprised 29.0% of cases of A.R.F 36% reported by Deb et al., but higher percentages in studies by Jyothimurugan et al., 56.6% and 43% kandoh et al., in their studies respectively.

11.Srivastava et al .,[66] Department of Nephrology, AIIMS, New Delhi, reported 68.4% of A.T.N and 31.5% of A.G.N in children presenting with A.R.F required peritoneal dialysis. Chugh et al., reported 55% of ATN and 8.75 % of AGN were managed with peritoneal dialysis in nephrology unit in Chandigarh which included adults and children.

12.Chug et al.,[67] reported 22.% of hemorrhagic dialysate. 4% of peritonitis and 1.16% of perforation of gut as major complications associated with peritoneal dialysis. Jyothimurugan et al., showed 48% of children had blocked catheters, 4% leak at insertion site, 14.3 peritonitis.

13.McNeil et al.,[68] Childhood acute renal failure: 22-year experience in a university hospital in southern Thailand. Department of Pediatrics, Faculty of Medicine, Prince of Songkla University, Hat Yai 90110, Thailand.The case records for children 1 month to 17 years of age who were diagnosed as having acute renal failure between February 1982 and December 2004, in the

Department of Pediatrics, Songklanagarind Hospital, in southern Thailand, were reviewed.Results were: A total of 311 children with 318 episodes of acute renal failure were included, that is, 177 boys (55.7%) and 141 girls (44.3%), 1 month to 16.7 years of age (mean age: 7.6 +/- 5.1 years; median age: 7.8 years). The causes of acute renal failure in each age group were significantly different. Overall, sepsis was the major cause of acute renal failure, accounting for 68 episodes (21.4%), followed by hypovolemia, poststreptococcal glomerulonephritis, systemic lupus erythematosus, and infectious diseases. Renal replacement therapy was performed in 55 cases (17.3%). The overall mortality rate was 41.5%. Logistic regression analysis showed that disease groups and creatinine levels were significant independent predictors of outcomes.The incidence of acute renal failure in Songklanagarind Hospital was 0.5 to 9.9 cases per 1000 pediatric patients, with a mortality rate of 41.5%.

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STUDY JUSTIFICATION

- 50%mortality rate seems to have remain unchanged despite technical process
- Among a number of children who die with terminal renal failure there are many with preventable causes .Early recognition of the cause and its prevention would reduce the mortality

AIM OF STUDY

This study aims at finding out the clinical profile and reversible(modifiable) risk factors associated with poor outcome in children with acute kidney injury seen in this part of the country in a referral pediatric centre at Chennai, Tamilnadu

SUBJECTS AND METHODS

1. Methodology

Study Place:

Institute of child health&hospital

for children, chennai 600008

Study Design:

Descriptive Study/Nested case control study

Study Period:

Dec.2009 to Oct 2011

Inclusion Criteria:

Children 1 month-12years presenting with clinical and biochemical evidence of Acute kidney injury characterised by an abrupt (48 hours) reduction in kidney function which is defined as an absolute increase in sr.cr ≥0.3mg/dl(26 μ mol/l) or more than 50% increase i.e., (1.5*baseline value) in sr.cr or a reduction in urine output (<0.5 ml/kg/hr) for more than 6 hours.

Exclusion Criteria:

Those children with chronic renal failure or who had prior dialysis therapy, Pre existing renal diseases.

Study Population:

All children satisfying the inclusion and exclusion criteria.

Cases:

All subjects With Poor Outcome

Controls:

All subjects With Recovery

Sample Size: n=100

(based on 50% mortality 20% precision rate with 5% alpha error)

2.MANEUVER

- All eligible children with defined criteria are recruited in to the study after obtaining parental consent. Using a standard data extraction form, information is abstracted in detail.
- After complete evaluation of the patient with a detailed history ,clinical examination and necessary investigation ,the cases were followed with standard line of treatment and the outcome and various factors affecting the final outcome were analysed.

3.STATISTICAL ANALYSIS

- Proportions and mean with standard deviation of outcome measures were arrived at as applicable. Odds ratio with 95% confidence interval were

calculated and to adjust for confounding factors, adjusted odds ratio with 95% confidence interval will be arrived at by Multivariate analysis.

- Applying chi-square test, logistic regression
- $P<0.05$ was considered as statistically significant.

4. ETHICS

Patient consent and IRB approval was obtained.

OBSERVATIONS

Of 100 children included in our study, with Acute kidney injury were due to varied etiology.

TABLE 1

AGE DISTRIBUTION

Age group	No.Of children	Percentage
1 month - <1 year	37	37%
1 year- <4 years	21	21%
4year- <8 years	28	28%
8-12 years	14	14%

Most of the children were under 1 year age group accounting 37% of total children. Next major group was noted in 4-8 years age group with 28% of total children. The minimum age of the child in our study was a 35 days old child. And the maximum aged child was 12 years old.

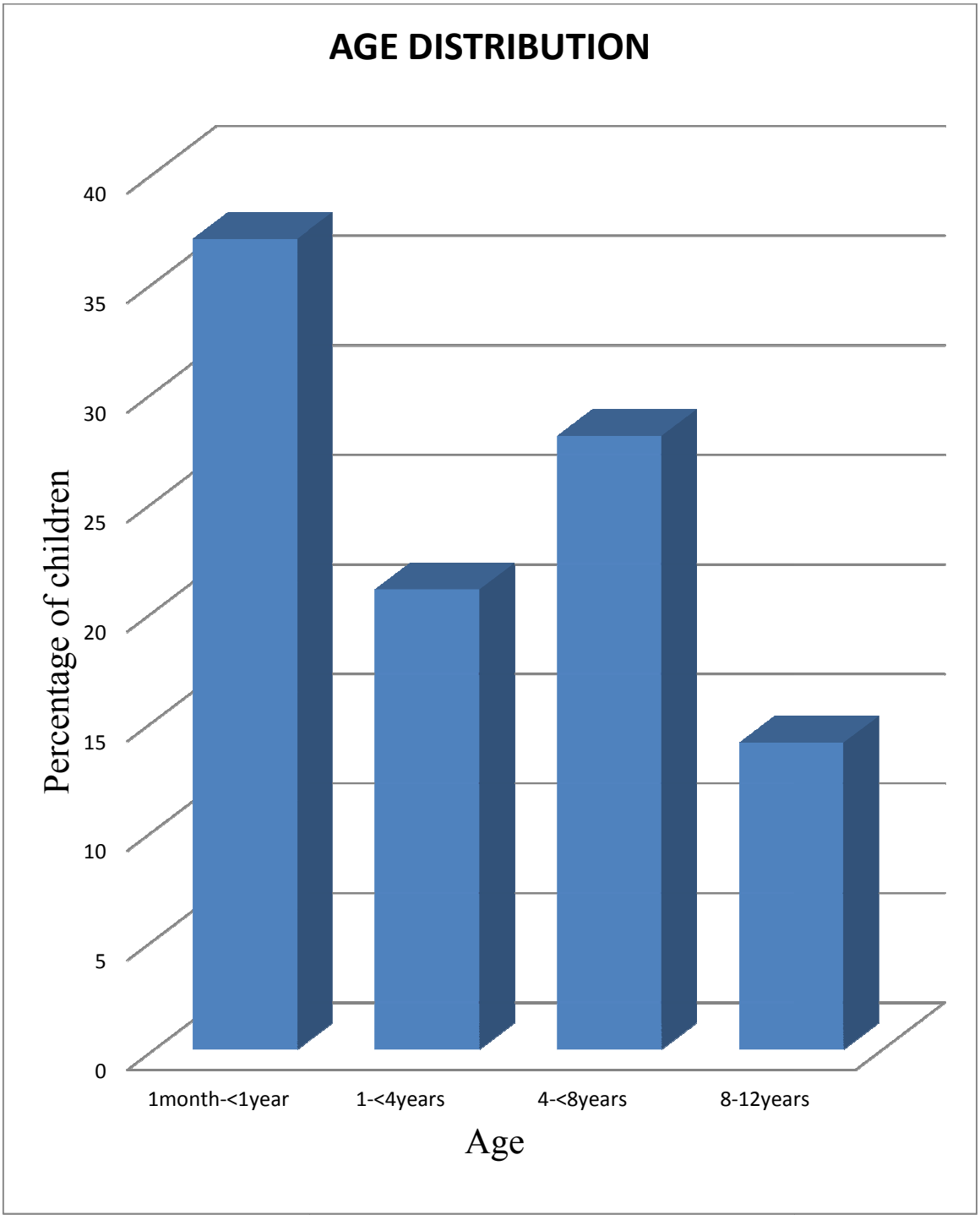
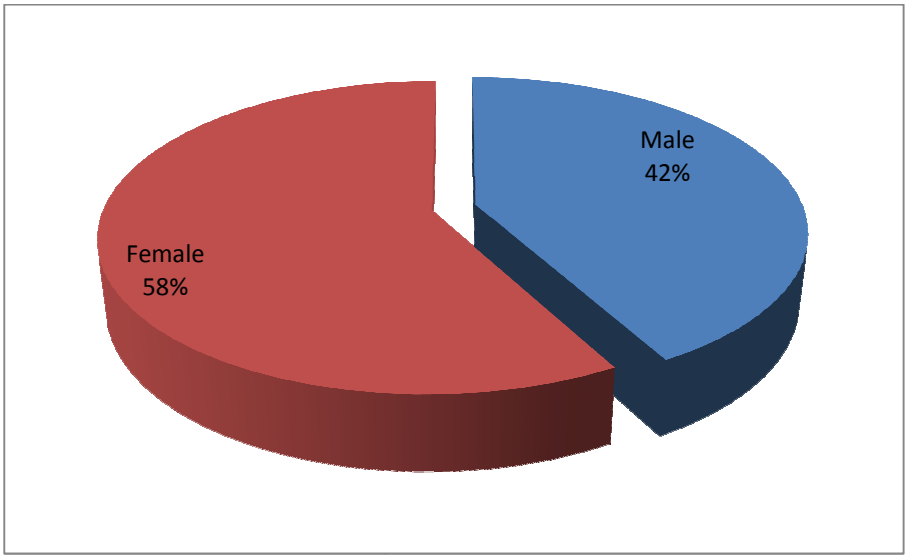

