THE TAMILNADU Dr. M.G.R. MEDICAL UNIVERSITY
CHENNAI – TAMILNADU.

DISSERTATION
ON

AETIOLOGICAL PROFILE AND OUTCOME IN ACUTE RENAL FAILURE

SUBMITTED FOR M.D. DEGREE EXAMINATION
BRANCH I
(GENERAL MEDICINE)

EXAMINATION
IN
September – 2006

THANJAVUR MEDICAL COLLEGE
THANJAVUR

CERTIFICATE

This is to certify that this dissertation entitled **"AETIOLOGICAL PROFILE AND OUTCOME IN ACUTE RENAL FAILURE"** is the bonafide record work done by **Dr. K. GUNASEKARAN**, submitted as partial fulfillment for the requirements of M.D. Degree Examinations, Branch I (General Medicine) to be held in September 2006.

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ACKNOWLEDGEMENT

I am extremely grateful to the **DEAN** and Professor of Therapeutics **Dr. S. BALAKRISHNAN, M.D.,** for granting me permission to do this dissertation in Thanjavur Medical College Hospital, Thanjavur. I am also thankful to our former DEAN **Dr. K. KALAISELVI, M.D**., for the same.

I am very grateful to our Professor and Head of the Department of Medicine **Prof. Dr. K. GANDHI, M.D**., for his valuable suggestion in preparing this dissertation.

I express my profound thanks to **Dr. V. BASKARAN, M.D.,** my unit chief, for his able guidance, constant encouragement and valuable discussion throughout the study.

I am deeply indebted to **Prof. Dr. N. MOHANDAS, M.D., D.M**., (Nephrology) Head of the Department of Nephrology who stood as the backbone of this dissertation in each and every step.

I consider it as a privilege to thank my former unit chiefs Prof. **Dr. DHANASEELAN JAYAKUMAR, M.D.,** and Prof**. Dr. N. JEEVA, M.D**., for initiating me to do this dissertation and encouragement.

I express my heartful gratitude to Dr. **C. PARANTHAGAN, M.D., Dr. BALASANJEEVI, M.D., and Dr. C. GANESAN, M.D.,** Assistant Professors of our unit for their guidance and valuable suggestions throughout the study.

I am thankful to Prof. **Dr. R. CHITRA, M.D**., Head of the Department of Biochemistry and **Dr. M. P. SARAVANAN, M.D.,** Reader in Biochemistry, who were kind enough of to help me in doing Biochemical study of ARF.

I am thankful to Prof. **Dr. VIJAYALAKSHMI, M.D.,** Head of Department of Radiology**, Dr. SRINIVASAN, M.D.,** Additional Professor of Radiology for the invaluable help in doing USG Abdomen study to find out the renal status.

I dedicate my humble work to all the poor patients who inspite of their sufferings, co-operated with me and made this venture a great success.

CONTENTS

INTRODUCTION

Acute renal failure (ARF) is a common clinical syndrome with a broad aetiological profile. It complicates about 5 per cent of hospital admissions and 30 per cent of admissions to intensive care units (ICU). It is associated with major morbidity and significant mortality due to the severity of the causative illness.

The true incidence of ARF is not easily discerned from published reports because of variation in methods of case ascertainment, definitions of ARF, and catchment populations[2]. Most surveys of hospital populations cannot estimate true population incidence because there is a category called unselected hospital populations which include many patients never referred for nephrological opinion. Hospital-based studies of ARF reflect selection bias as these populations are defined by the referral patterns to the site of care. (e.g. district general hospital, tertiary referral centre, general ICU, cardiothoracic ICU, etc.).

Likewise the aetiology of Acute renal failure varies from one geographic region of the world to another. While ARF secondary to diarrhoeal diseases, toxins, septic abortions, and other infections and environmental conditions are the common causes in the tropical countries, ARF following trauma due to high-speed traffic and industrial accidents, complex cardiovascular surgery, nephrotoxic drugs and chemicals, and cardiogenic shock are more prevalent in the industrialized world.[2,4]

In this perspective, I have endeavoured to study the various causes of ARF, in our hospital (Thanjavur Medical College Hospital) during a specified period, and to find out the incidence of Pre-renal, Renal and Post-renal causes by using renal failure indices and to analyse outcome of ARF pertaining to the aetiology.

AIM OF THE STUDY

1. To find out the various causes of ARF in patients admitted in Thanjavur medical college hospital.

2. To find out the incidence of prerenal, renal and post renal types of acute renal failure.

3. To determine the prognostic factors in ARF.

4. To find out the outcome of ARF.

5. To analyse the utilities of dialysis (both peritoneal and hemodialysis) in ARF.

REVIEW OF LITERATURE

HISTORICAL REVIEW

Acute renal failure of course must have followed on many major traumas and tragedies throughout the history, but had not been noted until the 20th century. Rainford's references to the description of acute renal failure in the survivors of crush injuries during the Messina Earth Quake of 1908 is, therefore, fascinating. Colmer had described the renal failure in association with crushed limbs following the Messina Earth Quake,[12] Frankenthal described a similar association in 1916 and subsequently reported a series of 30 patients with muscle necrosis and enlarged kidneys.[13]

Tructa's work on the injured during the Spanish civilwar led to a great advance in the understanding and recognition of acute renal failure. For most of us the history begins with the descriptions of acute renal failure by Bywaters and his colleagues during the London blitz in the early 1940's. His studies on patients injured by fallen masonry during the blitzon London showed acute tubular necrosis in association with muscle necrosis, confirming previous important but little known studies.[14]

DEFINITION

Acute renal failure (ARF) is a syndrome characterized by a rapid (hours to weeks) decline in glomerular filtration rate (GFR) and retention of nitrogenous waste products such as blood urea nitrogen (BUN) and creatinine. ARF is usually asymptomatic and diagnosed when routine biochemical screening of hospitalized patients reveals a recent increase in the concentrations of BUN and serum creatinine. Oliguria (urine output <400 mL/day) is a frequent (approximately 50%) but not invariable clinical feature.[27] The kidney is remarkable among organs of the body in its ability to recover from almost complete loss of function, and most ARF is reversible, albeit with subclinical residual defects in tubule and glomerular function

.

CLASSIFICATION

ARF for purposes of diagnosis and management are conveniently divided into three categories: 1) Diseases characterized by renal hypoperfusion in which the integrity of renal parenchymal tissue is preserved (prerenal azotemia, prerenal ARF:55% to 60%)[3];

(2) Disease involving renal parenchymal tissue (intrarenal azotemia, intrinsic renal ARF; 35% to 40%); and

(3) diseases associated with acute obstruction of the urinary tract (postrenal azotemia, postrenal ARF ; <5%). Most acute intrinsic renal azotemia is caused by ischemia or nephrotoxins and is classically associated with acute tubule necrosis (ATN). Therefore, the terms ischemic ATN or nephrotoxic ATN are commonly used in clinical practice to denote, respectively, ischemic or nephrotoxic ARF.

ETIOLOGY OF ACUTE RENAL FAILURE

Prerenal Azotemia

Prerenal azotemia is the most common cause of ARF and is an appropriate physiologic response to renal hypoperfusion. By definition, the integrity of renal parenchymal tissue is maintained and GFR is corrected rapidly with restoration of renal perfusion and glomerular ultrafiltration pressure.

Severe renal hypoperfusion may cause ischemic ATN. Thus, prerenal azotemia and ischemic ATN are part of a spectrum of manifestations of renal hypoperfusion. Indeed, clinical and biochemical features of prerenal ARF and ischemic ATN may coexist in some patients in a condition known as the **"intermediate syndrome"**[1].

S. No.	MAJOR CAUSES OF PRERENAL AZOTEMIA
1.	**Intravascular Volume Depletion** Hemorrhage: Traumatic, Surgical, gastrointestinal postpartum Gastrointestinal losses: Vomiting, nasogastric suction, diarrhea Renal losses: drug – induced or osmotic diuresis, diabetes insipidus, adrenal insufficiency. Skin and mucous membrane losses: burns, Hypertherima, other causes of increased insensible losses "Third –Space " losses : Pancreatitis, crush syndrome, hypoalbuminemia
2.	**Decreased Cardiac Output:** Diseases of myocardium valves pericardium, or conducting system pulmonary hypertension, pulmonary embolism , positive-pressure mechanical ventilation. Systemic vasodilatation Drugs: antihypertensives, afterload reduction, anesthetics, drug overdoses Sepsis, liver failure anaphylaxis.
3.	**Renal Vasconstriction** Norepinephrine, ergotamine, liver disease, sepsis, hyercalcemia
4.	**Phamacologic Agents That Acutely Impair Autoregulation and Glomerular Filtration Rate in Specific Settings** Angiotensin-converting enzyme inhibitors in renal artery stenosis or severe renal hypoperfusion. Inhibition of prostaglandin synthesis by nonsteroidal anti-inflammatory drugs during renal hypoperfusion.

Prerenal azotemia can complicate any disease characterized by hypovolemia, low cardiac output, systemic vasodilatation, or intrarenal vasoconstriction.Compensatory renal responses are overwhelmed during states of moderate to severe hypoperfusion, and ARF ensues.[28] Autoregulatory dilatation of afferent arterioles is maximal at a mean systemic arterial blood pressure of about 80 mm Hg, and hypotension below this level is associated with a precipitous decline in glomerular ultrafiltration pressure and GFR.

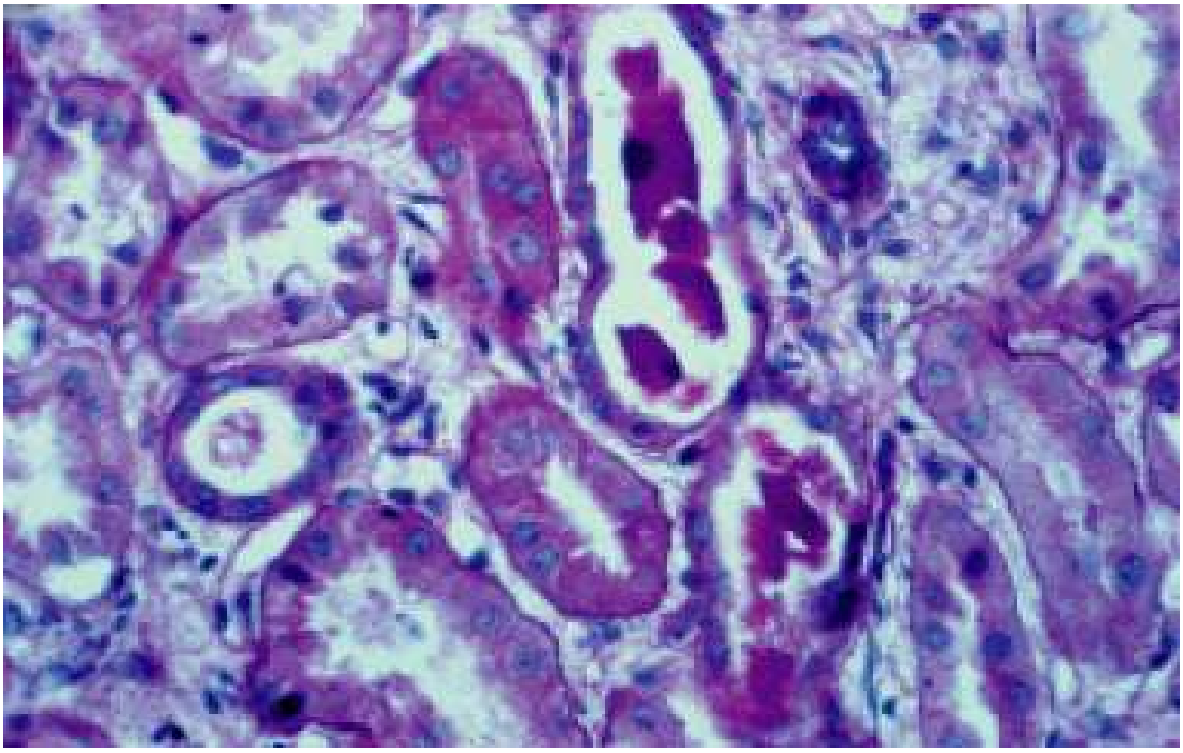
Intrinsic Renal Azotemia:

From a clinicopathologic viewpoint, it is helpful to categorize the causes of acute intrinsic renal azotemia as follows: 1) disease involving large renal vessels, 2) disease of the renal microvasculature and glomeruli, 3) ischemic and nephrotoxic ATN, and 4) other acute processes involving the tubulointerstitium Ischemic ATN and toxic ATN, account for about 90% of acute intrinsic renal azotemia.[29]

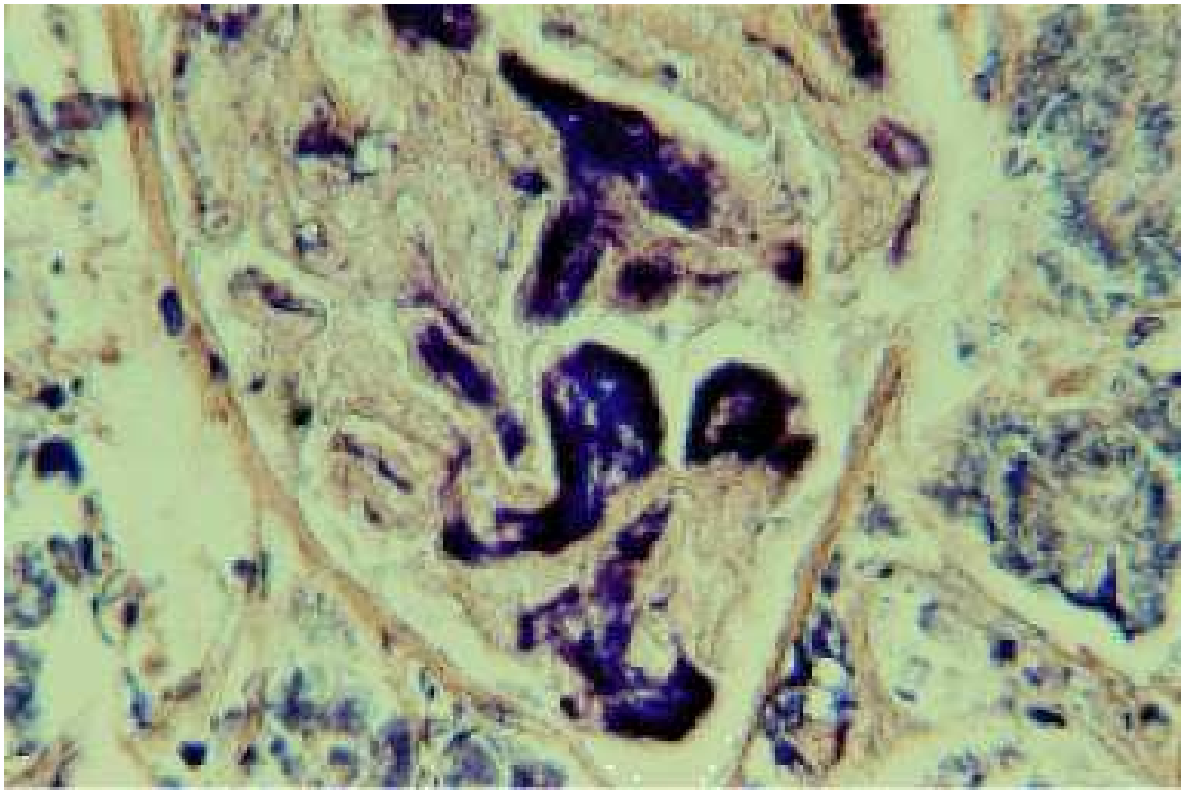
S. No.	MAJOR CAUSES OF ACUTE INTRINSIC RENAL AZOTEMIA
1.	**Diseases Involving Large Renal Vessels** Renal arteries: thrombosis, atherembolism , dissection, vasculitis (e.g., Takayasu)
2.	**Diseases of Glomeruli and the Renal Microvasculature** Inflammatory: acute or rapidly progressive glomerulonephritis, vasculitis allograft rejection, radiation Vasospastic: Malignant hypercalcemia, drugs radiocontrast agents Hematologic Hemolytic-uremic syndrome or thrombotic thrombocytopenic purpura, disseminated intravascular coagulation, hyperviscosity syndromes.
3.	**Diseases Characterized by Prominent Injury to Renal Tubules Often with Acute Tubule Necrosis** Ischemia caused by renal hypoperfusion Exogenous toxins (e.g., antibiotics, anticancer agents, radiocontrast agents, poisons) Endogenous toxins (e.g., myoglobin, hemoglobin, myeloma light chains, uric acid, tumour lysis)
4.	**Acute Diseases of the Tubulointerstitium** Allergic interstitial nephritis (e.g ., antibiotics, nonsteroidal anti-inflammatory) Infections (Viral, bacterial, fungal) Infiltration (e.g lymphoma, leukemia, sarcoid) Some Exogenous Nephrotoxins

Acute Tubule Necrosis:

Although the pathologic term ATN and the clinical term ARF are often used interchangeably when referring to ischemic and nephrotoxic renal injury evidence of frank necrosis of renal tubules is spares or absent in most cases. **Ischemic ATN and prerenal azotemia are part of a spectrum of manifestations of renal hypoperfusion**[1,2] – prerenal azotemia being a response to mild or moderate hypoperfusion and ischemic ATN being the result of more severe or prolonged hypoperfusion, often coexistent with other renal insults. It is notable that extracellular fluid losses or transient renal hypoperfusion (e.g., cardiac arrest, aortic cross clamping) generally do not cause ATN in the absence of either preexisting renal impairment or the coincidence of another nephrotoxic insult (e.g., vasoactive drugs, sepsis, rhabdomyolysis). Prerenal azotemia with injury to renal parenchyma dose not resolve immediately on restoration of renal hypoperfusion and may result in bilateral renal cortical necrosis and irreversible renal failure. Ischemic ATN is observed most frequently in patients who have major surgery trauma, severe hypovolemia, overwhelming sepsis, or burns. ATN complicating trauma is frequently multifactorial in origin and results from the combined effects of hypovolemia and myoglobin or other toxins released by damaged tissue. ATN occurs in 20% to 40% of patients who suffer burns involving more than 15% of their body surface area;



Renal histology reveals acute tubular necrosis.