AGE DISTRIBUTION
Percentage of children
40
35
30
25
20
15
10
5
0
1month-<1year
1-<4years
4-<8years
8-12years
Age

TABLE 2

SEX DISTRIBUTION WITH OUTCOME

Sex	Improved		Death		Total
	Number	Percent	Number	Percent	
Male	23	54.7	19	45.3	42
Female	34	58.6	24	41.4	58
Total	57	57	43	43	100
[OR (95% CI) = 0.854(0.383-1.904)]					



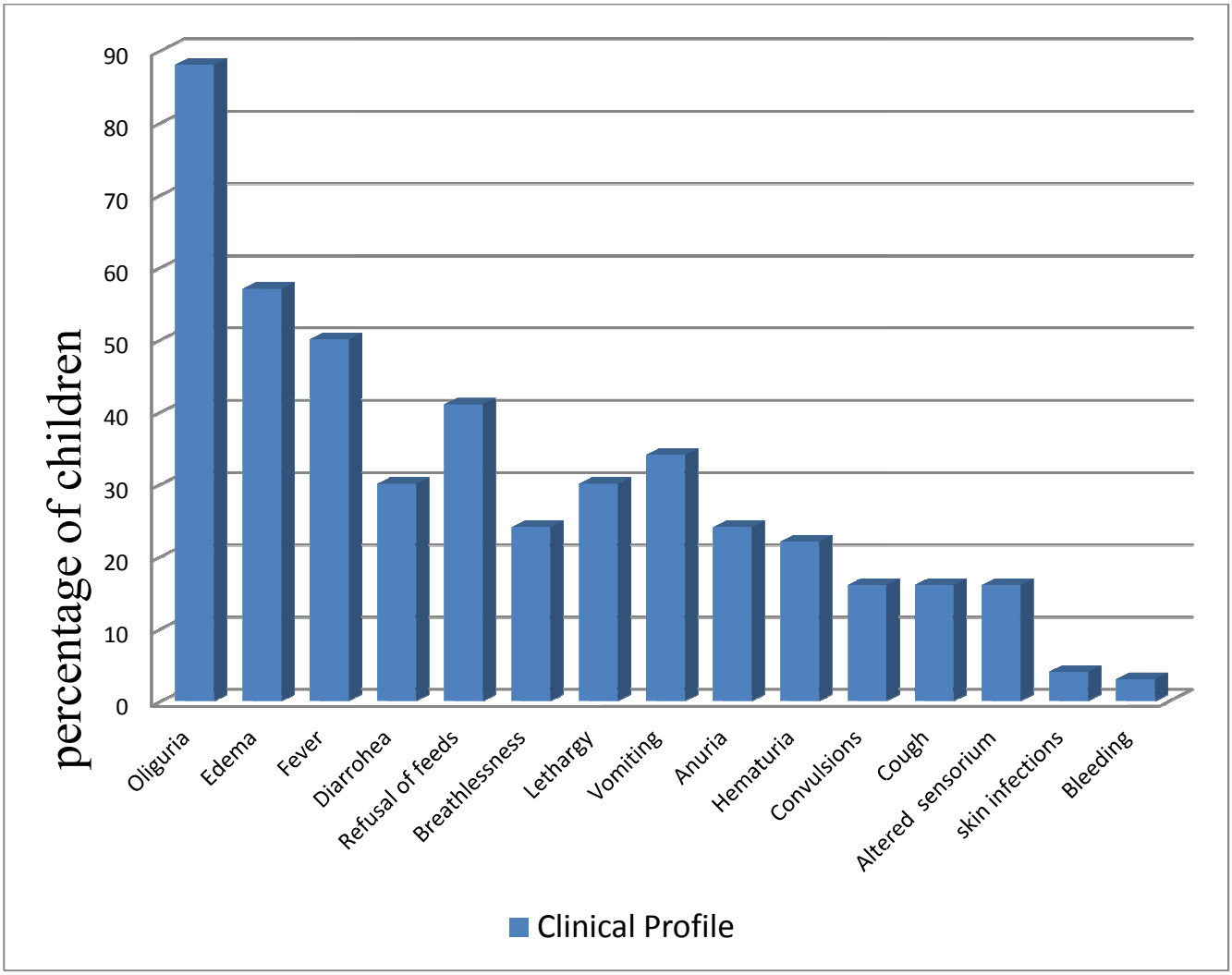
P : Value : not significant. Of 100 children in our study, there were 42 males as against 58 female children,with male:female ratio 1:1.4 and more number of male children have expired. There was no statistical association between sex and the outcome.

TABLE 3

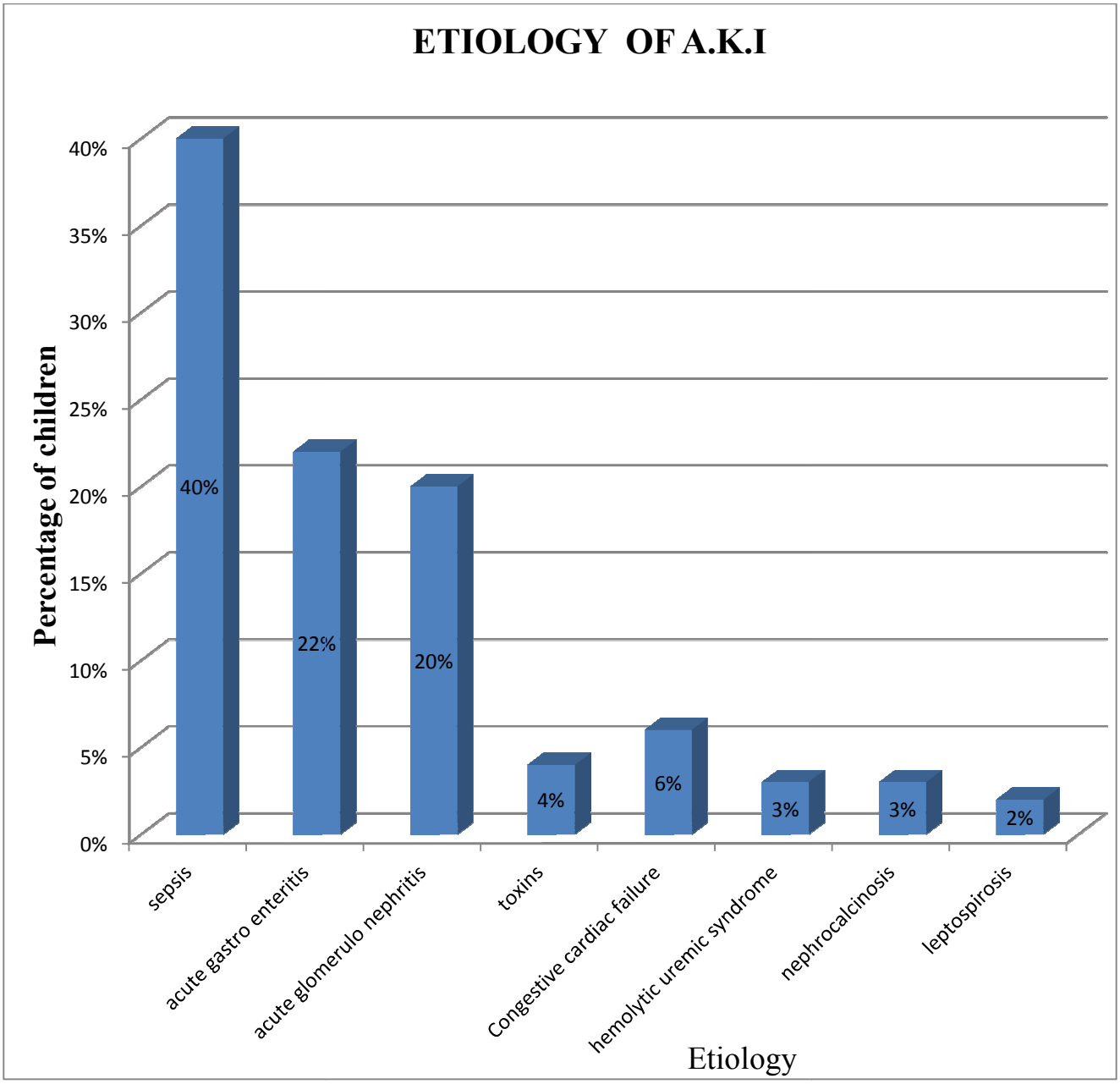
CLINICAL FEATURES ON ADMISSION

Clinical features	No.of children	Percentage	P Value	OR (95% CI)
Oliguria	88	88	0.602	0.725(0.217-2.428)
Edema	57	57	0.842	1.085(0.487-2.416)
Fever	50	50	0.313	0.664(0.299-1.472)
Diarrohea	30	30	0.216	0.701(0.701-4.723)
Refusal of feeds	41	41	0.574	1.259(0.564-2.815)
Breathlessness	24	24	0.880	0.931(0.367-2.358)
Lethargy	30	30	0.692	0.839(0.352-2.007)
Vomiting	34	34	0.062	2.217(0.955-5.147)
Anuria	24	24	0.027	2.857(1.106-7.739)
Hematuria	22	22	0.084	2.311(0.881-6.064)
Convulsions	16	16	0.537	1.400(0.479-4.088)
Cough	16	16	0.300	0.550(0.176-1.722)
Altered sensorium	16	16	0.086	2.576(0.855-7.760)
skin infections	4	4	0.458	0.429(0.043-4.270)
Bleeding	3	3	0.043	2.425(1.912-3.075)