Renal biopsy - acute diffuse cortical necrosis following snake bite. Single glomerulus - structureless. and capillaries contain thrombi.

It is frequently multifactorial and is caused by the combined effects of hypovolemia rhabdomyolysis, sepsis, and nephrotoxic antibiotics.[30] Sepsis induces renal hypoperfusion by provoking a combination of systemic vasodilatation and intrarenal vasoconstriction. In addition endotoxin sensitizes renal tissue to the deleterious effects of ischemia.[31] Nephrotoxic ATN complicates the administration of many structurally diverse pharmacologic agents and poisons.[32]

Some Exogenous Nephrotoxins That Are Common Causes of Acute Intrinsic Renal Azotemia With Acute Tubule Necrosis.

Antibiotics Acyclovir Cidofovir Indinavir Foscarnet Pentamidine Aminoglycosides Amphotericin B	Chemotherapeutic agents Cisplatin Ifosfamide
Organic solvents Ethylene glcol Toluene	Anti-inflammatory and immunosuppressive agents NSAIDs (including COX-2 inhibitors) Cyclosporin / tacrolimus Intravenous immune globulin Radiocontrast agents Bacterial toxins
Poisons Paraquat Snake bites	

In addition, some endogenous compounds provoke ARF when they are present in the circulation at high concentrations. Myoglobin, hemoglobin,uric acid, and myeloma light chains are the endogenous toxins that are most commonly associated with ATN. Renal dysfunction complicates approximately 30% of case of rhabdomyolysis.

Intratubular obstruction has been implicated as a central event in the pathophysiology of ATN induced by some other endogenous (e.g., myeloma light chains, uric acid) and exogenous (ethylene glycol) nephrotoxins. Casts composed of filtered immunoglobulin light chains and other urinary proteins such as Tamm-Horsfall protein, induce ARF in patients with multiple myeloma (myeloma-cast nephropathy).

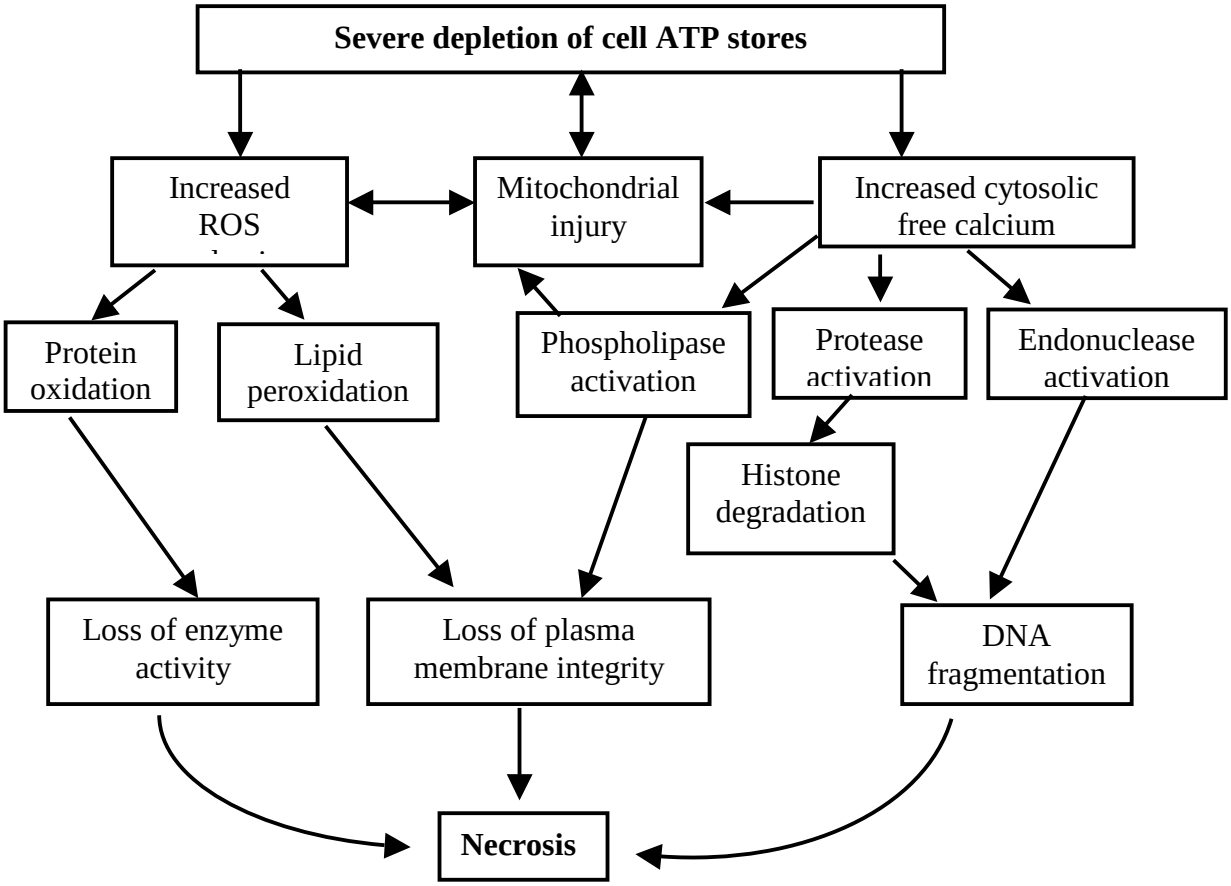
Postrenal Azotemia

Urinary tract obstruction accounts for fewer than 5% of cases of ARF. Because one kidney has sufficient clearance capacity to excrete the nitrogenous waste products generated daily, ARF resulting from obstruction requires either obstruction of urine flow between the external urethral meatus and the bladder neck, bilateral ureteric obstruction, or unilateral ureteric obstruction in a patient with only one functioning kidney or with underlying chronic insufficiency.

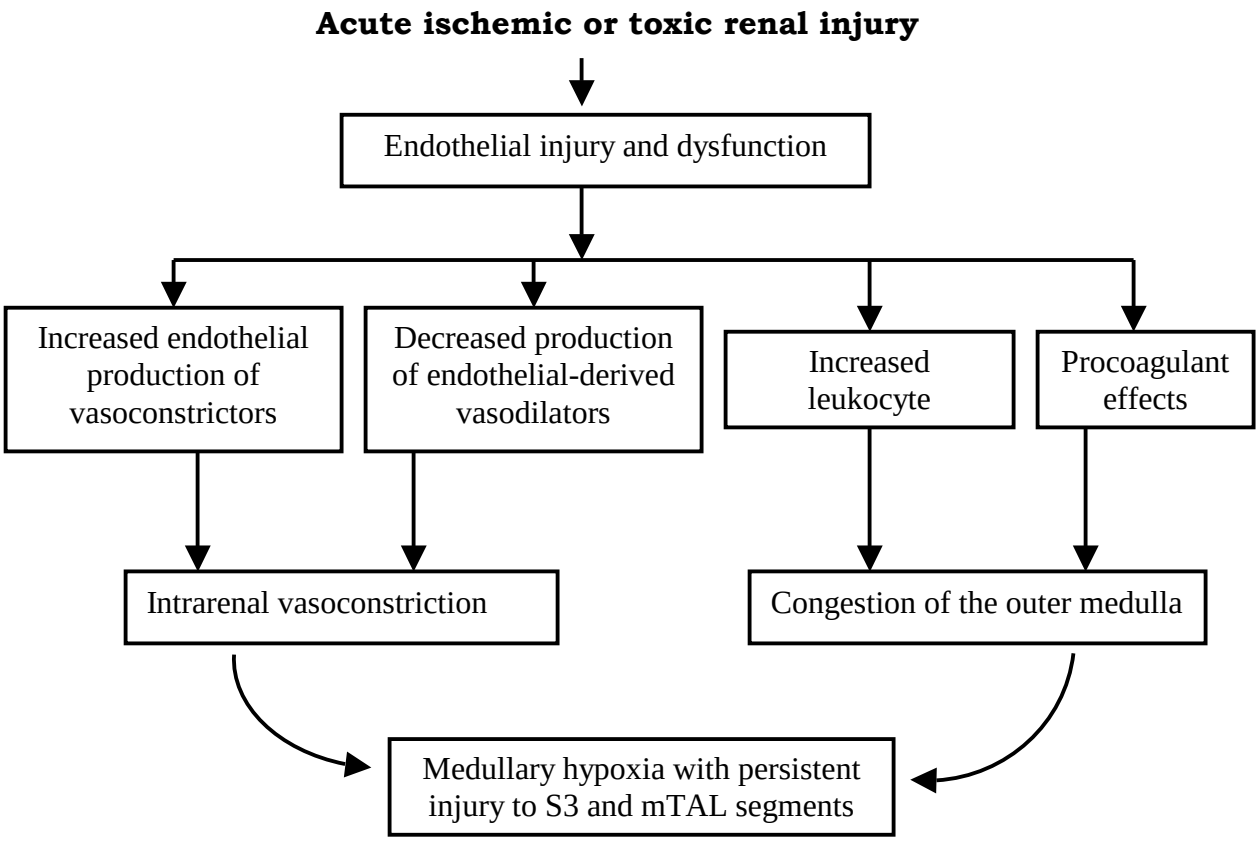
Pathophysiology of Acute Tubule Necrosis: An Overview

Three major factors contribute to the profound reduction in GFR that characterizes ATN: tubule injury, hemodynamic abnormalities, and intrarenal inflammation. Tubule injury leads to renal insufficiently by causing “back-leakage “of glomerular filtrate and intratubule obstruction. Intrarenal vasoconstriction can also directly impair GFR by reducing intraglomerular capillary hydrostatic pressure and plasma flow[33,35].

Biochemical Pathways Leading to Necrosis



Potential Causes of Tubule Cell Apoptosis in Acute Renal Failure[36]
Cell injury
Ischemia-reperfusion Oxidant stress Antibiotics (e.g., aminoglycosides, amphotericin B) Chemotherapeutic agents (cisplatin)
Death receptor ligands
Fas Tumor necrosis factor-α Transforming growth factor-β
Default pathway
Deficiency of growth factors Loss of adhension Cell-matrix Cell-cell



Role of caspase-1 and interleukin-18. The main function of caspase-1 is to activate the proinflammatory cytokines pro-IL-1β and pro-IL-18.[38,39] However, the role of caspase-1 in ARF is still somewhat controversial, because some investigators concluded that caspase-1 activation plays a minor role, if any, in the pathogenesis of ischemic renal injury. Additional studies are necessary to determine the importance of caspase-1 activation in ARF.

α-Melanocyte-Stimulating Hormone Ameliorates Acute Renal Failure: α-Melanocyte-Stimulating Hormone (α-MSH) has been shown to have marked protective effects in experimental models of ischemic renal injury even when started as late as 6 hours after the insult.[40]

Two effects of α-MSH appear to contribute to its protective effect in ARF. The peptide stimulates production of the anti-inflammatory cytokine IL-10 and also inhibits cytokine-induced expression of iNOS, which, promotes tubule injury.

Acute Tubule Necrosis in Sepsis: Sepsis is the result of an uncontrolled and ultimately injurious proinflammatory response to infection. Sepsis is the most common cause of ATN in humans. In septic patients who are not hypotensive, the incidence of ARF is about 20% to 25%. Once hypotension intervenes, the incidence of ARF exceeds 50%. Fundamental cause of ATN in sepsis is renal hypoperfusion and ischemic injury to tubule cells. There are many pathophysiologic similarities between ARF caused by renal ischemia and ARF caused by sepsis, including intrarenal vasoconstriction and microvascular injury. the intrarenal vasoconstriction associated with sepsis has been ascribed to the local release of endothelial – derived vasconstrictors, including endothelin, thromboxane A_2, PAF and leukotrienes.

Course of Acute Tubule Necrosis:

The clinical course of ATN can be divided into three phases: the initiation phase, the maintenance phase, and the recovery phase. The initiation phase is the period during which patients are exposed to the ischemia or toxin and parenchymal renal injury is evolving but not yet established.

ATN is potentially preventable during this period, which may last hours to days. The initiation phase is followed by a maintenance phase, during which parenchymal injury is established and GFR stabilizes at a value of 5 to 10 mL/min. Urine output is usually lowest during this period[27,35]. The maintenance phase typically lasts 1 to 2 weeks but may be prolonged for 1 to 12 months before recovery. The recovery phase is the period during which patients recover renal function through repair and regeneration of renal tissue. Its onset is typically heralded by a gradual increase in urine output and a fall in serum creatinine.

EVALUATION OF PATIENTS WITH ARF

The assessment of patients with ARF requires a meticulous history, physical examinations and urinalysis, in-depth review of previous records and recent drug history, and judicious utilization of laboratory tests, renal imaging, and occasionally renal biopsy.

An acute process is easily established if review of laboratory records reveals a recent rise in BUN and serum creatinine levels. If previous measurements are not available, anemia, hyperphosphatemia, hypocalcemia, neuropathy, band keratopathy, and radiologic evidence of renal osteodystrophy or small, scarred kidneys are useful pointers to a chronic process.

However, anemia, hyperhosphatemia, and hypocalcemia may also compliate ARF, particularly if it is prolonged, and renal size can be normal or increased in a variety of chronic renal diseases (e.g., diabetic nephropathy, amyloid, polycystic kidney disease). Once a diagnosis of ARF is established, attention should focus on differentiating between prerenal, intrinsic renal, and postrenal azotemia and on identifying the specific causative disease.

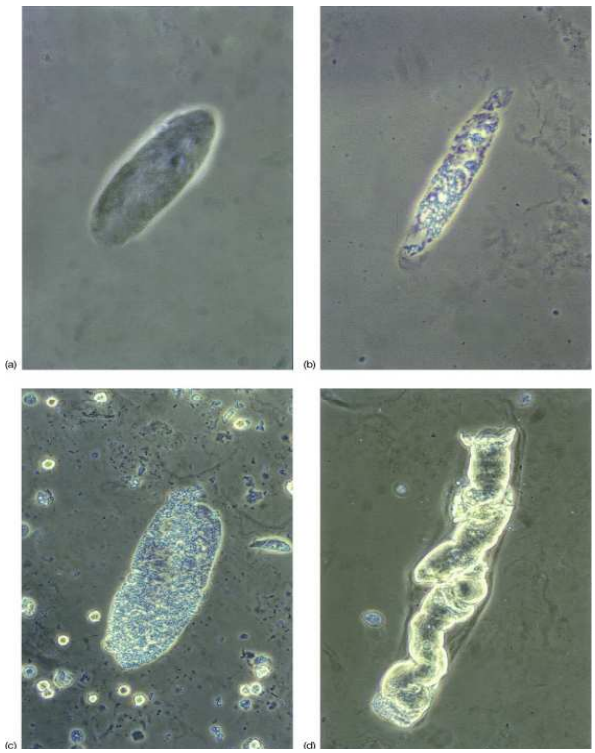
Clinical Assessment

Prerenal azotemia should be suspected if the serum creatinine value rises after hemorrhage; if there are excessive gastrointestinal, urinary, or insensible fluid losses; or if extensive burns are present, particularly if access to fluids is restricted (e.g., comatose, sedated, or obtunded patients).