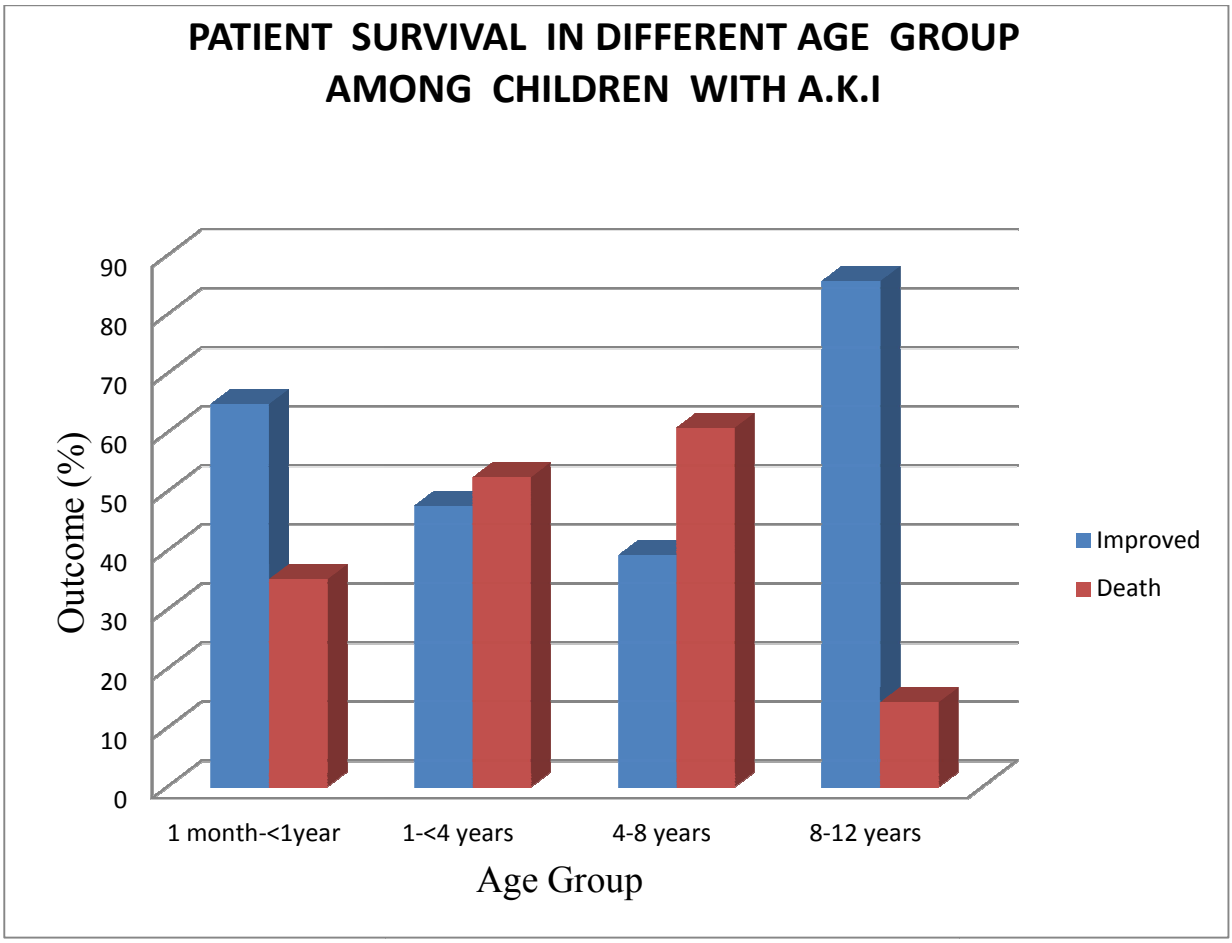
CLINICAL PROFILE



Children with A.K.I presented with various signs and symptoms, depending on the underlying causative factor. In this study most children presented with oliguria (88%), edema (57%), fever (50%), diarrohea (30%), breathlessness(24%). In infants major presenting features were refusal of feeds, vomiting , lethargy. Most of the manifestations were not per se due to A.K.I but can be associated complication. Hence a high degree of suspicion is necessary to diagnose A.K.I in children.



The various causes of A.K.I in relation to different age group were analysed. Sepsis constituted the most common cause of A.K.I contributing 40% of cases. Next common causes were acute gastroenteritis(22% of cases),and acute glomerulonephritis (20% of cases);toxin was attributed as a causative factor in 4% cases and leptospirosis in 2% cases.Nephrocalcinosis and haemolytic uremic syndrome accounted for 3%of cases each.



P= 0.019 significant

Children with A.K.I have different survival rate which depends on the age group.Children with age group 1 month to 4 years showed 55.8% of total mortality.Low mortality was noted in the age group of 8-12 years(14.3%mortality).Over all mortality recorded was 43%.Statistical significance was noted in patient survival in different age group among children with A.K.I.

TABLE-6

SEPSIS Vs OUTCOME

SEPSIS	Improved		Death		Total
	Number	Percent	Number	Percent	
Absent	40	66.6	20	33.4	60
Present	17	42.5	23	57.5	40
Total	57		43		100
OR(95% CI)= 2.706 (1.185-6.176)					

P< 0.05:Significant.

Clinical evidence of sepsis was noted in 40 children .23 out of 40 children expired.That is a mortality of 57.5% was noted in those cases presented with sepsis .Presence of sepsis is a poor prognostic indicator in children with A.K.I for that statistical significance was noted.

TABLE-7

SHOCK Vs OUTCOME

	Improved		Death		Total
	Number	Percent	Number	Percent	
Absent	46	64.7	25	35.3	71
Present	11	37.9	18	62.1	29
Total	57		43		100
OR(95% CI)= 3.011 (1.231-7.364)					

P < 0.05:Significant.

Shock at the time of admission or during hospital stay was due to many etiological factors. But presence of underlying shock , adversely affected the outcome, as its presence was statistically significant with outcome. Shock was noted mostly in cases of sepsis, acute watery diarrohea ,acute cns infection etc.

Shock was present in 29 cases and among those cases 18 had expired. That is 62.1% mortality. So, presence of shock adversely affects outcome

TABLE-8

HYPERTENSION Vs OUTCOME

	Improved		Death		Total
	Number	Percent	Number	Percent	
Absent	48	70.5	20	29.5	68
Present	9	28.1	23	71.9	32
Total	57		43		100
OR(95% CI)= 6.133 (2.419-15.554)					

P < 0.05:Significant.

Hypertension was noted in 32 cases .Hypertension was mostly in cases of acute glomerulo nephritis ,responded well to anti hypertensive patients, and hence more mortality rate (71.9%) may also attributed to underlying etiology like sepsis.

TABLE-9

DEHYDRATION Vs OUTCOME

	Improved		Death		Total
	Number	Percent	Number	Percent	
Absent	47	60.2	31	39.8	78
Present	10	45.4	12	54.6	22
Total	57		43		100
OR(95% CI)= 1.819 (0.701-4.723)					

P value: not significant.

Dehydration was noted in 22 cases. Among those cases 54.6% mortality rate was observed.Presence of dehydration did not alter the outcome as there was no statistical significance in our study.

TABLE-10

HYPOTENSION Vs OUTCOME

	Improved		Death		Total
	Number	Percent	Number	Percent	
Absent	52	62.6	31	37.4	83
Present	5	29.4	12	70.6	17
Total	57		43		100
OR(95% CI)= 4.026 (1.295-12.514)					

$P < 0.05$: Significant.

Hypotension at the time of admission or during hospital stay was noted in 17 patients .Among 17 patients 12 had expired amounting to 70.6% mortality. Hence presence of hypotension adversely affected the outcome . The cases with hypotension were subset population of shock patients with hypotension (Decompensated shock)

TABLE-11

TOXINS Vs OUTCOME

Toxins	Improved		Death		Total
	Number	Percent	Number	Percent	
Absent	55	57.2%	41	42.8%	96
Present	2	50%	2	50%	4
Total	57		43		100
OR(95% CI)= 1.341 (0.181-9.925)					

P value: not significant.

History of toxin ingestions was noted in 4 cases. Among those cases 50% mortality rate was observed.Toxin ingestion does not alter the outcome as there was no statistical significance in our study.

TABLE-12

CONGESTIVE CARDIAC FAILURE Vs OUTCOME

CCF	Improved		Death		Total
	Number	Percent	Number	Percent	
Absent	54	57.4%	40	42.6%	94
Present	3	50%	3	50%	6
Total	57		43		100
OR(95% CI)= 1.35 (0.258-7.041)					

P value: not significant.

Congestive cardiac failure was noted in 6 cases. Among those cases 50% mortality rate was observed.Presence of CCF does not alter the outcome as there was no statistical significance in our study.

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TABLE-13

ANURIA Vs OUTCOME

	Improved		Death		Total
	Number	Percent	Number	Percent	
Absent	48	63.1	28	36.9	76
Present	9	37.5	15	62.5	24
Total	57		43		100
OR(95% CI)= 2.857 (1.106-7.739)					

P value: Significant.

Anuria was present in 24 cases. Among 24 cases 15 had expired which amounted to 62.5% mortality. Presence of anuria , adversely affected the outcome ,as its presence was statistically significant with outcome.

TABLE 14

BLOOD UREA Vs OUTCOME

BLOOD UREA mg/dl	Outcome				
	Improved		Death		Total
	Number	Percent	Number	Percent	
<100	3	16.6	15	83.7	18
100-200	36	60	24	40	60
>200	18	81.8	4	18.2	22
Total	57		43		100

$P < 0.05$:Significant.

Various biochemical investigations were done to document A.K.I and also to look for recovery of the patient. The values were repeated at regular intervals after starting peritoneal dialysis (24 hours,72 hours, 120 hours)to assess the nature of disease process.

60 children presented with blood urea levels in the range of 100 to 200 mgs%.22 children had initial values of more than 200 mgs%. and 18 children with less than 100 mgs%.

TABLE 15

S.CREATININE Vs OUTCOME

S.CREATININE mg/dl	Outcome				
	Improved		Death		Total
	Number	Percent	Number	Percent	
<2	6	27.2	16	72.8	22
2-4	32	57.1	24	42.9	56
>4	19	86.2	3	13.7	22
Total	57		43		100

P <0.05: Significant

56 children had Serum creatinine values in the range of 2-4 mgs%. Higher values of >4 mgs was noted in 22 children and <2 mgs was noted in 22 children. statistical significance was noted .Other co-morbid illness contributed to poor outcome even in children with low creatinine values.

TABLE 16

MULTIPLE LOGISTIC REGRESSION MODELS, OF PREDICTIVE FACTORS OF A.K.I IN CHILDREN

Predictive Factors	Beta (Log odds)	Standard error	Significance	Exponential Beta (Odds)
Anuria	1.496	.693	.031	4.462
Shock	2.629	.864	.002	13.856
Hypertension	3.904	.906	.000	49.593
Bloodurea			.012	
Bloodurea(<100)	3.178	1.081	.003	23.998
Bloodurea(100-200)	1.299	.800	.104	3.666

MODAL SIGNIFICANCE

OBSERVED		Predicted		
		Outcome		
		Improved	Death	Percentage Correct
OUTCOME	Improved	49	8	86.0
	Death	8	35	81.4
	Overall Percentage			84.0

Method, Backward step wise (Conventional)

Risk factors associated with poor outcome were age group, anuria, bleeding, shock, hypotension, hypertension, sepsis, AGN, blood urea serum creatinine values as mentioned earlier. Statistical significance ($P<0.05$) was noted for these factors in univariate analysis. In multiple regression models, predictive factors for poor outcome in acute kidney injury found were anuria, shock, hypotension ,blood urea ($\leq$200 mg/dl) with P value (<0.05) and with 81.4% percentage correctness for poor outcome.

DISCUSSION

In our study, the maximum number of cases of acute kidney injury were under 1 year age group accounting 37% of total children. This is because of increased incidence of sepsis in this age group. Next major group was noted in 4-8 years age group with 28% of total children.

With regard to the sex distribution, the females are predominantly affected when compared to males. This fact of female predominance is consistent with ICH scenario of ARF cases in the year 2008 (male : female ratio 1:2).Whereas the study conducted by Gallego et al., showed that there is no sex predilection among the ARF cases and also contrary to the observation by Arora et al., which showed male predominance .

A.K.I can occur due to number of causes. Significant portion being acute glomerulonephritis, acute tubular necrosis following diarrhoeal dehydration ,posterior urethral valves, Post surgical etc., In our study ,sepsis is the leading cause of A.K.I. It differs from the study conducted by B.V. Singh et al., His study showed that Acute glomerulo nephritis is commonest. Our study showed that next to sepsis, diarrholeal dehydration and A.G.N were common. In study conducted by Arora et al., showed haemolytic uremic syndrome as the commonest cause of A.K.I. Patrick Niaudet et al., also showed that haemolytic uremic syndrome as the commonest cause of A.R.F. which he attributed mostly due to the precipitating factor like diarrhoea and high incidence of H.U.S may

be probably due to poor socio economic conditions and poor sanitary facilities, leading on to diarrhoea . H.U.S is a cause of A.K.I only in less than 5% of cases in our study. This is because of recognition of complication earlier and efficient treatment of acute watery diarrhoea .Besides that delayed referral increases the mortality rate. H.U.S was found to be a bad prognosticator by R.S Trompeter , he gave a mortality of 30% .In our study one child expired out of three children affected with haemolytic uremic syndrome.

In our study overall mortality rate of acute kidney injury in children aged between 1 month to 12 years noted was 43%.In male children mortality rate was 45.3% and in female children it was 41.4%. The mortality rate was similar to the study conducted by **McNeic et al** [68] which showed Overall, sepsis was the major cause of acute renal failure, accounting for 68 episodes (21.4%), followed by hypovolemia, poststreptococcal glomerulonephritis, systemic lupus erythematosus, and infectious diseases. The overall mortality rate was 41.5%.

Studies by James C.M. Chan,P.W.Kandoth et al., B,C.Shah et al., and Gallego et al., stressed the importance of ARF in children less than 1 year of age. The increased mortality in this age group may be probably related to the increased incidence of associated systemic complications, delay in identifying high risk problems and functional and structural immaturity of the infant kidneys .In our study Children with A.K.I have different survival rate which

depends on the age group.Children with age group 1 month to 4 years showed 55.8% of total mortality.Relatively lower incidence of mortality in in less than 1 year age group when compared to above mentioned studies may be attributed to non inclusion newborn babies. Usually Birth asphyxia besides sepsis attributes more number of incidence of mortality in newborn babies.Low mortality was noted in the age group of 8-12 years(14.3%mortality).Over all mortality recorded was 43%.The survival rate is higher in 8-12 years age group .In our study also showed the same result accounting 85.7%.

One of the important symptom of A.K.I is oliguria.But absence of oliguria does not rule out A.K.I. Non oliguric renal failure is well known entity and is seen commonly following septicaemia , PUV and the use of nephrotoxic drugs. In our study among 4 children with non oliguric AKI 2 children died. In all those 4 children sepsis was the underlying etiology.The commonest presentation of A.K.I in our study is oliguria. This fact is being assesssed by P.W.Kandoth et ai.,who also finds oliguria as the commonest presentation in their study. The prognosis is better when the oliguria is very short. This is considered by B.V.Shah et al. The delay in diagnosing AKI increases mortality rate.

One another factor that alters the mortality of A.K.I is associated systemic complications like respiratory , urinary tract infections , peritonitis and central nervous system involvement.

Clinical features seen by us were similar to other published data.Kandoth et al., reported unusually higher number of patients with hypotension and hypokalemia in ARF due to Acute gastro enteritis . In her study, oligoanuria was present in all children, hypertension in 23% , hypotension in 16.6%, neuropsychiatric manifestations in 20% and bleeding in 4.2% in children with ARF . who underwent peritoneal dialysis(PD).Choudry et al ., reported oliguria in all patients, hypertension in 36.6%sensorial disturbance in 25%, anemia in 53.3% and edema in 43.3%children who underwent PD.Jyothi murugan et al., reported 57% had oliguria and 33% anuria . 43% sensorial disturbance,12% hypertension ,7% shock and 16%bleeding disorders in ARF children.

In our series of 100 children with AKI most children presented with oliguria (88%), edema (57%), fever (50%), diarrohea (30%), breathlessness(24%). In infants major presenting features were refusal of feeds, vomiting , lethargy. Most of the manifestations are not per se due to A.K.I but can be associated complications. Hence a high degree of suspicion is necessary to diagnose A.K.I in children.

Chugh et al., reported 44.7% death of patients with ATN , 28.5%Of death of patients with AGN and 25% death in patients with refractory CCF in the nephrology unit needing dialysis. The overall mortality of ARF was 45%in his study of ARF .Choudhry et al., demonstrated 10% mortality in ATN and

22.7% mortality in AGN with overall mortality of 16.7%. Srivastava et al , reported mortality of 23.1%in ATN and death of all four children with AGN needing dialysis with overall mortality of 31.6%.

Choudhry et al, reported children with hypertension ,neurological changes and significant increase in mortality.Blood urea levels more than 300 mg/dl did not alter the prognosis . Kandoth et al, demonstrated much higher mortality in girl children , infants, ARF Due to HUS/ATN ,those with neuropsychiatry manifestation and hypotension. Shah et al.,and Deb et al.

demonstrated that young age and anuria were indicators of poor outcome.In our study also statistical significance was noted for anuria and poor outcome. Jyothimurugan et al.,showed that infancy,female sex, associated neurological symptoms,shock,and hypertension were the factors associated with poor outcome though statistically significant poor prognosis($P<0.5$) was noted only with factors like infancy and in etiology of ATN/HUS alone. In our study risk factors associated with poor outcome were age group (4-8 years), anuria, bleeding, shock, hypotension ,hypertension, sepsis, AGN, blood urea serum creatinine values as mentioned earlier. Statistical significance ($P<0.05$) was noted for these factors in univariate analysis. In multiple regression modes, predictive factors for poor outcome in acute kidney injury found were anuria, shock, hypotension ,blood urea with P value (<0.05) and with 81.4%

percentage correctness for poor outcome. In our study all AKI children with shock /hypotension solely have underlying sepsis.