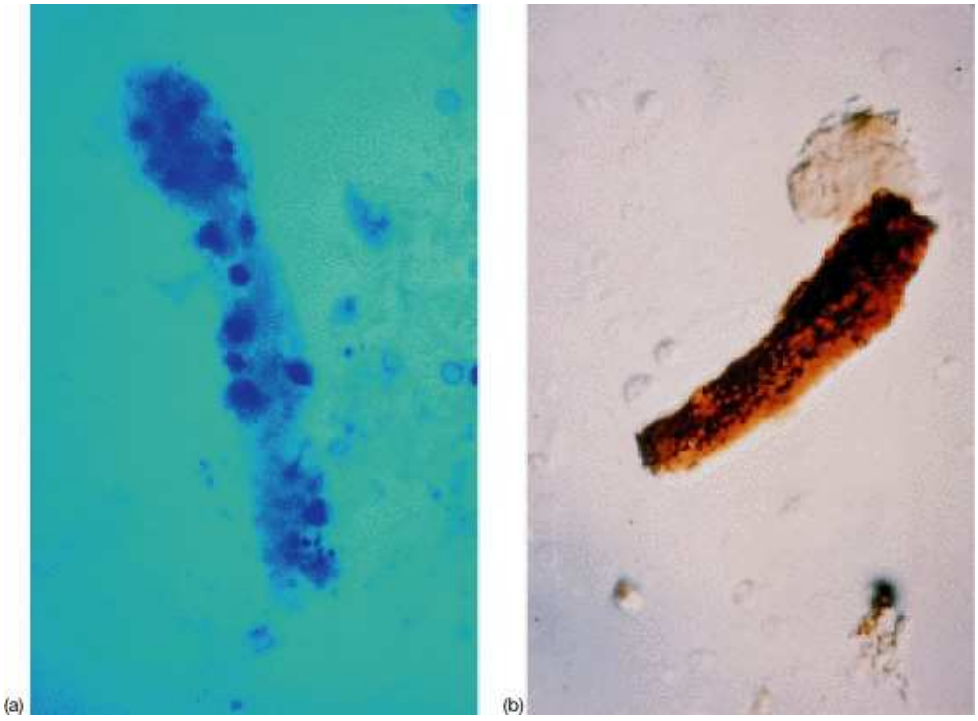
Definitive diagnosis of prerenal azotemia hinges on prompt resolution of ARF after restoration of renal perfusion. There is a high likelihood of ischemic ATN if ARF follows a period of severe renal hypoperfusion and persists despite restoration of renal perfusion.

Urinalysis

Assessment of the urine is a mandatory and inexpensive tool in the evaluation of ARF. Urine volume is a relatively unhelpful parameter in the differential diagnosis. Anuria suggests complete urinary tract obstruction, but it may be a complication of severe prerenal or intrinsic renal azotemia.

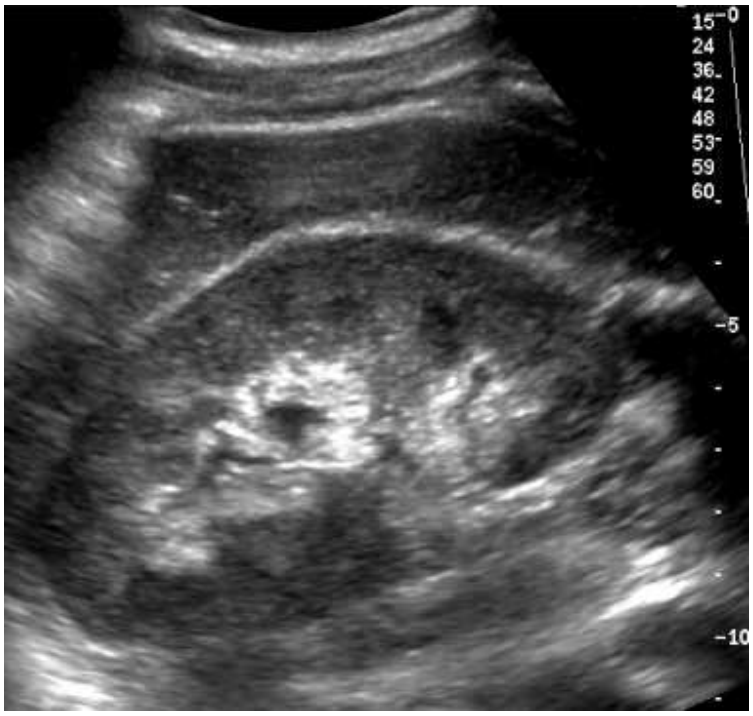


A hyaline cast 'fluffy' appearance due to the fibrillary substructure of Tamm–Horsfall glycoprotein. (b) A hyaline–granular cast. (c) A finely granular cast (d) A waxy cast



A red cell cast seen in urine with (a) and without (b) counterstaining

(a) An erythrocyte cast. Inset a haemoglobin cast. (b) A leucocyte cast (c) An epithelial cast (d) A fatty cast



Longitudinal view of a normal kidney

Wide fluctuations in urine output suggest intermittent obstruction. Patients with partial urinary tract obstruction may present with polyuria caused by secondary impairment of urine concentrating mechanisms. In contrast, analysis of the sediment and supernatant of a centrifuged urine specimen is valuable for distinguishing between prerenal, intrinsic renal, and posrenal azotemia and for elucidating the precise cause of intrinsic renal azotemia.

URINE SEDIMENT IN THE DIFFERENTIAL DIAGNOSIS OF ACUTE RENAL FAILURE

Normal or Few Red Blood Cells or White Blood Cells
Perenal azotemia Arterial thrombosis or embolism Hemolytic-uremic syndrome or thrombotic thrombocytopenic purpura Postrenal azotemia
Granular Casts
Acute tubule necrosis (muddy brown) Glomerulonephritis or vasculitis Interstitial nephritis
Red Blood Cell Casts
Glomerulonephritis or vasculitis Malignant hypertension
White Blood Cell Casts
Acute interstitial nephritis or exudative glomerulonephritis Severe pyelonephritis
Eosinophiluria (>5%)
Allergic interstitial nephritis (antibiotics > NSAIDs) Atheroembolic disease
Crystalluria
Acute urate nephropathy Calcium oxalate (ethylene glycol toxicity) Radiocontrast agents

Confirmatory Tests

The pattern of change in serum creatinine concentration often provides clues to the cause of ARF. Prerenal azotemia is typified by rapid fluctuations in creatinine that parallel changes in hemodynamic function and renal perfusion. Additional diagnostic clues can be gleaned from routine biochemical and hematologic tests. Hyperkalemia, hyperphosphatemia, hypocalcemia, and increased concentrations of serum uric acid and creatine kinase (CK3 isoenzyme) suggest a diagnosis of rhabdomyolysis. Severe anemia in the absence of hemorrhage may reflect the presence of hemolysis, multiple myeloma, or thrombotic microangiopathy (e.g., HUS, TTP, toxemia, disseminated intravascular coagulation, accelerated hypertension, SLE, scleroderma, radiation injury). Imaging of the urinary tract by plain film of the abdomen, ultranography, computed tomography (CT), or magnetic resonance imaging is useful to distinguish between acute and chronic renal failure and to exclude acute obstructive uropathy. However, the capacity of ultrasonography to determine cortical thickness, differences in cortical and medullary density, and the integrity of the collecting system, in addition to kidney size, makes it the screening modality of choice in most cases of ARF. Magnetic resonance angiography (MRA) of the kidneys is extremely useful for detecting renal artery stenosis.

Renal biopsy[1,2,34]:

It is usually reserved for patients in whom prerenal and postrenal failure have been excluded and the cause of intrinsic renal azotemia is unclear. Renal biopsy is particularly useful when clinical assessment, urinalysis, and laboratory investigation suggest diagnoses other than ischemic or nephrotoxic injury that may respond to specific therapy. Examples include anti-glomerular basement membrane disease and other forms of necrotizing glomerulonephritis, vaculitis, HUS and TTP, allergic interstitial nephritis, myeloma cast nephropathy, and acute allograft rejection.

Renal Failure Indices for Differentiation of Prerenal Azotemia and Acute Tubule Necrosis[41,42]

Analysis of urine and blood biochemistry is useful for discriminating between the major categories of ARF, namely prerenal azotemia and intrinsic renal azotemia caused by ischemia or nephrotoxins.

It should be noted that the FENa is usually less than 1.0% in ARF caused by urinary tract obstruction, glomerulonephritis, or disease of the renal vasculature, and other parameters must be employed to distinguish these conditions from prerenal azotemia.

Urine Indices Used in the Differential Diagnosis of Prerenal and Intrinsic Renal Azotemia*[41,42,43]

DIAGNOSTIC INDEX	PRERENAL AZOTEMIA	INTRINSIC AZOTEMIA
Fractional excretion of Na^+ (%) $\frac{UNa \times Pcr}{PNa \times Ucr} \times 100$	<1	>1
Urinary Na^+ concentration (mEq/L)	<10	>20
Urinary creatinine/plasma creatinine ratio	>40	<20
Urinary urea nitrogen/plasma urea nitrogen ratio	>8	<3
Urine specific gravity	>1.018	<1.012
Urine osmolality (mOsm/kg H_2O)	>500	>250
Blood urea nitrogen/creatinine ratio	>20	<10-15
Renal failure index (Una/Ucr/Pcr)	<1	>1
Urine sediment	Hyaline casts	muddy brown granular casts

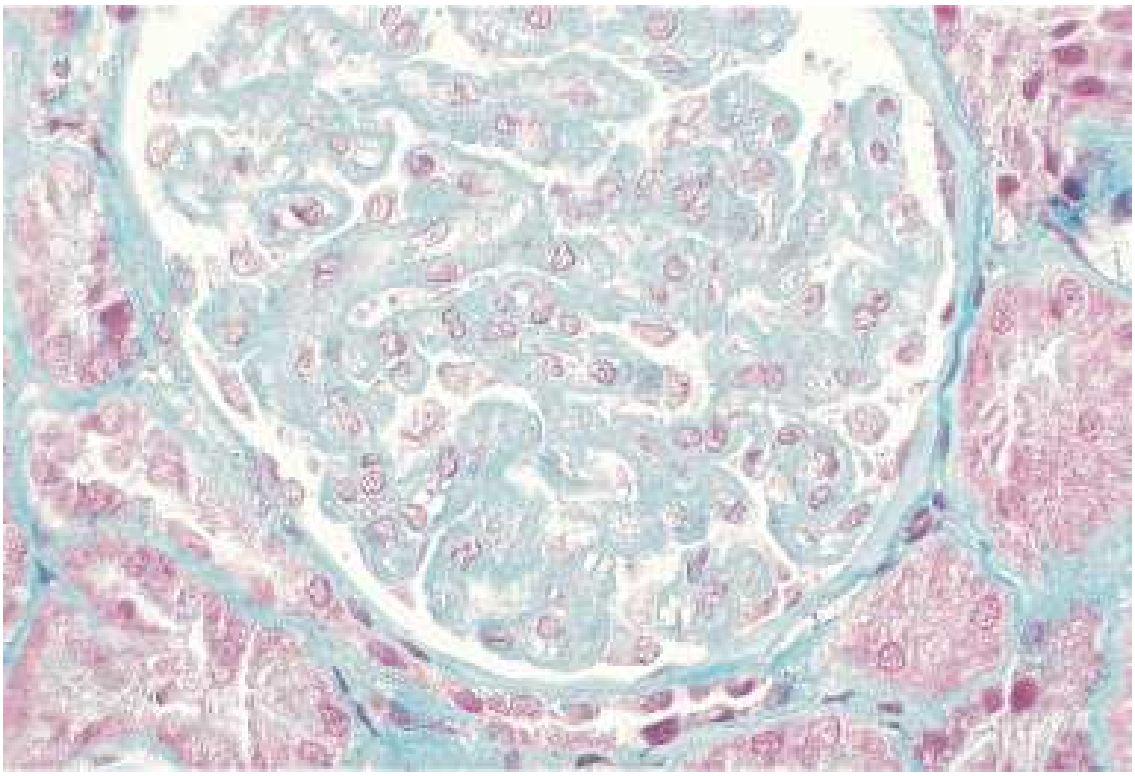
* The most sensitive indices are fractional excretion of Na^+ and the renal failure index. PNa, Plasma Na^+ concentration; Pcr, Plasma creatinine concentration; UNa, Urine Na^+ concentration; Ucr, Urine creatinine concentration.

Acute Renal Failure in Pregnancy

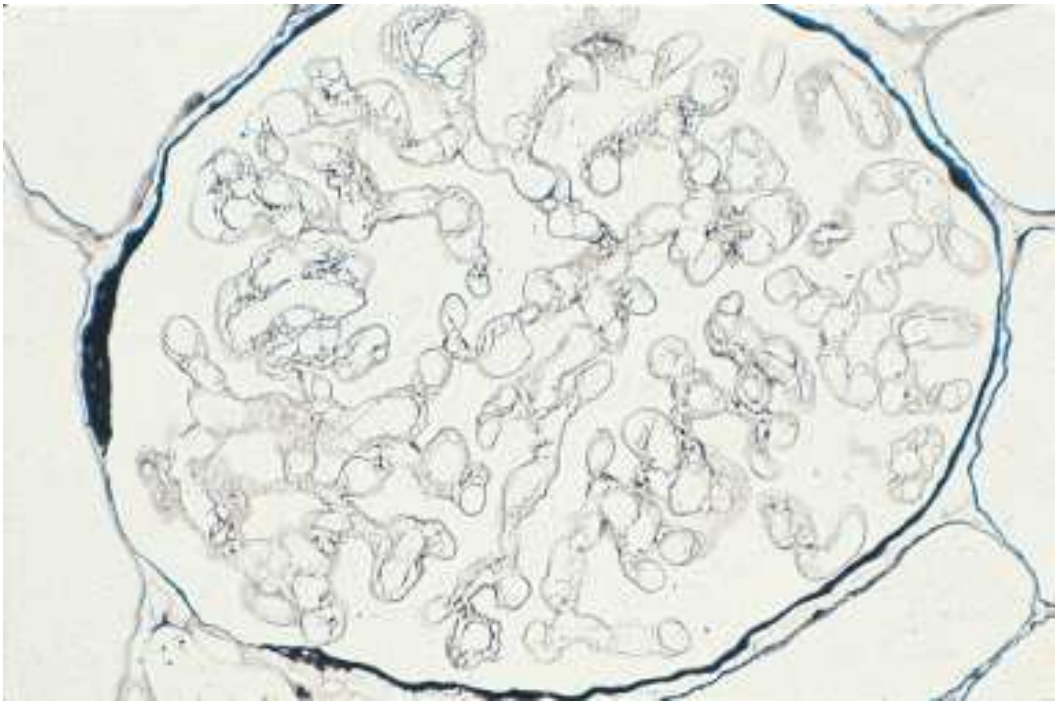
The incidence of ARF requiring dialysis complicating pregnancy in industrialized countries is approximately 1 in 20,000 births.[44] ATN induced by nephrotoxic abortifacients is still a relatively common cause of ARF in developing countries but is rarely seen in the developed world.[44,45] Ischemic ATN, severe toxemia of pregnancy, and postpartum HUS and TTP are the most common causes later in pregnancy. Ischemic ATN is usually provoked by postpartum hemorrhage or abrupio placenatae and less commonly by amniotic fluid embolism or sepsis.

A distinct variant of pre-eclampsia, the HELLP syndrome (hemolysis, elevated liver enzymes, low platelets), is characterized by an initial benign course that can rapidly deteriorate with marked hemolysis and derangement of coagulation and hepatic and renal function. Acute fatty liver of pregnancy (AFLP) occurs in approximately 1 in 7000 pregnancies. It includes acute renal impairment, probably by triggering intrarenal vasoconstriction, as in other hepatorenal syndrome.

Acute bilateral pyelonephritis may also precipitate ARF in pregnancy and should be obvious from clinical assessment (fever, flank pain) and routine urinalysis (bacteria, leukocytes) and laboratory tests (leukocytosis, increases in serum creatinine from basal levels.



Renal biopsy 8 days postpartum in a patient with pure pre-eclampsia
Thickening of capillary walls with lumens narrowed by swollen endothelium



Silver stain of the same glomerulus seen in above fig. Diffuse 'double contours' of capillary walls

ARF IN THE TROPICS[4]

The basic pathophysiological changes and management of ARF remain the same for a patient in the tropics or in the other parts of the world. However, there are a number of important differences in the pattern of ARF in the tropics. While post-surgical and post-traumatic acute renal failure is more common in the west, in the developing countries of the tropical region, ARF usually follows medical conditions like diarrhoeal diseases, envenomation from poisonous snakes, insects and toxic plants, chemicals, obstetric accidents, septic abortions, tropical infections and intravascular haemolysis following various conditions.

(i) Acute Renal Failure In Malaria[2,4]

The incidence of ARF in patients with P. falciparum malaria may vary from 4% to 60%. Severe jaundice, hypoglycaemia, hyponatraemia, hypercatabolic state, hyperuricaemia, encephalopathy, disseminated intravascular coagulopathy (DIC) and oliguric acute renal failure may set in during the course of the disease.Parasitized red blood cells become fragile leading on to severe intravascualr haemolysis. Treatment with primaquine or pyrimethamine may further induced intravascular haemolysis in patients with G6PD deficiency. Massive intravascular haemolysis leads on to frank haemoglobinuria which is characteristics of “black water fever”.

Heavy parasitaemia and the damaged red cells may adhere to the vascular endothelium or to other red cells leading to interference with microcirculation resulting in renal ischaemia and acute renal failure.Therapy should include early diagnosis, parenteral antimalarials, and supportive treatment for the hypercatabolic state, DIC, shock and acute renal failure. Dialysis is indicated when there is hypercatabolic state, hyperkalaemia or acute renal failure.

(ii) Acute Renal Failure Following Intravascular Haemolysis In G6PD Deficiency

Acute intravascular haemolysis is an uncommon cause of ARF in the Western countries. However, in the tropics, acute haemolysis in G6PD deficient individuals is a frequent cause of ARF. In India, 5-10% of ARF following intravascular haemolysis is in individuals deficient in G6PD. The incidence of G6PD deficiency varies from 2.2% to 15% in various ethnic groups in India. Patients often develop a haemolytic crisis within hours of exposure to the drugs or toxins, causing haemolysis. The commonly incriminated drugs are primaquine, sulphonamides, acetylsalicylic acid, nitrofurantion, nalidixic acid, furazolidone, phenazopyridine and chloroquine.