LIMITATIONS. 1. A minority of group members, both intensivists and nephrologists, felt that a urine output reduction of less than 0.5 ml/kg per hour over the span of six hours was not specific enough to lead confidently to the designation of AKI. It was recognized that the hydration state, use of diuretics, and presence of obstruction could influence the urine volume, hence the need to consider the clinical context. Additionally, accurate measurements of urine output may not be easily available in all cases, particularly in patients in non-intensive care unit settings. Despite these limitations, it was felt that the use of changes in urine offers a sensitive and easily discernible means of identifying patients, but its value as an independent criterion for diagnosis of AKI will need to be validated.

2. No matter how carefully conducted, single – centre studies are inherently limited in terms of sample size and external validity (i.e., generalizability to AKI at other medical centers)

CONCLUSION

1.Acute kidney injury is one of the important causes of morbidity and mortality in children.

2.Female predominance with a male : female ratio 1:1.4 is observed.

3.Sepsis is the commonest cause of acute kidney injury in children. Other common causes of acute kidney injury in children are acute gastro enteritis, acute glomrulonephritis.

4.Overall mortality rate of acute kidney injury in children aged between 1 month to 12 years noted was 43%.In male children mortality rate was 45.3%and in female children it was 41.4%.

5.Non oliguric AKI is usually diagnosed by high index suspicion and by periodic biochemical monitoring.

6.Mortality among non oliguric AKI patients was due to septicaemia.

7. Risk factors associated with poor outcome were, anuria, bleeding, shock, hypotension,hypertension, sepsis, AGN, high blood urea, serum creatinine values.

8.Clinically, predictive factors for poor outcome in acute kidney injury found were anuria, shock, hypotension. Hence extra attention should be given for these cases in future to reduce the mortality.

9. Significant reversible factors of acute kidney injury observed were dehydration , sepsis, shock .

10. Hence,prevention or early diagnosis and treatment of these factors will improve the outcome.

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LIST OF ABBREVIATIONS USED IN THIS STUDY.

PD - Peritoneal dialysis.

ARF - Acute Renal failure.

CRF - Chronic Renal Failure

CCF - Congestive Cardiac failure.

ATN - Acute Tubular Necrosis.

ANS - Acute Nephritic Syndrome

AGN - Acute Glomerulonephritis.

HUS - Hemolytic Uremic syndrome.

CAPD - Continuous Ambulatory Peritoneal dialysis.

CCPD - Continuous Cyclic Peritoneal dialysis.

AWD - Acute Watery Diarrhoea.

ESRD -End Stage Renal Disease.

NEC - Non Enteric Culture.

PROFORMA

PATIENT DATA ENTRY FORM

NAME	
AGE	
SEX	
ADDRESS	
I.P.NO	
WARD	
D.O.A	
DATE OF DISCHARGE/AMA	
DURATION	

PRESENTING FEATURES

	YES	NO
Oliguria		
Hematuria		
Dysuria		
Polydipsia		
Polyuria		

Nocturia		
Thin stream		
Dribbling		
Straining		
Rec.UTI		
Edema		
Abd.distension		
Fever		
Not thriving		
Drug intake		
Diarrhoea		
Vomiting		
Refusal of feeds		
Lethargy		
Convulsions		
Altered sensorium		
Skin infection		
Breathlessness		
Surgery		
Rashes		

Arthritis		
Tetany		
Abd.pain		
Head ache		
Bleeding manifestations		
Cough		

GENERAL EXAMINATION

Altered Sensorium		
Edema		
Jaundice		
Sclerema		
AF		
Respiratory distress		
Hydration status		

VITALS

RR	I	N	D
HR	I	N	D
BP	I	N	D
TEMP	I	N	D
SHOCK	PRESENT		ABSENT

I-INCREASED

N-NORMAL

D-DECREASED

NUTRITIONAL STATUS

NORMAL	
MALNOURISHED	

SYSTEMIC EXAMINATION

RS

CVS

P/A

CVS

INVESTIGATIONS

LEVELS

Blood urea

Sr.Creatinine

Sr.elecrolytes

Sr.calcium,

Phosphorus,

Alk.phosphatase

Sr.Total protein,

Albumin

Globulin

Urine Routine

C/S,

P/Cr

USG KUB,

cranium

X-ray chest

NEC

Fever work up

TREATMENT

IVF

Antibiotic ;Type

Duration

Inotropes ; Type

Duration

PERITONEAL DIALYSIS

Duration

Complications if any

Duration of hospital stay;

OUTCOME

1.FULL RECOVERY

2.MORTALITY

FINAL DIAGNOSIS;

´ôÒ¾ø ÀÊÅõ

ÌÆó¨¾¸ÙìÌ ²üÀÎõ °¢Ú¿£Ã¸ À¡¾¢ôüÌ ¸¡Ã½Á¡É ÀøSÅÚ Á¡üÈ¢Â¨Áì¸ì ÜÊÂ ¸¡Ã½¢¸û ÁüÚõ À¢ýÅ¢¨Ç×¸û ÀüÈ¢ ´Õ ¬ö×.

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ÁÕ.K.þÃ¡Á¾¡Š,M.D.,(PG) Ó¾ý¨Á ¬öÅ¡Ç÷.

§ÁüÀ¡÷¨ÅÂ¡Ç÷

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§ÀÃ¡°¢¡¢Â÷ ÁÕ.À¢ÃÀ¡¦°íÌðÎÅý,M.D.,D.C.H.D.M.

¾¸Åø¾¡û :

¾ü§À¡Ð þó¾¢Â¡ §À¡ýÈ ÅÇ÷óÐ ÅÕõ ¿¡Î¸Ç¢ø °¢Ú¿£Ã¸ À¡¾¢ôÒ ÌÆó¨¾¸¨Ç ¦ÅÌÅ¡¸ À¡¾¢òÐ ÅÕ¸¢ÈÐ. þôÀ¡¾¢À¢É¡ø ²üôÀÎõ þÈôÒ Å¢¸¢¾õ «¾¢¸Á¡¸ þÕó¾¡Öõ, «ÅüÈ¢ø ¦ÀÕõÀ¡Ä¡É¨Å ¾Îì¸ ÜÊÂ¨Å ±ýÀÐ Å¢ÂôÒõ, ÅÕò¾ÓÁÇ¢ôÀ¾¡¸ ¯ûÇÐ. þó¾ ¾Îì¸ÜÊÂ ¸¡Ã½¢¸¨Ç ÓýÉ§Ã «È¢óÐ, ¾ì¸Å¡Ú °¢¸¢î¨° ¸¢¨¼ìÌÁ¡Ú ¦°öÅ¾ý ãÄõ þÈôÒ Å¢¸¢¾ò¨¾ ¸½¢°Á¡¸ Ì¨Èì¸ ÓÊÔõ. °¢Ú¿£Ã¸ À¡¾¢ôÒ ÀüÈ¢Â ÓýÉ÷ §Áü¦¸¡ñ¼ ¬ö×¸û ¦ÀÕõÀ¡Öõ þÃò¾ Íò¾¢¸¡¢ôÒ §¾¨ÅôÀÎõ þÕ¾¢¸ð¼ °¢Ú¿£Ã¸ ¦°ÂÄ¢Æô¨À ¨ÁÂÁ¡¸ ¦¸¡ñ¼¨Å. ¬É¡ø, °¢Ú¿£Ã¸ À¡¾¢ôÒ «À¡Âõ ¯ûÇÅ÷¸¨Ç ÓýÉ§Ã ¸ñ¼È¢óÐ ¾ì¸ ¿¼ÅÊì¨¸¸¨Ç §Áü¦¸¡ûÅÐ «Å°¢Âõ ±ýÀ¾¡ø þó¾ ¬ö× «Å°¢Âõ §Áü¦¸¡ûÇ §ÅñÂ ´ýÈ¡Ìõ.