(iii) Acute Renal Failure Due To Snake Bite[2,4,7,15]

Snake bite is an occupational hazard in the rural areas in the tropics. Of the 2700 species of snakes recognized in the world over, only 450 are poisonous and are distributed mainly in the tropical and subtropical areas.

Of the varius species of snakes seen in India, only a few like Indian cobra, krait, Russel's viper, Echis carinatus and sea snakes are poisonous.

Acute and renal failure is the most frequently reposted and clinically important effect of envenomiation following viper and krait bites. The venom of elapid snakes is mainly neurotoxic and renal involvment in unsual.

Clinical Features

The clinical picture following the bite of vipers depends upon the dose of the venom injected and may vary from mild local reaction to extensive systemic manifestations. Pain and swelling appear at the site of the bite within a few minutes and may be followed by blister formation or ecchymosis. Bleeding diathesis is the major manifestations of systemic poisoning and is seen in over 65% of cases. This may manifest as continuous ooze from fang marks, venepuncture sites, haematemesis, melaena and haematuria. Patients may develop hypotension, shock and often there will be severe intravasculr haemolysis and DIC.

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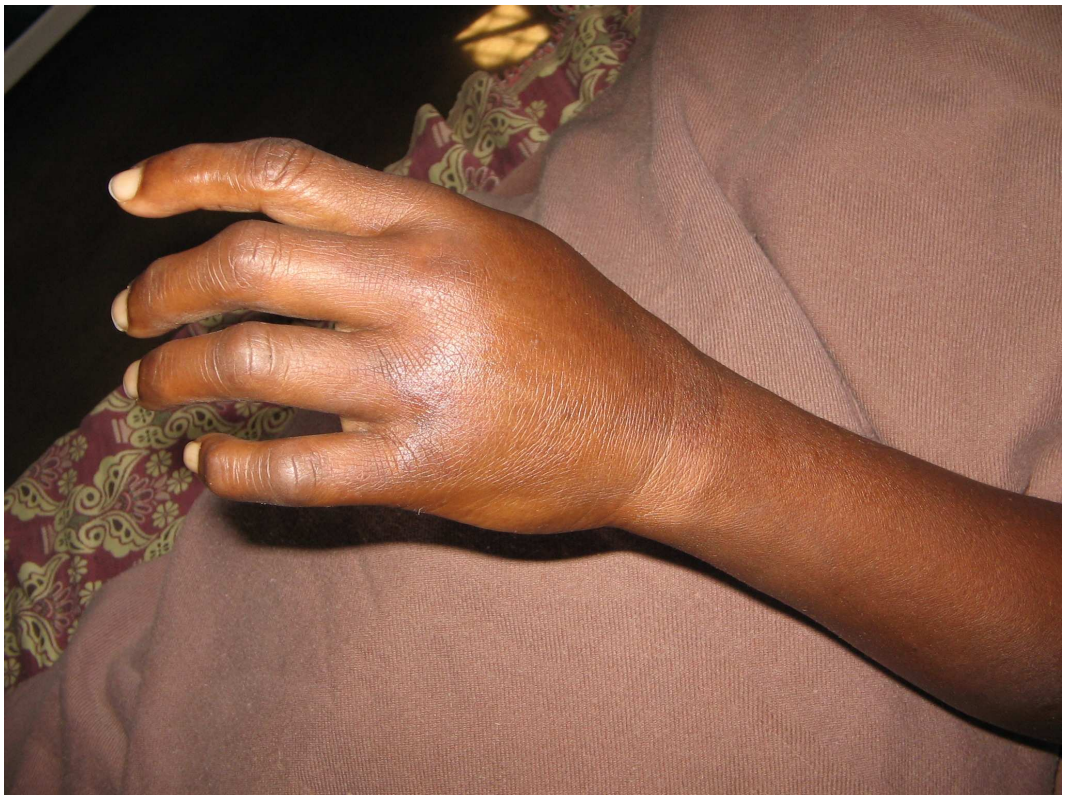


Snake Bite - cellulitis hand.

Snake-Viper. Its bite is the leading cause of ARF in this study.

The onset of renal failure is signaled by the development of oliguria or anuria within a few hours or as late as 96 hours after the bite. A history of passing cola-coloured urine may be obtained in 50% of patients. Non-oliguric renal failure has also been observed

Pathology : Light microscopic examination of the kidneys show acute tubular necrosis in 70-80% of patients. Other lesions including acute cortical necrosis, acute interstitial nephritis, necrotising arteritis of interlobular arteries, acute papillary necrosis and occasional crescentic glomerulonephritis have been described.

Pathogenesis: Snake venom contain a variety of enzymes, polypeptides, glycoproteins, neurotoxins, vasculotoxins and haemolysins. Phospholipase A is the principal constituent of the venom especially that of viper. It has damaging effect on mitochondria, vascular endothelium and the membranes of red cells, leucocytes and on the platelets. Haemorrhagin, another constituent of the venom acts on the vascular endothelium and produces bleeding. Disseminated intravascular coagulation (DIC) is well known to occur after envenomation.

The acute renal failure that ensues in snake bite poisoning could be due to a variety of reasons including direct nephrotoxic effect of the venom, hypovolaemia, hypotension, severe intravascular haemolysis, microangiopathic haemolytic anaemia, myoglobinuria or disseminated intravascular coagulaiton (DIC).

In the majority of cases, renal failure is due to acute tubular necrosis and recovery takes place in 2-3 weeks time. However, persistence of renal failure over a month is suggestive of acute cortical necrosis which has been confirmed by renal biopsy.

(iv) Leptospiral Acute Renal Failure[4,16]

Leptospirosis is very prevalent in the tropics. Recently, the incidence has become very high in Mumbai and in the southern states of India. Although seen all around the year, there is a sharp increase in the incidence during or soon after the rainy season and following floods. Usually, patients develop fever, chills, severe muscle aches and tenderness, subconjunctival haemorrhage, jaundice and later develops features of severe hepatic and non-oliguric acute renal failure. The pathognomonic features is the severe degree of acute interstitial nephritis. Acute tubular necrosis and glomerular lesions including mesangial proliferation and mesangiolysis

Renal involvement in leptospirosis is thought to occur from direct invasion of the renal tissue by the organism Hypovolaemia, hypotension, haemoglobinuria and myoglobinuria may also contribute to the development of acute renal failure. Definitive diagnosis is based on a positive culture of leptospira in the blood or by dark ground microscopy of urine or by a positive serology. The gold standard test is the microscopic agglutination test (MAT).

(v) Bee, Wasp, Hornet And Scorpion Stings[4,10,11]

Honey bees, hornet and wasps are stinging insects found in most of the tropical countries. An isolated sting by any of them will be followed by a local allergic reaction only. However, when a swarm of these insects attack an individual, a large dose of the venom gets injected leading on to systemic symptoms like vomiting, diarrhoea, hypotension, intravasculr haemolysis and rhabdomyolysis resulting in acute renal failure. A direct nephrotoxic role of these venoms has also been postulated. Scorpion venom often releases acetyl choline, catecholamines and other mediators with toxic effects on the neuromuscular system. They can also produce disseminated intravascular coagulation, myocarditis, cardiac failure, pancreatitis, pulmonary oedema, convulsions and hypotension finally resulting in acute renal failure.

(vi) Spider Bites

Spider bites are also common in the tropics. After the bite, there will be nausea, vomiting, salivation, sweating, muscular twitching, hypotension, respiratory paralysis and acute renal failure resulting from intravascular haemolysis, or direct nephrotoxicity as well as neurotoxicity.

(vii) Poisoning[4]

Copper sulphate, paraquat and formic acid poisoning can lead to ARF. Marking nut poisoning and consumption of organophasphorus compound can also lead to ARF. Symptomatic treatment and early haemodialysis are often necessary to tide over the crisis.

(viii) Obstetric Acute Renal Failure – in the tropics

In the tropics, the incidence of acute renal failure in pregnancy was as high as 20-22% in the 1960s. however, with the significant improvement in the care of pregnant women, and in the diagnosis and therapeutic advancement including safe hospital delivery practices, the reduction in the number of septic abortions and early intervention in the management of complicated pregnancies, the incidence has come down to less than 8% in 1990s.However, the mortality can be high in 15 to 20% of cases with severe septicaemia and multiorgan failure.

(ix) Acute Renal Failure Due To Surgical Causes

The incidence of acute renal failure following surgery have been quite low in the tropics till the 1980s when it was about 11% of all the cases of ARF in the major hospitals of our country. However, in the course of time, during the 1990s the incidence has increased to around 30%. While trauma and complicated major abdominal and cardiac surgery are the common causes for ARF in the developed countries, obstructive uropathies and postoperative complications following major abdomino-thoracic surgical interventions are the important causes in the developing countries.

COMPLICATIONS OF ACUTE RENAL FAILURE

ARF impairs renal excretion of Na^+, K^+, and water; divalent cation homeostasis; and urinary acidification mechanisms. As a result, ARF is frequently complicated by intravascular volume overload and multisystem involvement. The following are the important complications:[3,35,46]

METABOLIC[46]

- Hyperkalemia
- Metabolic acidosis
- Hyponatremia
- Hypocalcemia
- Hyperphosphatemia
- Hypermagnesemia
- Hyperuricemia

CARDIOVASCULAR

- Pulmonary edema
- Arrhythmias
- Pericarditis
- Pericardial effusion
- Pulmonary embolism
- Hypertension
- Myocardial infarction

GASTROINTESTINAL

- Nausea
- Vomiting
- Malnutrition
- Gastrointestinal
- Gastrointestinal hemorrhage

NEUROLOGIC

- Neuromuscular irritability
- Asterixis
- Seizures
- Mental status changes

HEMATOLOGIC

- Anemia
- Bleeding

INFECTIOUS

- Pneumonia
- Septicemia
- Urinary tract infection

OTHERS

- Hiccups
- Elevated parathyroid hormone
- Low total triiodothyronine and thyroxine
- Normal free thyroxine

MANAGEMENT OF ACUTE RENAL FAILURE

Prerenal Azotemia

By definition, prerenal azotemia is rapidly reversible on restoration of renal perfusion.Hypovolemia caused by hemorrhage is ideally corrected with packed red blood cells. In the absence of active bleeding or hemo dynamic instability, isotonic saline may suffice. Serum K^+ concentration and acid-base status should be monitored in all subjects. Cardiac failure may require aggressive management with loop diuretics, antiarrhythmic drugs, positive inotropes, preload – and/or after load-reducing agents, and mechanical aids such as an intra-aortic balloon pump.

Intrinsic Renal Azotemia

Aggressive restoration of intravascular volume dramatically reduces the incidence of ATN after major surgery or trauma, burns, or cholera.[49,50] The administration of high-dose intravenous diuretics to individuals with oliguric ARF is commonly practiced. Although this strategy may minimize fluid overload, there is no evidence that it alters the mortality rate or the dialysis free survival rate. Indeed, a recent study reported an increased risk of death and nonrecovery of renal function in patients treated in this manner.[51,52] ARF caused by other intrinsic renal diseases, such as acute glomerulonephritis or vasculitis, may respond to corticosteroids, alkylating agents, and/or plasmaphereis, depending on the primary disease.

Aggressive control of systemic arterial pressure is of paramount importance for limiting renal injury in malignant hypertensive nephrosclerosis, and other vascular diseases.

MANAGEMENT OF COMPLICATIONS

Complication	Treatment
Intravascular volume overload	Restrictionof salt (<1-1.5 g/day) and water (<1 L/day) Consider diuretics (usually loops ± thiazide) Ultrafiltration
Hyponatremia	Restriction of oral and intravenous free water
Hyperkalemia	Restriction of dietry potassium Discontinue K^+ supplements or K^+ - sparing diuretics K^+ - binding resin Loop diuretic Glucose (50 mL of 50% dextrose) + insulin (10-15 U regular insulin) IV Sodium bicarbonate (50-100 mEq IV) Calcium gluconate (10 mL of 10% solution over 5 min) Dialysis / hemofiltration
Metabolic acidosis	Restriction of dietary protein Sodium bicarbonate (if HCO_3 - < 15 mEq/L) Dialysis / hemofiltration
Hyperphosphatemia	Restrictionof dietary phosphate intake Phosphate-binding agents (calcium acetate, sevelamer)
Hypocalcemia	Calcium carbonate (if symptomatic or sodium bicarbonate is to be administered)
Hypermagnesemia	Discountinue magnesium-containing antacids
Nutrition	Restriction of dietary protein (<0.8 g/kg/day up to 1.5 g/kg/day on continuous venovenous hemodialysis) 25-30 keal/day Knteral route of nutrition preferred
Drug dosage	Adjust all doses for glomerular filtration rate and renal replacement modality

Indications and Modalities of Dialysis

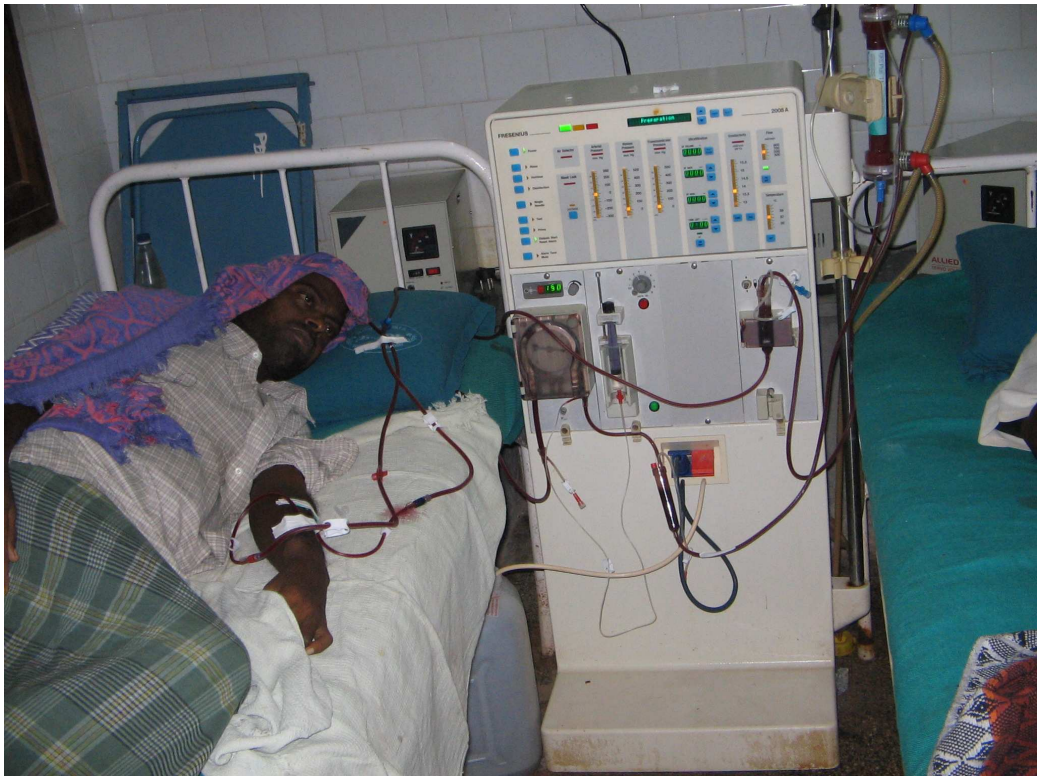
Dialysis does not hasten recovery from ARF. Indeed, early and unnecessary hemodialysis may potentially exacerbate renal hypoperfusion,[53] because transient hypotensionis a common complication of this treatment modality, and leukocytes activated on exposure to dialysis membranes may potentially aggravate ischemic renal injury.

Absolute indications for the commencement of renal replacement therapy include symptomatic uremia (asterixis, pericardial rub, encephalopathy) and acidosis, hyperkalemia, or volume overload that proves refractory to medical management.

Peritoneal Dialysis

Peritoneal dialysis in ARF is effected through a temporary intraperitoneal catheter. Peritoneal dialysis has the advantage of being relatively "low-tech" and portable, which facilitates its use in remote or resource-constrained areas.[54] Systemic hypotension is typically avoided, and other benefits include the avoidance of systemic anticoagulation and need for angioaccess. Solute clearance and control of metabolic disarry in critically ill patients may be inferior to that achieved with continuous venovenous hemofiltration, and this has been associated with an adverse outcome in infection-associated ARF.[55] Other drawbacks include the risk of visceral injury during catheter placement and peritonitis subsequently.[56]

Patient undergoing Peritoneal dialysis



Patient on Hemodialysis

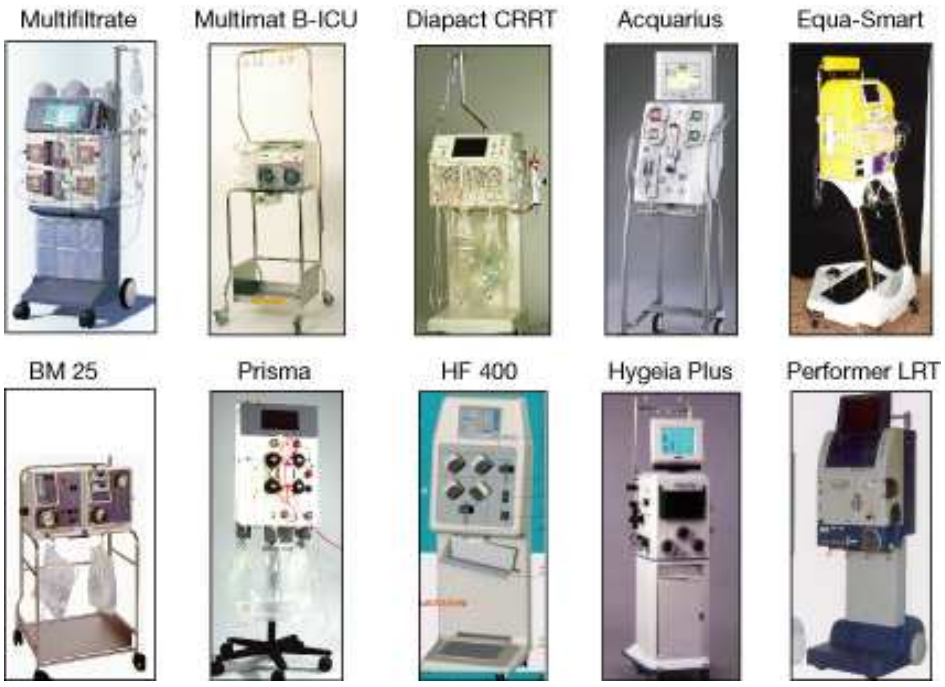
Acute Intermittent Hemodialysis

Acute intermittent hemodialysis has been mainstay of renal replacement therapy in ARF. Typically, patients undergo dialysis for 3 to 4 hours daily or on alternate days depending on their catabolic state. Vascular access for short-term hemodialysis or hemofiltrationis usully achieved with the use of a double-lumen catheter inserted into the internal jugular vein. Subclavian cannulation offers an alternative, but this access is associated with high rates of venous stenosis.[56] Femoral vein catheterization is technically easy and relatively free of complications.

It is useful for patients who cannot tolerate the Trendelenburg position Jugular lines are preferred for more prolonged treatment courses, but with careful nursing management it is possible to maintain a femoral line in situ in the bed-bound patient without incurring a significant excess infection risk.[56]

The major complications of acute intermittent hemodialysis relate to rapid shifts in plasma volume and solute composition, the angioaccess procedure, and the necessity for anticoagulation.[55] Intradialytic hypotension is common in patients undergoing acute intermittent hemodialysis. In addition, hypotension can further compromise renal perfusion and exacerbate tubule necrosis.

Hemodialysis - femoral catheter.



The latest renal replacement therapy machines used in the treatment of acute renal failure

The immediate management of hypotension involves the discontinuation of hemofiltration, placing the patient in the Trendelenburg position, and rapid infusion of 250 to 500 mL of normal saline.

The dialysis disequilibrium syndrome is a self-limited condition characterized by nausea, vomiting, headache, altered consciousness, and, rarely, seizures or coma.[53] It typically occurs after a first dialysis in very uremic patients. The syndrome is triggered by rapid movement of water into brain cells after the development of transient plasma hypoosmolality as solutes are rapidly cleared from the blood-stream during dialysis.

Slow Continuous Hemofiltration And Hemodialysis

Many patients with ATN are critically ill, hypercatabolic, and hemodynamically unstable. They frequently have large obligate fluid requirements owing to intravenous medication administration and parenteral alimentation. In this setting, ultrafiltration of large volumes of plasma over a relatively short period by acute intermittent hemodialysis may induce circulatory compromise. Even if tolerated hemodynamically, acute intermittent hemodialysis may not achieve adequate ultrafiltration or solute clearance to avoid life-threatening pulmonary edema or uremia.

Hemofiltration is better in those situations. Following are various methods: Continuous venovenous hemodialysis (CVVHD), continuous venovenous hemodiafiltration (CVVHDF) are the techniques favored by most centers.

Although most patients recover from ARF, a few (<5%) require long-term renal replacement therapy. With this in mind, every effort should be made to preserve veins (i.e., avoid venipuncture) on the nondominant arm of patients with severe ARF, because these veins may be required for chronic hemodialysis access (arteriovenous fistula) at a later date.

Postrenal Azotemia

Management of postrenal azotemia usually involves a multidisciplinary approach. Urethral or bladder neck obstruction is usually relieved temporarily by transurethral or suprapubic placement of a bladder catheter, which provides a window for identification and treatment of the obstructing lesion. Similarly, ureteric obstruction may be treated initially by percutaneous catheterization of the dilated ureteric pelvis or ureter.

OUTCOME

The mortality rate among patients with acute intrinsic renal azotemia approximates 50% and has changed little over the past three decades.[47,48,57]

Mortality rates differ markedly, depending on the cause of ARF: approximately 15% in obstetric patients, 30% in patients with toxin-related ARF, and 60% to 90% in patients with sepsis.[47,48] Factors associated with a poor prognosis include male sex, advanced age, oliguria (<400 mL/day), and a rise in the serum creatinine value of more than 3 mg/dL. These factors reflect more severe renal injury and failure of other organ system.[47,48]

With appropriate supportive management of ARF, death is usually a consequence of the primary disease that induced ARF and rarely of a direct complication of uremia per se. Most patients who survive an episode of ARF recover sufficient renal function to live normal lives.[58] However, 50% have subclinical functional defects in glomerular filtration, tubule solute transport, ARF is irreversible in approximately 5% of patients, usually as a consequence of complete cortical necrosis, and necessitates long-term renal replacement therapy with dialysis or transplantation.

MATERIALS AND METHODS

STUDY PERIOD:

This study was conducted in Thanjavur Medical College Hospital during the period of August 2005 to December 2005.

STUDY DESIGN:

Single centre non-randomised prospective study

STUDY POPULATION:

A total of 70 patients were studied. This includes those patients admitted in Nephrology ward and various medical units and also those patients admitted in ID ward RMH, surgical and orthopaedics wards for whom nephrologist had been called for renal failure management.

There were 38 male and 32 female patients ranging from 15 years to 65 years of age, average age 38 years. These patients were subjected to detailed history (e.g., Previous renal disease, drug ingestion, etc.) and careful clinical examination. They were subjected to following investigations

Blood- Urea, creatinine,

Sugar, Electrolytes like Na^+ , K^+

Hb, Total count, Differential Count

Urine: Albumin, sugar, deposits

Renal failure indices:

Urine-Na^+

Urine-Creatinine

Urine creatinine/plasma creatinine ratio

Plasma urea/ Creatinine ratio

[1]**Fractional Excretion of Na^+(%)**

$$= \left[\frac{\text{Urine Na+ X Sr.Creatinine}}{\text{Sr.Na}^+ \text{ X Urine Creatinine}} \right] \quad \text{X 100}$$

[1]**Renal Failure index =**

$$\frac{\text{Urine Na}^+}{\text{[Urine Creatinine/ Plasma creatinine]}}$$

Other relevant blood investigations like clotting time, Liver function tests in relevant cases. Abdominal ultrasonagram also done in these cases to assess the size and echogenic pattern of the kidneys.

Inclusion Criteria:

Diagnostic criteria used in this study are a relevant history which could have precipitated Acute Renal Failure, symptoms like oliguria (urine output less than 400ml/day or less than 20ml / hour) anuria (less than 100ml in 24 hours)[1,4] with elevated renal parameters like urea (Normal value 15-45mg%), and Creatinine (Normal: 0.6-1.2 mg%) or non oliguric patients with elevated renal parameters. Other symptoms those considered are oedma legs, facial puffiness, dyspnoea, vomiting and hiccup, if they fulfill the above criteria.

These patients were subjected to ultrasonagram abdomen. If the size of the kidneys are less than 7 cm, taken as contracted (scarred) kidneys indicative of chronic renal failure and excluded from this study. Also USG abdomen was useful in evaluating obstruction of the urinary collecting system.

Exclusion Criteria:

Those patients with previous history of Hypertension, Diabetes Mellitus and previous history of renal diseases were excluded from this study.

Present blood sugar value suggestive of diabetes also excluded. Ultrasoundwise patients having bilaterally contracted kidneys were excluded. Patients who were anaemic also excluded as it may be an indicator of chronic renal disease (except for one patient for whom PPH was the precipitating event of ARF). Patients admitted in IMCU with multiorgan dysfunction were not included in this study.

Variables Recorded:

Variables recorded during this study were:

1. Time of presentation to hospital after the onset of symptoms.
2. Variation in biochemical reports depends on stage of ARF.
3. Presence or absence of symptoms.
4. Type of renal involvement weather prerenal or renal.
5. Comorbid conditions like trauma, surgery, sepsis.
6. Patients belonging to various age groups

ARF was diagnosed according to the following criteria:

1. Rapid reduction of GFR leading to sudden / progressive elevation of Blood Urea (>40mg%) and serum creatinine (>1.5mg%).
2. Absence of pre-existing renal disease.

Once the diagnosis of ARF was made, the underlying aetiology was determined by combining, history, examination and investigation reports. At the same time patients were started on appropriate conservative therapy. Whenever possible, the aetiological factors were treated. PD was begun (a) When biochemical or clinical deterioration occurred despite conservative measures or (b) When patients presented in severe uremia. HD was started when (a) PD failed to control uremic symptoms or (b) PD was contraindicated.

The observations and results were recorded and analysed.

RESULTS AND OBSERVATION

This study included 70 patients, admitted in nephrology ward, medical wards and surgical wards. The end point taken for this study was symptomatic improvements as well as improvement in biochemical results. In a few patients who died during study, death was taken as final end point. The data were analysed prospectively in these patients.

PATIENT CHARACTERISTICS IN GENERAL:

Most of the patients studied were from the areas in and around Thanjavur, Thiruvarur, Ariyalur, Nagapattinam and Pudukkottai and majority of them were involved in agricultural works and belong to low socio-economic status. Many patients admitted in nephrology ward had been referred from our medical wards, OG ward and Infectious Diseases ward. Patients admitted in medical ward are equally admitted directly as well as referred from other district head quarters and taluk hospitals. About surgical cases, nearly all were attended as 'Nephrologist call over' basis and patients were examined and renal failure mangement was given in their wards itself.

The most common (97.1%) complaint was oliguria or anuria. Average hospital stay was 10 days, excluding surgical, ortho and burns ward cases.

AETIOLOGY:

The breakup figure of Aetiology in these 70 cases of acute renal failure are as follows:

S. No	Aetiology	No. of Patients	Percentage
1	Snake Bite	20	28.6%
2	Postdiarrhoeal	19	27.1%
3	Post surgical	8	11.4%
4	RPGN	5	7.1%
5	Burns	4	5.7%
6	Obstetric	3	4.3%
7	Posttraumatic	3	4.3%
8	Sepsis	2	2.9%
9	Other causes	4	5.7%
10	Post renal	2	2.9%
	Total	**70**	

Other causes included : ARF due to

Kadhandu bite* - 1 case

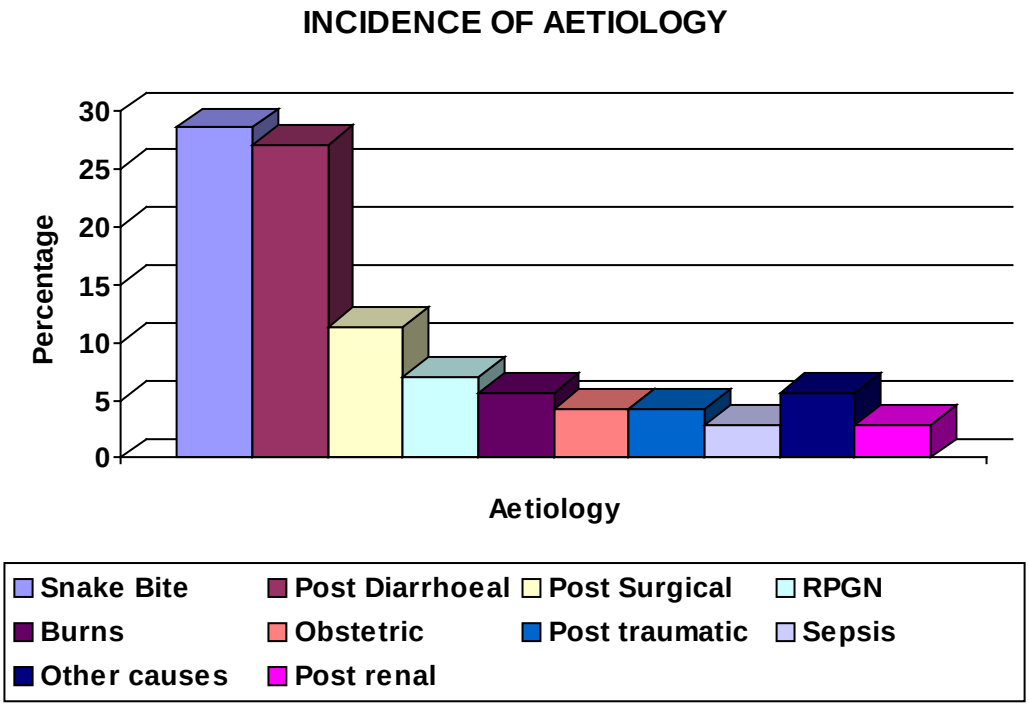
$CuSo_4$ poisoning - 1case

Paraquat poisoning - 1 case

Leptospirosis - 1 case

* Foot Note

Kadhandu is a type of wasp. It's bite can induce renal failure.[10,11]


INCIDENCE OF AETIOLOGY
30
25
20
15
10
5
0
Percentage
Aetiology
Snake Bite
Post Diarrhoeal
Post Surgical
RPGN
Burns
Obstetric
Post traumatic
Sepsis
Other causes
Post renal

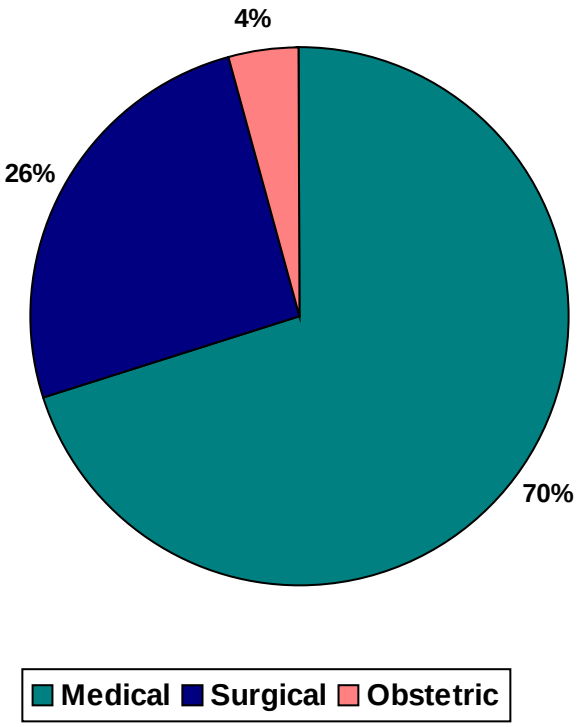
INCIDENCE ACCORDING TO TYPE OF RENAL DISEASE :

Medical		Surgical		Obstetric	
Causes	**No. of. Cases**	**Causes**	**No. of Cases**	**Causes**	**No. of Cases**
Snake Bite	20	Post surgical	8	Toxemia	1
Post Diarrhoeal	19	Burns	4	PPH	1
RPGN	5	Post traumatic	3	Midtrimester Abortion	1
Poisoning	2	Post renal	2	-	-
Kadhandu Bite	1	Sepsis	1	-	-
Leptospirosis	1	-	-	-	-
Sepsis	1	-	-		
Total	**49**	**Total**	**18**	**Total**	**3**

Incidence of medical, surgical and obstetric causes is as follows:

Aetiology	No. of Patients	Percentage
Medical	49	70%
Surgical	18	25.7%
Obstetric	3	4.3%