¬öÅ¢ý §¿¡ì¸õ

1. °¢Ú¿£Ã¸ À¡¾¢ôÒ ²üÀð¼ ÁüÚõ À¡¾¢ôÒ «À¡Âõ ¯ûÇ ÌÆó¨¾¸Ç¢ý Å¢ÅÃí¸û, «¡¢ÌÈ¢¸û Ó¾Ä¡É¨Å ÀüÈ¢ «Ã¡öÅÐ.
2. °¢Ú¿£Ã¸ À¡¾¢ôÀ¢ý ¾Îì¸ ÜÊÂ ÜÊÂ ¸¡Ã½¢¸¨Ç ¬ÃöÅÐ ÁüÚõ À¢ýÅ¢¨Ç×¸¨Ç ¬Ã¡öÅÐ.

¬Ã¡öî°¢ ¿¼ÅÊì¨¸¸û

«ÃÍ ÌÆó¨¾û ¿Ä ÁÕòÐÅÁ¨É ÁüÚõ ¬Ã¡öî°¢ ¿¢ÄÂò¾¢ø ÀøS§ÅÚ Å¡¡Î¸Ç¢ø «ÛÁ¾¢ì¸ôÀÎõ 1 Á¡òõ Ó¾ø 12 ÅÂ¾¢üÌðÀð¼ °¢Ú¿£Ã¸ À¡¾¢ôÒ «À¡Âõ ¯ûÇ / À¡¾¢ì¸ôÀð¼ ÌÆó¨¾¸û þó¾ ¬ö×ì¸¡¸ §¾÷ó¦¾Îì¸ôÀÎ¸¢È¡÷¸û. þ¾ü¸¡É ÌÆó¨¾¸û ÀüÈ¢Â Å¢ÅÃõ. «È¢ÌÈ¢¸û Ó¾Ä¡É¨Å ¦Àü§È¡¡¢¼ò¾¢ø §¸ð¼È¢óÐ, ¦Àü§È¡¡¢ý «ÛÁ¾¢ ¦ÀüÈÀ¢ý þÃò¾ À¡¢§°¡¾¨É ÁüÚõ þ¾Ã À¡¢§°¡¾¨É¸û §Áü¦¸¡ûÇôÀÎõ. «¾ü§¸üôÀ Å¨¸ôÀÎò¾ôÀðÎ ¯¡¢Â °¢¸¢î¨° Ó¨È «Ç¢ì¸ôÀÎõ. ±ó¾ Å¢Çì¸Óõ ¦¸¡Îì¸¡Áø ¿£í¸û ¯í¸û ÌÆó¨¾¨Â ¬öÅ¢Ä¢ÕóÐ ±ó¾ ¸ð¼ò¾¢Öõ Å¢Äì¸¢ì¦¸¡ûÇ ÓÊÔõ.

þÃ¸°¢Â ¸¡ôÒ

¾¡É¡¸ ÓýÅóÐ ¬öÅ¢ø ÀíÌ¦Àüõ ÌÆó¨¾¸¨Ç ÀüÈ¢Â Å¢ÅÃí¸û «¨ÉòÐõ þÃ¸°¢ÂÁ¡¸ °ð¼ôÀÊ À¡Ð¸¡ì¸ôÀÎõ. þó¾ ¬ö¨Å ¿¼òÐÀÅ÷¸Ùõ ¿£¾¢¦¿È¢ ÌØ ¯ÚôÀ¢É÷¸Ùõ þó¾ ÁÕòÐÅÁ¨É¨Â §°÷ó¾ ÁÕòÐÅ÷¸û ÁüÚõ °Æ¢Â÷¸Ùõ ¯í¸û ÌÆó¨¾¨Â ÀüÈ¢Â Å¢ÅÃí¸¨Ç ¦¾¡¢óÐ¦¸¡ûÇ §ÅñÎÁ¡É¡ø ÌÆó¾Â¢ý À¾¢× ¦°öÂôÀð¼ ÀÊÅí¸û, Á¡¾¢¡¢¸û ÀüÚ, ¦¾¡¼÷ ¬öÅ¢ý §¸¡ôÒ¸û ãÄõ ¦¾¡¢óÐ ¦¸¡ûÅ¡÷¸û.

Ì¨ÈÀ¡Î¸û ÁüÚõ ¿ý¨Á¸û

±ùÅ¢¾ ¾£¨Á¸Ùõ þó¾ ¬öÅ¢É¡ø ²üÀ¼¡Ð. þó¾ ¬öÅ¢ý ÓÊ¨Åì ¦¸¡ñÎ ¯¡¢Â °¢¸¢î¨°Ó¨È ¦°öÂôÀÎõ. Ò¾¢Â °¢¸¢î¨° Ó¨È¸û ÀüÈ¢Â À¡¢§°¡¾¨É ÓÂü°¢¸û ±Ð×õ þó¾ ¬öÅ¢ø §Áü¦¸¡ûÇôÀ¼Á¡ð¼Ð.

¦°Ä×¸û Áüêõ ¿‰¼ ®Î

ÀÄ À¡¢§°¡¾¨É¸û þÄÅ°Á¡¸ þõÁÕòÐÅÁ¨ÉÂ¢ø ¦°öÂôÀÎ¸¢ÈÐ. ¯¡¢Â °¢¸¢î¨° Ó¨ÈÔõ þÄÅ°Á¡¸ «Ç¢ì¸ôÀÎ¸¢ÈÐ.

þó¾ ¬ö× ÌÈ¢òÐ °ó§¾¸í¸û ±ÐÅ¡¸ þÕó¾¡Öõ «Ð ÀüÈ¢Â °¡¢Â¡É Å¢Çì¸õ ¾ÃôÀÎõ.

¾¸Åø «Ç£ì¸ôÀð¼ ´ôÒ¾ø ÀÊÅõ

±ÉÐ ÌÆó¨¾ìÌ °¢Ú¿£Ã¸ À¡¾¢ôÒ ²üÀðÎûÇÐ ±ýÀ¨¾ ÁÕòÐÅ¡¢ý ãÄõ ¦¾¡¢óÐ¦¸¡ñ§¼ý. þÃò¾À¡¢§°¡¾¨É ¦°öÐ ¦¸¡ûÇ §ÅñÊÂÐ «Å°¢Âõ ±ýÀÐ ÁÕòÐÅ¡¢ý ãÄõ «È¢óÐ ¦¸¡ñ§¼ý.

þó¾ ¬ö× ÀüÈ£ ±ÉìÌ Å¢Çì¸Á¡¸ ±ÉÐ ¾¡ö¦Á¡Æ¢Â¢ø (¾Á¢ú) ¦°¡øÄÀ¡ð¼Ð. þó¾ ¬öÅ¢ø Àí¦¸ÎòÐì ¦¸¡ûÅ¾¡ø ±ÉÐ ÌÆó¨¾ìÌ ²üÀ¼ì ÜÊÂ «À¡Âí¸û ÁüÚõ ¿ý¨Á¸û ÀüÈ¢ ±ÉìÌ Å¢Çì¸ôÀð¼Ð¼ý °õÁ¾¢ì§Èý. §¸ûÅ¢¸û §¸ðÀ¾üìÌ ±ÉìÌ Å¡öôÒ «Ç¢ì¸ôÀð¼Ð.