INCIDENCE OF MEDICAL, SURGICAL AND OBSTETRIC CAUSES

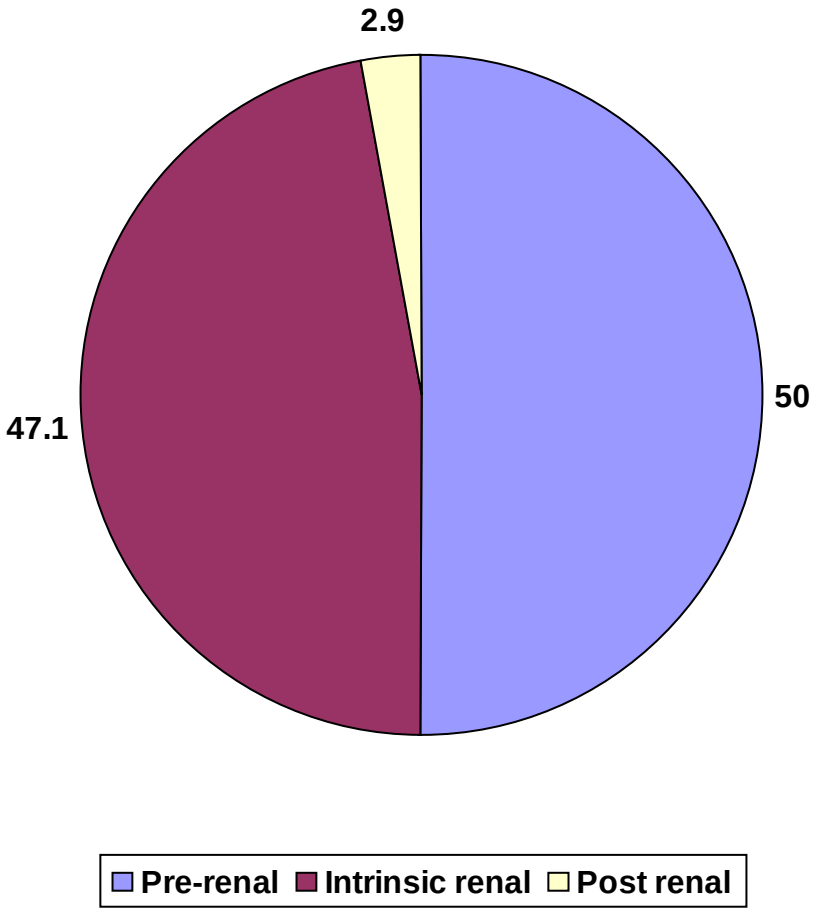


Aetiology and Incidence of Prerenal renal and postrenal failure is as follows:

Prerenal		Renal		Post Renal	
Causes	**No. of Cases**	**Causes**	**No. of Cases**	**Causes**	**No. of cases**
Post diarrhoeal	19	Snake Bite	20	Calculus Disease	1
Post Surgical	8	RPGN	5	Prostatic hypertrophy	1
Burns	4	Obstetric	3	-	-
Posttraunatic	3	Sepsis	1	-	-
Sepsis	1	Other cases	4	-	-
Total	**35**	**Total**	**33**	**Total**	**2**

Type of renal failure	No. of Patients	Incidence
Prerenal	35	50%
Intrinsic renal	33	47.1%
Post Renal	2	2.9%
Total	**70**	

INCIDENCE OF TYPE OF RENAL FAILURE

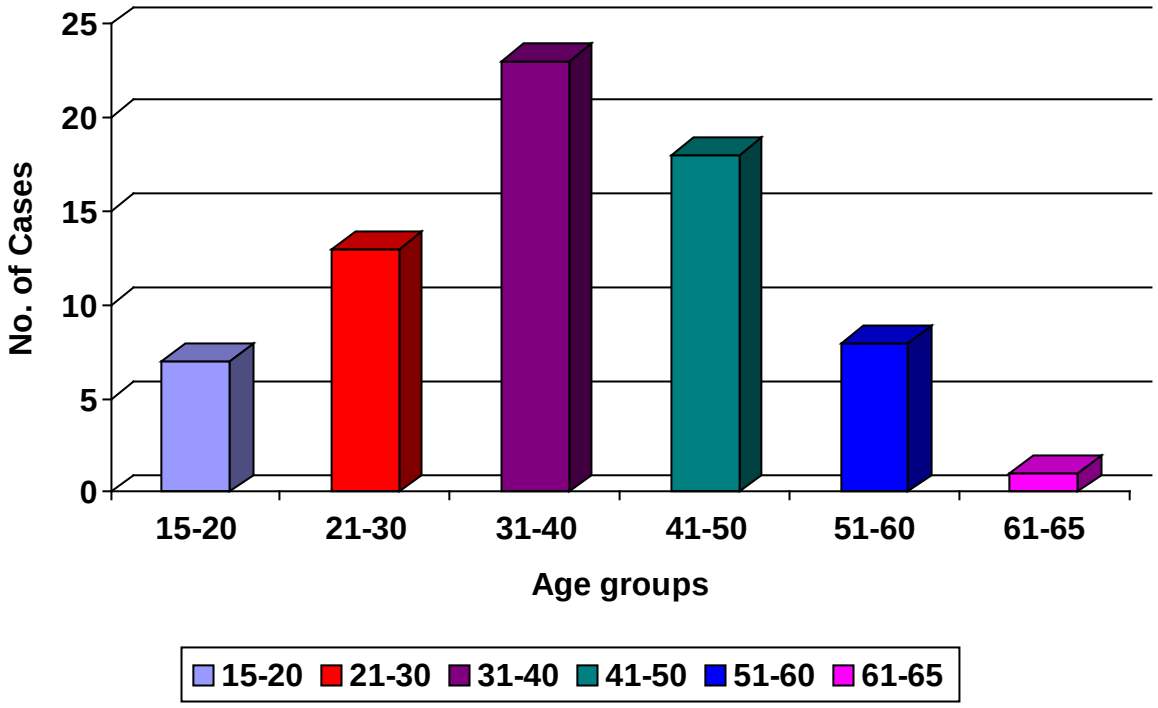


Age Characteristics of the patients:

Distribution of the patients according to age group is as follows:

Age Group	No. of. Patients	Percentage
15-20	7	10%
21-30	13	18.6%
31-40	23	32.9%
41-50	18	25.7%
51-60	8	11.4%
61-65	1	1.4%

NO. OF CASES ACCORDING TO AGE GROUP



Sex Characteristics:

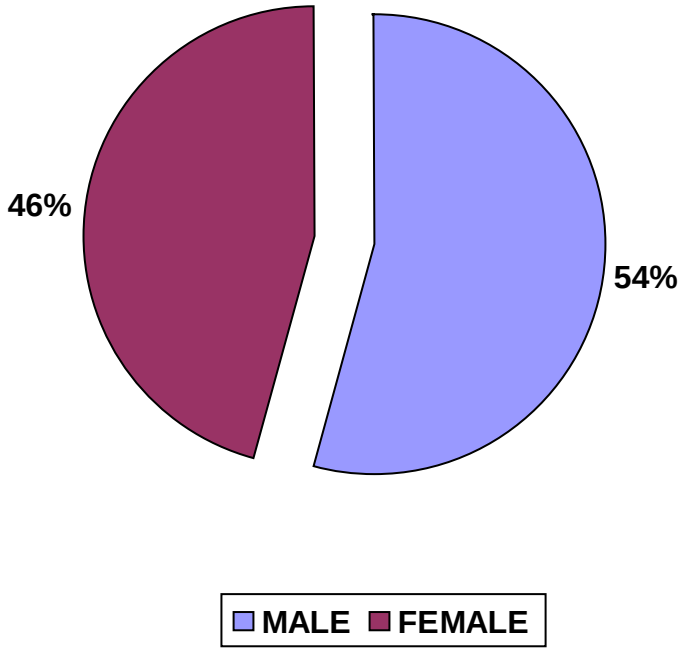
Out of the 70 patients 38[54.3%] were males and 32[45.7%] were females. Among those who expired [11 cases] 6 cases were males and 5 cases were females. Thus the proportional mortality according to sex was 54.5% and 45.5% respectively for males and females.

Sex	No. of Patients	Percentage
Male	38	54.3%
Female	32	45.7%
Total	**70**	

Male : Female

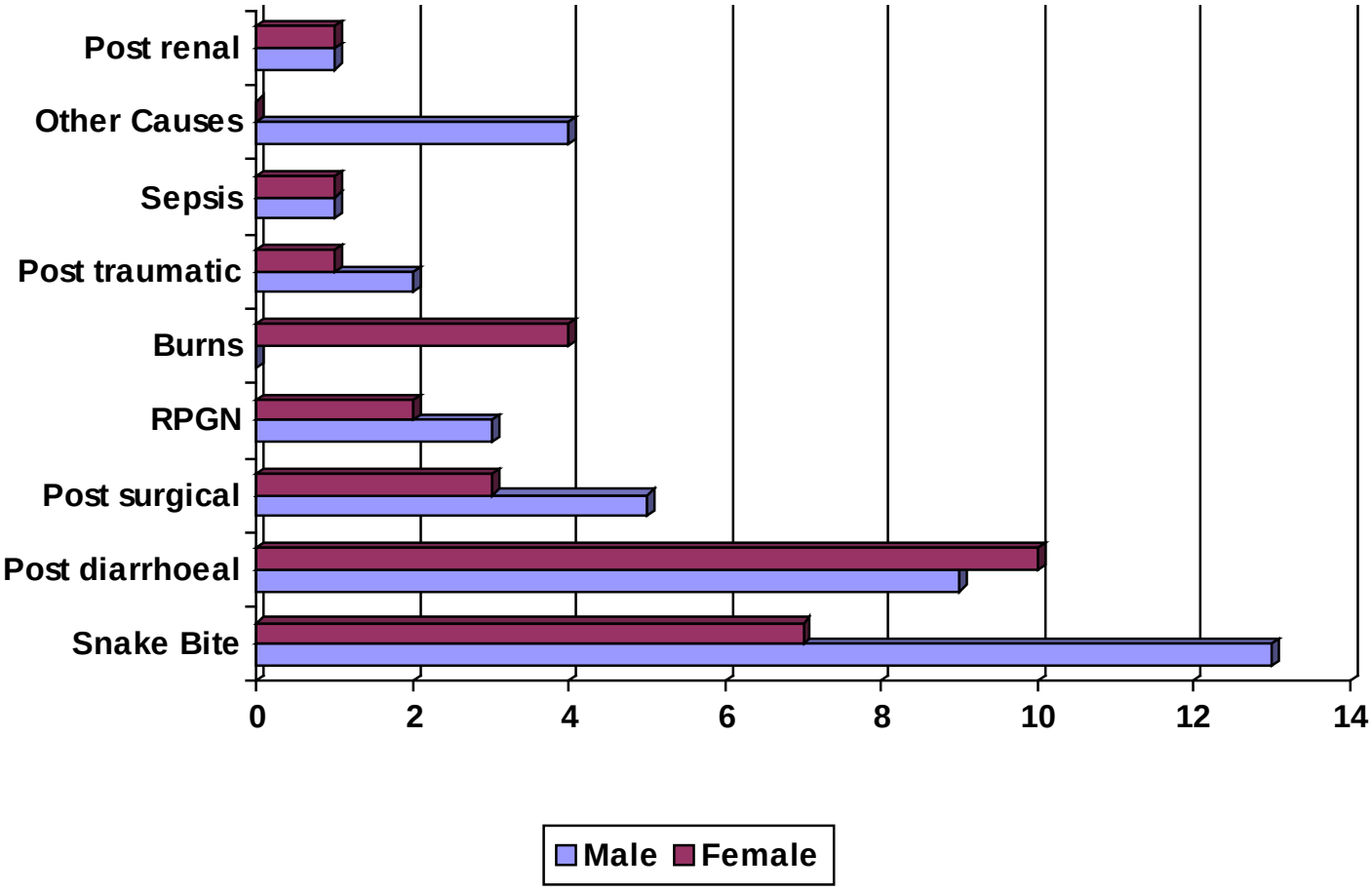
1.2 : 1

SEX RATIO OF ARF



Sex ratio depending on aetiology is as follows: Snake bite [Male 13 cases, Female 7 cases] more common in male patients, may be related to their place of working (agricultural fields). Poisoning going for renal failure more in males (2 cases) than females. Otherwise other aetiologies are equal in both sexes.

S. No	Aetiology	Male	Female
1	Snake Bite	13 cases	7 cases
2	Post Diarrhoeal	9cases	10 cases
3	Post Surgical	5cases	3 cases
4	RPGN	3 cases	2caes
5	Burns	0	4cases
6	Post traumatic	2 cases	1 case
7	Sepsis	1 case	1 case
8	Other causes i) Poisoning ii) Leptospirosis iii) Kadhandu Bite	 2cases 1case 1case	 0 0 0
9	Post renal	1 case	1case


Post renal
Other Causes
Sepsis
Post traumatic
Burns
RPGN
Post surgical
Post diarrhoeal
Snake Bite
0
2
4
6
8
10
12
14
Male
Female

RESULTS OF LABORATORY VALUE

Patients showing initial creatinine Value < 3mg%		Mortality	Proportional Mortality
No	Percentage		
38	54.3%	3	7.9%

Patients showing initial creatinine Value > 3mg%		Mortality	Proportional Mortality
No	Percentage		
32	45.7%	8	25%

Treatment given:

Among 70 patients, 35 patients [50%] were managed conservatively, 35 patients [50%] underwent dialysis

Total No. Patients	Conservative Management	Dialysis Therapy
70	35 cases (50%)	35 cases (50%)

Among the patients who received dialysis [35 cases],

Received Only PD	Received Only HD	Both PD & HD
24	2	9

Among those received peritioneal Dialysis

PD Once	PD Twice
19	5

Among those received HD

HD Once	HD Twice	HD Thrice	HD> Three
2	4	3	2

Dialysis was given mostly to intrinsic renal failure patients. Among 33 cases of Intrinsic renal failure, 24 patients (72.7%) received dialysis. Among this 8 patients [24.2%] received both PD and HD. 2 obstetric patients received Hemodialysis.

'Prerenal type renal failure patients were totally 35. Among this, only 9 patients (25.7%) received dialysis. Among this, 8 patients received only PD, 1 patient received both PD and HD.

Type of Renal failure	No. of patients	No. of patients received dialysis	Percentage
Prerenal	35	9	25.7%
Renal	33	24	72.7%
Postrenal	2	2	100%

All these patients who received dialysis in this 'prerenal' group initially showed urinary indices value in the range of 'prerenal' pattern. But later as the illness progressed and not responded to conservative line of management they were treated with dialysis and investigations taken at that time showed 'Intrinsic Renal' pattern.

Two Post renal cases, both underwent peritoneal dialysis once each.

Aetiology and management of ARF - an overview

S. No	Aetiology	Total No. of patients	Management		
			Conservative	PD	Both HD&PD
1	Snake Bite	20	6	9	5
2	Post Diarrhoeal	19	10	8	1
3	Post Surgical	8	8	-	-
4	RPGN	5	1	3	1
5	Burns	4	4	-	-
6	Post Traumatic	3	3	-	-
7	Obstetric	3	1	2 Patients received only HD	
8	Sepsis	2	2	-	-
9	Others	4	0	2	2
10	Post renal	2	0	2	0

Mortality:

Among the 70 patients studied, 11 patients expired. Among this 6 were males (54.5%) and 5 were females (65.5%)

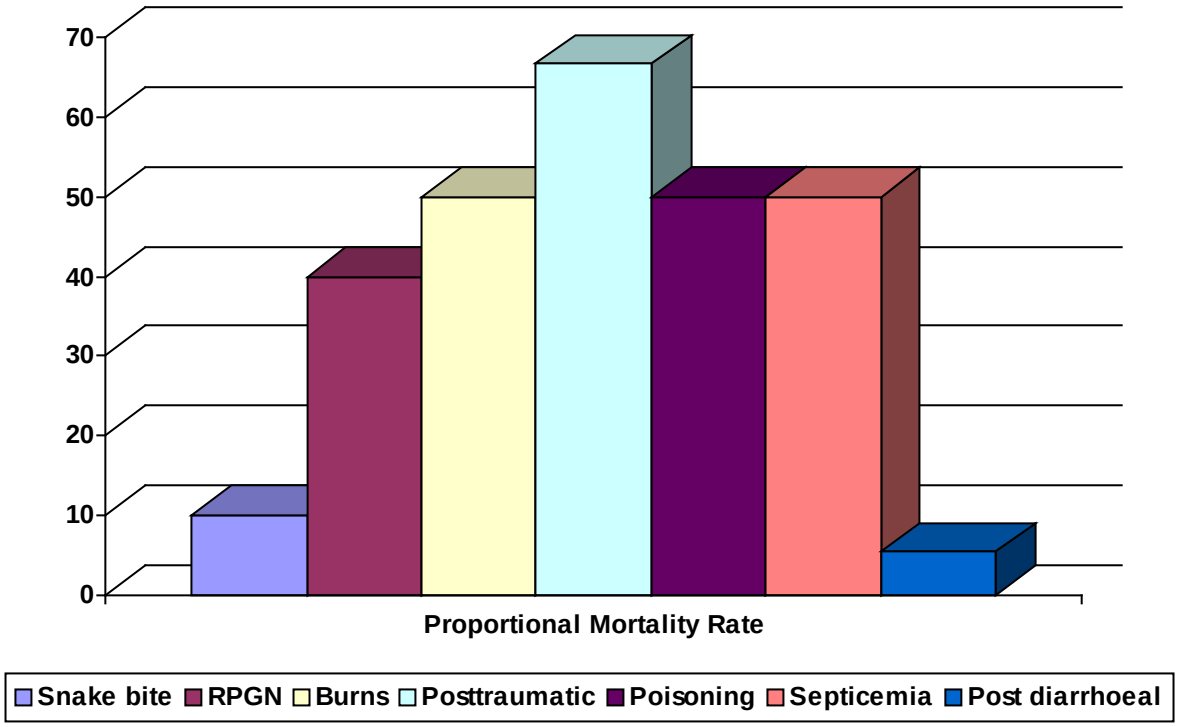
No. of Patients Studied	No. of Patients expired	Mortality rate
70	11	15.7%

Mortality according to aetiology is as follows:

S. No	Aetiology	No. of Patients Expired	Percentage in Total Mortality
1	Snake Bite	2	18.2%
2	RPGN	2	18.2%
3	Burns	2	18.2%
4	Posttraumatic	2	18.2%
5	Paraquat poisoning	1	9.1%
6	Septicemia	1	9.1%
7	Post diarrhoeal	1	9.1%

Proportional Mortality is as follows:

S. No	Aetiology	No. of Patients	No. of Patients Expired	Proportional Mortality Rate
1	Snake Bite	20	2	10%
2	RPGN	5	2	40%
3	Burns	4	2	50%
4	Posttraumatic	3	2	66.7%
5	Poisoning	2	1	50%
6	Septicemia	2	1	50%
7	Post diarrhoeal	19	1	5.3%


70
60
50
40
30
20
10
0
Proportional Mortality Rate
Snake bite
RPGN
Burns
Posttraumatic
Poisoning
Septicemia
Post diarrhoeal

Among the two cases of septicemia, one patient showed pre-renal pattern of renal failure and improved with conservative management and one showed ‘Intrinsic renal’ pattern of renal failure, and presented in ciritically-ill state, and was not in a state of undergoing peritoneal dialysis, and was managed conservatively and died next day.

Among the two cases of postrenal ARF, one woman, a known patient of ureteric stone [calculus disease] presented with anuria and elevated renal parameters, improved after peritoneal dialysis once. Likewise a old man who presented with anuria and increased renal paramenters, passed 300ml of urine after catheterization, improved well with peritoneal dialysis once. Later investigations showed prostatic hypertrophy. Both patients were transferred to urology department later.

ANALYSIS OF RESULTS AND DISCUSSION

The main aim of this study is to find out the incidence of 3 types of ARF, aetiology, prognostic factors, effectiveness of dialysis and outcome in acute renal failure patients admitted in Thanjavur medical college hospital during a specified period (August 2005 to December 2005).

As already discussed, hospital based studies of ARF reflect selection bias as these population are defined by the referral patterns to the site of care. Here in our study those cases that are admitted in nephrology ward directly or taken over by nephrology department from other medical wards were taken into account. On the other hand, those ARF cases in medical wards and surgical wards for whom nephrologist opinion not obtained either because they were treated by concerned unit physician or patients got discharged at their own request would not have come into account. Especially surgical and trauma ward cases the incidence would have been more than what we have observed. But because either of the death of the patients or discharge at request or some other reasons would not have come to our notice. Likewise ARF patients admitted in IMCU with multiorgan dysfunction and co- morbidity were not entered into this study. So exact incidence of ARF in cases admitted in Thanjavur medical college hospital could not be made out.

Incidence of various types and causes of renal failure:

Incidence of prerenal, renal and post renal types of ARF in our study is 50%, 47.1%, 2.9% respectively. But according to various studies conducted in Western countries [1,3] prerenal constitute 55-60%, renal 40-45% and post renal around 5%. But the slight change in incidence in our study may be due to increased incidence of 'intrinsic' ARF in this region. Otherwise, study by Kaufman J et al shows increased incidence of prerenal (70%) and study by Liano F et al shows low incidence of prerenal than our study [probably older age group has been taken in later study].

And one important observation in our study is some prerenal cases, even though they initially showed urinary indices of 'prerenal' pattern latter progressed to 'intrinsic' pattern [probably because of persistence of underlying pathology like ischemia, dehydration, etc.][1,2] At that stage, patients were symptomatically worse and they were instituted on dialysis.

Incidence of Medical, Surgical and Obstetric causes:

In our study, Medical causes account for 70%, Surgical causes 25.7%, and Obstetric causes 4.3%. According to Ramprabakar et al (88.1%) and Muthusethupathi et al (90%) studies medical causes slightly higher than our study. But Chugh et al study shows 64%, slightly lower than ours.

Comparative incidence of acute renal failure in various tropical countries is as follows[2]

Country	References	No. of cases	Medical (%)	Surgical (%)	Obstetric (%)
Singapore	Ku *et al* . (1975) [19]	143	60	16	24
Thailand	Sitprija and Benyajati (1975)[20]	162	61	15	24
India	Chugh *et al* . (1978)	325	67	22	11
India	Shah *et al* . (1985)	816	56	32	21
India	Muthusethupathi and Shivakumar (1987) [5]	187	85	9	6
India	Chugh *et al* . (1987)[8]	1862	60	25	15
Sri Lanka	Ramachandran (1994)[9]	317	79	15	6

Analysis of Aetiology:

Aetiology of ARF is varying compared to temperate countries. Common aetiologies in our study are snake bite (28.6%), diarrhoeal disorder (27.1%), whereas in the developed world extensive trauma, abdominal and vascular catastrophes and complicated open heart surgery are the leading causes.[2] But studies conducted in India show more or less similar aetiologies. For example, M. Ramprabakar et al,[22] shows post diarrhoeal, RPGN, Leptospirosis are leading causes. Chugh et al[24] shows chemicals, drugs, RPGN, Post-diarrhoeal are leading causes. Muthusethupathi et al[18] shows leptospirosis, ADD, Glomerulonephritis are the leading causes.

Comparison of our study with following studies:

1. Muthusethupathi et al Acute Renal Failure in South India[5]

Aetiology	No.	Dialysed	Mortality (%)
ATN (Ischemia)	49	31	14 (28)
GN	49	18	10 (20)
CuSo4 (Poison)	21	18	16 (76)
Obstetric	17	13	6 (35)
Post renal	11	9	4 (36)
Leptospirosis	10	6	2 (20)
Others	30	25	0
Total	187	120	52 (27.8)

(Others included snake bite – 6, Septicemia – 5, Papillary necrosis – 2, Malignant hypertension – 1, G6PD ↓ - 1)

2. Muthusethupathi M.A. et al Acute Renal Failure in Madras City – changing profile.[18]

Category	No	%
I Medical	**348**	**90**
Leptospirosis	120	31
ADD	118	30.5
GN	33	8.5
Sepsis	24	6.2
Drugs	21	5.4
Snake Bite	18	4.7
CuSo4	13	3.4

II Obstetric	**33**	**8.5**
Septic abortion	12	3.1
Eclampsia	8	2.1
Abruptio placentae	4	1.0
Puerperal sepsis	4	1.0
Post partum Renal failure	3	0.8
Post partum Haemorrhage	2	0.5
III Surgical	**6**	**1.5**
Vascular Surgery	2	0.5
Urological Surgery	2	0.5
GI Surgery	1	0.25
Orthopedic Surgery	1	0.25
Total	**387**	

Comparison of above study with our study is as follows:

	Muthusethupathi M.A. et al[18] (1993)	**Our Study**
Conservative Management	99 (25.6%)	35 (50%)
Dialysis	288 (74.4%)	35 (50%)
PD	195 (65.7%)	24
HD	93 (32.3%)	9
Mortality	104 (26.4%)	11 (15.7%)

3. M. Ram Prabakar, Edwin Fernando, R Venkatraman et al[22]

	M. Ram Prabakar et al[22]	Our Study
Mean age	33.58	38
Male	60.8%	54.3%
Female	39.2%	45.7%
Medical Causes	88.1%	70%
Surgical Causes	3.3%	25.7%
Obstetric Causes	8.6%	4.3%
Leading causes	Post Diarrhoeal (30.1%)	Snake Bite (28.6%) Post Diarrhoeal(27.1%)
RPGN	9.3%	7.1%
Leprosirosis	8.2%	1.5%
Snake Bite	8.5%	1.5%
$CuSo_4$ Poisoning	4.7%	1.5%
Overall Morality	19.8%	15.7%
Death due to medical Causes	17.9%	12.2%

4. Liano F, Pascual J et al[23]

	Liano F, Pascual J et al[23]	Our study
Mean Age	63 $\pm$ 17 yrs	38 yrs
Prerenal	21%	50%
Renal	45%	47.1%
Post renal	10%	2.9%
Acute on chronic	12.7%	-
Over all mortality	45%	15.7%
Dialysis required	36%	50%

5. Kaufman J, Dhakal M, Patel B et al[21]

	Kaufman et al[21]	Our Study
Total Patients	100	70
Prerenal	70%	50%
Renal	11%	47.1%
Postrenal	17%	2.9%
Mortality in prerenal	7%	14.2%
Mortality in Intrinsic renal	55%	18.2%
Prerenal-Leading Causes	Vomiting, ↓ fluid intake & Diarrhoea	ADD
Renal-Leading Cause	Drug induced & infection	Snake Bite

6. Chugh K.S., Sakhuja, V.,Malhotra, H.S., and Pereira, B.J.G. et al[24]

Aetiology	Chugh et al[24]	Our Study
RPGN	11%	7.1%
Sepsis	10%	2.9%
Transplant related	8%	-
Post diarrhoeal	5%	27.1%
Due to G6PD ↓	4%	-
Snake Bite & Insect sting	2%	30%
$CuSo_4$ Poisoning	0.3%	1.5%
Chemical & drugs	13%	-
Medical Causes	64%	70%

Among the various aetiologies in our study, snake bite (28.6%), ADD (27.1%), RPGN (7.1%) are the common medical causes. The factors appear to be responsible for such a high incidence of snake bite induced ARF are most of the patients admitted in TMCH are from rural areas where snake bite is common and important occupational hazard,[4] and people have inadequate knowledge about the management and available treatment in this tertiary centre and late presentation to hospital.

Another common medical cause in our study is ARF following ADD. The factors appear to be responsible for such a high incidence of ADD are poverty poor socioeconomic status and poor hygiene and the high incidence of ARF following ADD are due to scarcity of primary health care and late referrals from peripheral centers. But the mortality in these 19 cases is only one. This is notable because previous studies conducted in many places showed more mortality. This indicates improvement in care, and effectiveness of dialysis.

Next to ADD, RPGN is a common cause of ARF in our study, which constitutes 10.2%. The obstetric causes of acute renal failure in the present study are 4.3%. But studies conducted in many places show increased incidence (for example, M. Ramprabakar et al 8.6%, Muthusethupathy et al 8.5%, Chugh et al 15%). Septicemia, causing renal failure constitutes 2.9% relatively low compared to Chugh et al[24] [10%] and Muthusethupathi et al (6.2%).

Other aetiologies like CuSo4 poisoning, paraquat poisoning, leptospirosis, and kadhandu bite constitute small percentage [each 1 case] of causes of ARF, which are more or less similar to M. Ramprabakar et al and Chugh et al. But in Muthusethupathi et al leptospirosis is the leading cause (31%) of ARF, probably more incidence of leptospirosis in Chennai.

'Post-renal' causes of ARF constitute 2.9% in aetiology. Comparatively less than Muthusethupathi et al[5] (5.8%) and Liano F et al[23] (10%) may be due to age group is more (older age group) in later study.

Incidence according to age:

Incidence of ARF in younger to middle age group is more in our study as well as studies conducted in India[2] and other developing countries, where as ARF is more in older age group in developed countries.[25]

Analysis of morbidity and mortality:

Among the 70 patients studied, 11 patients expired (15.7%). In this 5 patients died of surgical aetiology.

Surgical cases (post- surgical, post-trauma, burns, post-renal cases and septicemia) constitutes notable percentage [25.7%] of causes of ARF in the present study. Their initial investigation reports showed 'Prerenal' pattern of involvement eventhough some patient's (especially who expired) subsequent investigation reports showed 'intrinsic renal' pattern of involvement.

This indicates that the underlying pathology is progressing and persistent ischemia led on to cause acute tubular necrosis.[1,2] So the morbidity and mortality in surgical causes could not be attributed to acute renal failure alone, instead it is multifactorial[1,2] and acute renal failure is caused and worsened by underlying problem. So the proportional mortality is higher [27.8%] when compared to medical causes (12.2%).

In remaining 6 patients death is due to medical causes. Mortality in M.Ramprabakar et al 19.8% and in Liano F et al[23] 45% and Muthusethupathi et al[18] (26.4%) also shows higher mortality. The relatively low mortality rate in our study (15.7%) indicates improvement in patients care and timely institution of dialysis.

Among the death due to medical causes, death due to snake bite is 2 cases, among the 2 cases one patient underwent PD once and HD Twice and stayed in hospital for 7-10 days inspite of intensive care he expired probably because of acute cortical necrosis[6] which can be proved only by biopsy. Another patient, 50 years old, presented 3-4 days after the development of ARF, and he was critically ill, and died in one day.

Mortality due to RPGN [proportional mortality 40%] could not be prevented eventhough both peritoneal dialysis and hemodialysis were instituted. It indicates the rapid progression of disease.

Death due to ADD occurred in old man, who was having loose tools and vomiting of 5 days duration, and treated at taluk hospital and when he was admitted he was critically ill, in hypotension, altered sensorium, was in 'Intrinsic renal' pattern of failure, and PD could not be instituted, and patient expired within 6 hours of hospital stay. Contributing factor for the death may be late referral.[26] In death due to paraquat poisoning, patient underwent both PD and HD. Inspite of this intensive care he could not be revived. It indicates the nephrotoxicity of the poison

Outcome in 3 obstetric patients is relatively good. 2 patients improved with dialysis and one patient improved with conservative management and so mortality is nil. But Muthusethupathi et al[5] shows significant mortality (36%).

Role of dialysis therapy:

Present study showed 50% of patients were managed conservatively 50% underwent dialysis [either PD or HD]. 'Intrinsic renal' failure patients received more dialysis than prerenal failure patients. Of the total 11 cases died, 6 cases didn't undergo any form of dialysis. This indicates the better prognosis in patients who underwent dialysis.

In Muthusethupathi et al[18] dialysis required in 74.4%. In Liano F, Pascual J et al, dialysis required in 36%. More institution of dialysis in our study, may be the reason for less mortality in our study compared to others.[23]

Analysis of prognostic factors:

Prognostic factors in this study were symptoms (weather oliguric or non-oliguric), initial creatinine value, age institution of dialysis and time of presentation to hospital. According to the present study most of the patients present with oliguria. Initial creatinine value, that is those having initial creatinine value of <3 mg%[1,48] (38 cases) [54.3%] have better prognosis and proportional mortality is less [7.9%] in them. Likewise institution of dialysis (both peritoneal as well as Hemodialysis) reduces the mortality, for example in Liano F et al,[23] dialysis given to 36% Patients and mortality was 45%. In our study, dialysis given to 50% and mortality is 15.7%.

According to studies mortality is increased with advancement of age. Here, in our study mortality and age goes as follows: Mortality age wise [15-20yrs-1, 21-30yrs-3, 31-40yrs-3, 41-50yrs-4, 51-60-nil] shows more incidence in middle age group. Mortality is not only determined by age but also by underlying aetiology. That is death due to RPGN occurred in young whereas death due to ADD occurred in old age, and death due to septicemia/renal failure also occurred in old age. Snake bite/renal failure death occurred both in young and old age.

Death due to poisoning occurred in middle aged man. So according to this study causes of death in young are: RPGN, snake bite and poisoning and causes in old age are ADD, septicemia and snake bite. It indicates that cause of mortality varies in different age group and depends on aetiology. Time of presentation to hospital, after the development of ARF, also determines the out come[26]. In our study, 2 patients (1 case of snake bite presented 3 to 4 days after the development of symptoms, and 1 case of ADD who presented 5 days after the development of symptoms of renal failure) could not be revived because of late presentation to our centre.

CONCLUSION

1. Snake bite and acute diarrhoeal disorders are the common aetiologies of acute renal failure in cases admitted in Thanjavur medical college hospital. RPGN comes the next common cause of ARF.

2. According to the type of ARF, incidence of prerenal is 50%, renal 47.1%, post renal 2.9%, more or less similar to studies conducted in other places.

3. In this study, incidence of medical causes of ARF is 70%, surgical causes 25.7%, obstetric causes 4.3%.

4. Dialysis is required in 50% cases.

5. Requirement of dialysis treatment is mostly for intrinsic renal failure patient.

6. Peritoneal and hemodialysis play an important role in improving the prognosis.

7. Proportional mortality is more in RPGN (40%), among medical causes.

8. Eventhough both snake bite and acute diarrhoeal disorder are equally common causes, morbidity is more in snake bite and cost of treatment also more in snake bite as many of them require dialysis.

9. Prognosis is determined by age, aetiology of renal failure, initial creatinine value, time of presentation to the hospital and comorbidity.

10. As morbidity and mortality due to snake bite induced renal failure is high, people must be made aware of this rural occupational hazard, and they should be taught about the preventive aspects. And as acute diarrhoeal disorder is another common cause of ARF people should be given health education and to be taught the importance of fluid replacement therapy and early referral to hospital, which may reduce the incidence of ARF.

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PROFORMA

CLINICAL STUDY OF ACUTE RENAL FAILURE

Name: Age / Sex: Ward: I.P. No.