þó¾ ¬öÅ¢Ä¢ÕóÐ ¸¢¨¼ìÌõ ÓÊ×¸¨Ç ÀÂýÀÎòÐÀÅ¨Ã ¸ðÎôÀÎò¾¡ÁÄ¢Õì¸ ¿¡ý °õÁ¾¢ì¸¢§Èý.

ÌÆó¨¾Â¢ý ¦ÀÂ÷ :

ÌÆó¾Â¢ý ¦Àü§È¡÷ /

¸ñ¸¡½¢ôÀÇ÷ ¦ÀÂ÷ :

ÌÆó¨¾Â¢ý ¦Àü§È¡÷ /

¸ñ¸¡½¢ôÀÇ÷ ¨¸¦Â¡ôÀõ :

§¾¾¢ :

°ð°¢Â¢ý ¦ÀÂ÷ :

°ð°¢Â¢ý ¨¸¦Â¡ôÀõ :

§¾¾¢ :

¬öÅ¡Ç÷ / ¬öÅ¡Ç÷ ÁÕòÐÅ÷ ¦ÀÂ÷ :

¬öÅ¡Ç÷ / ¬öÅ¡Ç÷ ÁÕòÐÅ÷

¨¸¦Â¡ôÀõ :

§¾¾¢ :

CONSENT FORM

A STUDY CLINICAL PROFILE ,REVERSIBLE RISK FACTORS AND OUTCOME IN ACUTE KIDNEY INJURY;

INVESTIGATOR'S NAME; Dr.K.Ramdass M.D Ist year

(To be read to caretakers in the presence of witness)

Your child is suffering from acute kidney injury.It has diverse etiology .to know the clinical profile and reversible risk factors the questionnaire is formed.By analyzing disease , appropriate mode of treatment can be carried out with available investigation modalities .

HOW IS THE STUDY BEING DONE ?

Thiis study will be conducted in the children 1month to 12 years of age admitted in nephrology ,PICU,Medical wards in ICH&HC.The doctor will ask you questions &examine the child to make sure that it is safe for him/her to enter the trial. If your child is suffering from acute kidney injury you(Parent/care taker) will have to answer the questions and answers will be entered in the proforma..

Selected children will be subjected for some blood investigations according to maneuver protocol which ll be done under strict aseptic precautions only.

CAN I REFUSE TO JOIN THE STUDY?

You may refuse to participate or withdraw from this study at any time.

IS THERE A BENEFIT OR HARM?

Appropriate treatment can be started based on this study details

It may be possible to reduce the severity of the disease by appropriate treatment

CONFIDENTIALITY;

The data collected from the study will be used for the purpose of the study only.The results for the study to be published in medical journals.personal informations of the children participatory in the study ll be kept confidential , Ther will not be any disclosure about your child's information without your attention

COST & COMPENSATION;

Investigations warranted will be done at free of cost in our hospital &by the investigator .Any furher questions that may arise at any point of time will be willingly clarified.

SUBJECT RIGHTS

I Understand that if I wish further information regarding my child's rights as a research subject ,I may contact the hospital where the study is taking place..

Section II

INFORMED CONSENT FORM

- I understand that my child is suffering from Acute Kidney Injury and it is essential to answer the questionnaire and to collect Blood Sample for Biochemical examination from my child as told by the treating physician.
- I confirm that have been told about this study in my mother tongue (Tamil) and have had the opportunity to ask questions. I confirm that I have told about the risks and potential benefits for my child's participation in the study. I agree to give my consent for the participation of my child in this study.
- I understand that my consent for my child's participation in the study is voluntary and I can withdraw my child from participation in the study at any time, without giving any reason, without my child's medical care being affected.
- I agree not to restrict the use of any data or results that arise from this study.

Name of the Child : _____

Name of the Parent / Guardian : _____

Signature of the Parent / Guardian : _____

Date : _____

Thumb Print of Illiterate Parent / Guardian

Name of the Witness : _____

Signature of the witness : _____

Date : _____

Name of the investigation / Medical Officer : _____

Signature of the Investigator/Medical Officer : _____

Date : _____

ABSTRACT:

Background: Acute kidney injury Acute Kidney Injury (AKI), the new term heralds a paradigm shift for our conceptualization of the syndrome which is previously called "acute renal failure." AKI retains one word, substitutes a synonym for another, but supplants "failure" with "injury." This substitution may very well redefine the epidemiology of the syndrome.Acute kidney injury is defined as an abrupt (within 48 hours) reduction in kidney function currently defined as an absolute increase in serum creatinine of more than or equal to 0.3 mg/dl (≥ 26.4 μmol/l), a percentage increase in serum creatinine of more than or equal to 50% (1.5-fold from baseline), or a reduction in urine output (documented oliguria of less than 0.5 ml/kg per hour for more than six hours). 50%mortality rate seems to have remain unchanged despite technical process .Among a number of children who die with terminal renal failure there are many with preventable causes .Early recognition of the cause and its prevention would reduce the mortality.**Objective:** This study aims at finding out the clinical profile and reversible(modifiable) risk factors associated with poor outcome in children with acute kidney injury seen in this part of the country in a referral pediatric centre at Chennai, Tamilnadu **Subjects and methods:** This study was done Institute of child health&hospital for children, chennai 600008 during the period Dec.2009 to Oct 2011Study Design is Descriptive

Study/Nested case control study. Children 1 month-12years presenting with clinical and biochemical evidence of acute kidney injury are included in the study.Those children with chronic renal failure or who had prior dialysis therapy, Pre existing renal diseases are excluded.Cases are all subjects with poor OutcomeControls are all subjects with recovery. All eligible children with defined criteria are recruited in to the study after obtaining parental consent. Using a standard data extraction form, information is abstracted in detail.After complete evaluation of the patient with a detailed history ,clinical examination and necessary investigation ,the cases were followed with standard line of treatment and the outcome and various factors affecting the final outcome were analysed.Proportions and mean with standard deviation of outcome measures were arrived at as applicable. Odds ratio with 95% confidence interval were calculated and to adjust for confounding factors, adjusted odds ratio with 95% confidence interval will be arrived at by Multivariate analysis.Chi-square test, logistic regression are applied. $P<0.05$ was considered as statistically significant. **Results:** Acute kidney injury is one of the important causes of morbidity and mortality in children.Female predominance with a male : female ratio 1:1.4 is observed. 3.Sepsis is the commonest cause of acute kidney injury in children. Other common causes of acute kidney injury in children are acute gastro enteritis, acute glomrulonephritis. Overall mortality rate of acute kidney injury in children aged between 1 month to 12 years noted was 43%.In male children mortality rate was 45.3%and in female children it was 41.4%.Non

oliguric AKI is usually diagnosed by high index suspicion and by periodic biochemical monitoring.Mortality among non oliguric AKI patients was due to septicaemia. Risk factors associated with poor outcome were, anuria, bleeding, shock, hypotension,hypertension, sepsis, AGN, high blood urea, serum creatinine values.**Conclusion:** Significant reversible factors of acute kidney injury observed were dehydration , sepsis, shock . Hence,prevention or early diagnosis and treatment of these factors will improve the outcome.**Key words:** Acute kidney injury, children,reversible risk factors, outcome.