I AETIOLOGY:

II HISTORY:

Oliguria	Anuria	Heamaturia

III CLINICAL EXAMINATION:

1. Pulse Rate

Normal	**Bradycardia**	Tachycardia

Volume

Normal	**Small**	Large

2. Blood Pressure

Normal	**Hypertension**	Hypotension

3. Respiratory Rate

Normal	**Tachypnoea**

4. Temperature:

5. Pallor

Present	**Absent**

6. Jaundice

Present	**Absent**

7. Hydration

Normal	**Dehydrated**

8. Others

IV PAST HISTORY:

Hypertension		Diabetes Mellitus		Previous Renal Diseases	

V INVESTIGATIONS:

A. BLOOD

1. Urea (mg/dl)

< 45	> 45

2. Creatinine (mg/dl)

< 1.2	> 1.2

3. Sugar (Random) (mg/dl)

4. Serum Electrolytes (meq/l) Na^+ K^+

5. Total Count (Per cu. mm)

6. Differential Count (%) P L E M B

7. Hemoglobin (gm/dl)

B. URINALYSIS

	Nil	+	++	+++
1. Albumin				
2. Sugar				

	Nil	+	++	+++
3. Deposits				

Others

4.	Urine Sodium (meq/l)		
5.	Urine Creatinine / Plasma creatinine		

< 10	> 20
> 40	< 20

6.	Fractional excretion of Na^+ [FENa] (%)		
7.	Renal Failure index (UNa^+/[Ucr / Pcr]) (%)		
9.	**Plasma Urea / Creatinine**		

< 1	> 1
< 1	> 1
> 20	< 20

10.

Prerenal	Renal	Postrenal

C. OTHERS

D. ULTRASONOGRAM – ABDOMEN

Kidney size		
< 7 cm	7 – 11 cm	> 11 cm

VI TREATMENT GIVEN

CONSERVATIVE	PERITONEAL DIALYSIS	HEMODIALYSIS

VII OUTCOME

RECOVERED	SUCCUMBED

MASTER CHART

S. No.	Name	Age (yrs)	Sex	I.P. No.	Aetiology	Urine Output	PR	BP (mmHg)	Others	Past History of			Blood Biochemistry				
										HT	DM	RD	Urea	Creatinine	Sugar (R) mg%	Na^+ meq/L	K^+ meq/L
1	Suresh	21	M	855318	Snake Bite	O	82	130/80	-	Nil	Nil	Nil	119	5.2	124	133	3.5
2	Mallika	40	F	855516	Snake Bite	O	98	160/110	-	Nil	Nil	Nil	178	5.8	96	128	3.7
3	Selvi	32	F	855972	Burns	O	98	100/60	-	Nil	Nil	Nil	90	2.1	110	130	4.6
4	Muthu	57	M	989907 (ID)	Post Diarrhoeal	O	90	110/70	-	Nil	Nil	Nil	106	2.4	100	127	3.2
5	Shakul Hameed	37	M	856098	Snake Bite	O	80	130/90	-	Nil	Nil	Nil	64	2.6	70	127	4.8
6	Rangammal	44	F	856283	Post Diarrhoeal	O	88	100/70	-	Nil	Nil	Nil	82	2.4	102	129	3.8
7	Anjalai	52	F	856290	Post Surgical	O	90	100/70	-	Nil	Nil	Nil	52	1.5	78	130	3.4
8	Alagarsamy	40	M	856837	Kadhandu Bite	O	92	130/80	-	Nil	Nil	Nil	96	3.4	92	130	5.2
9	Pakirisamy	50	M	992286 (ID)	Post Diarrhoeal	O	98	100/70	-	Nil	Nil	Nil	98	2.6	98	130	4.0
10	Karuppayi	40	F	858162	Snake Bite	O	90	170/100	-	Nil	Nil	Nil	224	9.9	128	127	5.6
11	Krishnamurthy	36	M	858912	Cuso4 Poisoning	O	98	130/90	-	Nil	Nil	Nil	108	5.4	98	128	4.9
12	Veeraiyan	50	M	859124	Post Surgical	O	92	110/70	-	Nil	Nil	Nil	80	1.4	98	130	4.6
13	Arumugam	45	M	859498	Post Diarrhoeal	O	94	106/76	-	Nil	Nil	Nil	78	2.2	96	126	3.6
14	Selvam	24	M	859553	RPGN	O/H	96	130/90	-	Nil	Nil	Nil	174	3.6	76	118	5.0
15	Alexander	40	M	859734	Snake Bite	O/H	96	140/100	-	Nil	Nil	Nil	166	8.4	117	127	5.3
16	Baskar	33	M	859844	Post Surgical	O	96	96/70	-	Nil	Nil	Nil	84	3.0	101	129	4.6
17	Nagammal	45	F	994172 (ID)	Post Diarrhoeal	O	94	110/80	-	Nil	Nil	Nil	72	1.8	88	127	3.8
18	Ponnammal	45	F	860171	Post Surgical	O	94	110/80	-	Nil	Nil	Nil	98	2.6	98	129	3.8
19	Anand	40	M	860178	Paraquat Poisoning	O	98	100/70	-	Nil	Nil	Nil	285	18.4	118	127	3.5

MASTER CHART

20	Dhanam	16	F	860343	Snake Bite	A	94	130/90	-	Nil	Nil	Nil	102	5.3	67	110	5.4
21	Marimuthu	40	M	860385	Snake Bite	A	92	130/90	-	Nil	Nil	Nil	263	4.6	63	136	4.7
22	Veerapathran	40	M	860662	Snake Bite	O	110	140/110	-	Nil	Nil	Nil	141	6.1	116	136	3.3
23	Santhanakrishnan	40	M	994417 (ID)	Post Diarrhoeal	O	98	110/70	-	Nil	Nil	Nil	66	1.6	114	125	3.4
24	Shanthi	32	F	860856	Calculus Disease	A	96	150/90	-	Nil	Nil	Yes	136	4.2	108	135	3.2
25	Chelladurai	42	M	861163	Post Diarrhoeal	O	90	100/70	-	Nil	Nil	Nil	87	2.2	111	129	4.8
26	Mani	45	M	861241	Snake Bite	O	92	110/70	-	Nil	Nil	Nil	49	1.6	88	135	3.6
27	Jothy	20	F	861244	Obstetric	A	88	150/80	-	Nil	Nil	Nil	126	5.2	106	136	5.2
28	Mahalingam	50	M	861362	Post Diarrhoeal	A	94	80/60	-	Nil	Nil	Nil	98	3.8	86	126	3.2
29	Samyapan	36	M	861395	Post Surgical	O	92	100/70	-	Nil	Nil	Nil	73	1.7	100	132	4.8
30	Kaliyaperumal	55	M	861659	Leptospirosis	No oli	86	120/80	Icteric	Nil	Nil	Nil	127	7.4	86	133	2.7
31	Sagayaraj	48	M	862055	Post traumatic	O	98	96/70	-	Nil	Nil	Nil	141	4.3	127	128	3.4
32	Amsavalli	35	F	862390	Post Surgical	O	86	110/80	-	Nil	Nil	Nil	116	2.8	90	131	3.6
33	Upagaram	55	M	862400	Snake Bite	A	94	100/70	-	Nil	Nil	Nil	180	8.9	90	130	6.2
34	Sumathy	38	F	1000082 (ID)	Post Diarrhoeal	O	98	110/70	-	Nil	Nil	Nil	78	1.8	116	128	3.6

MASTER CHART

35	Thilagavathy	43	F	863158	Post Diarrhoeal	O	94	110/80	-	Nil	Nil	Nil	35	1.2	82	131	4.8
36	Gajendran	18	M	863393	RPGN	O/H	86	170/100	-	Nil	Nil	Nil	41	1.1	83	141	4.9
37	Vijaylakshmi	28	F	863426	Burns	O	92	100/70	-	Nil	Nil	Nil	53	2.2	104	130	4.3
38	Lakshmi	38	F	1000821 (ID)	Post Diarrhoeal	O	90	116/80	-	Nil	Nil	Nil	76	2.0	102	124	3.2
39	Pattammal	50	F	863444	Septicemia	O	110	90/60	-	Nil	Nil	Nil	72	4.6	112	130	4.8
40	Arulpandiyan	15	M	863611	RPGN	O/H	98	140/80	subcon-junctival Hemorrhage (+)	Nil	Nil	Nil	100	3.3	73	119	3.5
41	Johnpeter	18	M	863629	Snake Bite	O/H	96	170/100	-	Nil	Nil	Nil	104	3.2	120	130	4.9
42	Nagarathinam	47	F	864098	Snake Bite	O/H	86	130/80	-	Nil	Nil	Nil	185	6.6	87	130	4.4
43	Chinnadurai	27	M	864113	Post Diarrhoeal	O	92	100/70	-	Nil	Nil	Nil	45	1.5	75	127	3.8
44	Ravi	36	M	864437	Post traumatic	O	94	90/60	-	Nil	Nil	Nil	124	3.5	101	132	4.7
45	Saravanan	26	M	864591	Snake Bite	A	96	130/90	-	Nil	Nil	Nil	90	3.0	88	133	5.0
46	Venkatachalam	52	M	864826	Post Surgical	O	98	100/80	-	Nil	Nil	Nil	69	1.6	108	132	4.4
47	Sudha	23	F	864846	Burns	O	90	110/70	-	Nil	Nil	Nil	66	2.3	92	137	4.0
48	Velmurugan	23	M	1003254 (ID)	Post Diarrhoeal	O	92	110/86	-	Nil	Nil	Nil	115	2.8	104	126	3.6
49	Muthulakshmi	35	F	865131	Post Diarrhoeal	O	90	110/80	-	Nil	Nil	Nil	40	1.3	68	133	4.1
50	Gomathi	30	F	865168	Post Diarrhoeal	O	94	100/60	-	Nil	Nil	Nil	29	0.7	75	136	3.7
51	Murugesan	23	M	865279	Septicemia	No oliguria	90	106/66	-	Nil	Nil	Nil	156	7.2	72	125	5.2
52	Valarmathy	24	F	865401	RPGN	A	86	170/110	-	Nil	Nil	Nil	102	5.0	108	129	4.8
53	Gandhi	32	M	865684	Snake Bite	No oliguria	88	150/90	-	Nil	Nil	Nil	58	2.0	76	126	4.7

MASTER CHART

54	Ramaiah	60	M	865839	Obstructive Nephropathy	A	86	150/100	-	Nil	Nil	Yes	164	9.6	107	110	5.2
55	Shanmugam	35	M	865941	Snake Bite	O	82	150/90	-	Nil	Nil	Nil	99	5.6	114	132	4.8
56	Radhika	17	F	866364	Snake Bite	A	88	130/80	-	Nil	Nil	Nil	84	1.8	67	127	3.6
57	Ramalingam	45	M	866413	Post Diarrhoeal	O	88	100/86	-	Nil	Nil	Nil	113	2.4	125	132	4.8
58	Subramani	50	M	866738	Snake Bite	O	86	116/70	-	Nil	Nil	Nil	196	9.5	70	133	5.6
59	Suganthi	30	F	866740	Burns	O	106	106/76	-	Nil	Nil	Nil	88	2.3	94	130	4.2
60	Thavamani	50	F	866995	Post Diarrhoeal	O	86	110/70	-	Nil	Nil	Nil	60	1.1	104	131	4.7
61	Swaranalatha	17	F	867021	RPGN	O/H	96	180/100	-	Nil	Nil	Nil	130	11.1	74	130	4.2
62	Rakkammal	35	F	867029	Snake Bite	O	92	130/90	-	Nil	Nil	Nil	74	3.2	111	126	4.8
63	Shanthi	28	F	867468	Obstetric	O	112	100/70	-	Nil	Nil	Nil	75	3.8	91	132	4.9
64	Palaniyammal	45	F	868735	Post traumatic	O	86	120/76	-	Nil	Nil	Nil	141	4.3	80	128	3.4
65	Gowri	65	F	868946	Snake Bite	O	92	90/60	-	Nil	Nil	Nil	45	2.0	120	131	4.6
66	Valliyammal	58	F	1006751 (ID)	Post Diarrhoeal	O	92	106/70	-	Nil	Nil	Nil	58	1.5	110	127	3.4
67	Ravichandran	45	M	869031	Post Surgical	No oli	88	110/80	-	Nil	Nil	Nil	106	2.8	88	120	2.5
68	Duraikannan	60	M	869421	Snake Bite	O/H	96	140/96	-	Nil	Nil	Nil	252	6.4	63	131	5.4
69	Anjalaiammal	40	F	869607	Post Diarrhoeal	O	90	106/70	-	Nil	Nil	Nil	52	1.5	78	130	3.4
70	Firudose	30	F	869713	Obstetric	O	92	150/100	-	Nil	Nil	Nil	134	4.1	86	135	3.8

O - Oliguria Po. - Post renal Rec. - Recovered RPGN- Rapidly progressiv

MASTER CHART

H	-	Haematuria	N	-	Normal size kidneys (I.e; between 7-11cm size) ultrasonagram wise	Exp.	-	Expired	No oli - No oliguria
A	-	Anuria	ND	-	USG abdomen could not be done	Pus	}	Urine deposits like PUS cells Epithelial cells RBC	
R	-	(instrinsic) Renal	PD	-	Peritoneal Dialysis	Epi			
Pr.	-	Prerenal	HD	-	Hemodialysis	RBC			

MASTER CHART

Hb gm%
10.8
10.6
10.6
11.6
11.8
10.2
9.6
10.8
11.4
9.2
10.2
11.6
11.8
9.2
10.8
11.2
11
9.6
10.6

MASTER CHART

10.6
9.2
10.4
12
9.2
12
11.6
6.8
9.6
10.6
11.2
9.8
10
12.6
9.2

MASTER CHART

10.2
11.2
9.2
10.6
9.8
9.8
10.6
10.4
11.4
10.4
10.8
9.6
9.6
11.2
9.8
9.8
12.4
9.8
12.2

MASTER CHART

11
12.4
11
11.2
9.4
9.4
9.6
11.6
11.4
9.2
8.6
9.2
9.2
10.2
11.6
8.6
8.6

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MASTER CHART

MASTER CHART

	Urine			Uc/Pc	$FENa^+$ (%)	RFI (%)	Plasma U/C	Result	Others	USG Abd	Treatment given	Outcome
	Albumin	Deposits per HPF	Na^+ (meq/L)									
1	Nil	Epi. 6-8 RBC 2-4	58	18.5	2.4	3.1	22.9	R	-	N	PD - once HD - Twice	Rec.
2	+	Pus :10-20 RBC - 6-8 Epi - 8-10	42	13.1	2.5	3.2	30.7	R	-	N	PD - once	Rec.
3	+	Pus 2-4 Epi 4-6	26	41.9	0.5	0.6	42.9	Pr.	-	ND	C	Exp.
4	+	Pus 1-2 Epi 2-4	22	35.8	0.5	0.6	44.2	Pr.	-	N	C	Rec.
5	+	Pus 5-7 Epi 4-6	28	18.5	1.2	1.5	24.6	R	-	N	C	Rec.
6	Nil	Pus 2-4 Epi 2-4	28	44.2	0.5	0.6	34.2	Pr.	-	N	PD - once	Rec.
7	+	Epi 2-4	34	85.3	0.3	0.4	34.7	Pr.	-	N	C	Rec.
8	+	Epi 6-8 Pus 1-2 RBC 2-4	46	9.4	3.8	4.9	28.2	R	-	N	PD - Twice	Rec.
9	Nil	RBC - 2-4 Epi 4-6	24	24.6	0.8	1.0	37.7	Pr.	-	N	C	Rec.
10	Nil	Epi: 8 - 10 RBC 4-6	56	7.3	6.1	7.7	22.6	R	-	N	PD - Twice	Rec.
11	+	Epi: 6-8 RBC 2-4	34	6.3	4.2	5.4	20	R	-	N	PD - once HD - Thrice	Rec.
12	Nil	Epi: 1-2	32	74.3	0.3	0.4	57.1	Pr.	-	N	C	Rec.
13	Nil	Epi: 1-2	34	50	0.5	0.8	35.5	Pr.	-	N	PD - once	Rec.
14	+	RBC - 4-6 Epi 8-10 Pus: 6-8	39	8.8	3.7	4.4	48	R	-	N	PD - once	Rec.
15	+	Epi 12-16 RBC 6-8	59	6.6	7	8.0	23.2	R	-	N	C	Rec.
16	Nil	Epi 2-4	34	76.7	0.3	0.4	28	Pr.	-	N	C	Rec.
17	Nil	Epi 2-4 Pus 4-6	18	52.2	0.2	0.3	40	Pr.	-	N	C	Rec.
18	Nil	Pus 2-4 Epi 4-6	22	30	0.6	0.7	37.7	Pr.	-	ND	C	Rec.
19	+	Epi 8-10 Pus 4-6	35	2.6	10.6	13.4	15.5	R	-	N	PD - once HD - Thrice	Exp.

MASTER CHART

20	+	Epi 8-10 RBC 4-6 Pus: 6-8	38	2.1	16.3	17.9	19.2	R	-	N	PD - once HD - four	Rec.
21	+	EPI: 4-6 Pus: 2-4	47	2.5	13.9	18.9	57.2	R	-	N	PD - Twice	Rec.
22	+	Epi: 6-8 Pus: 2-4	60	3.3	13.5	18.3	23.1	R	-	N	PD - Twice HD - Twice	Rec.
23	Nil	Epi 2-4	16	63.7	0.2	0.3	41.3	Pr.	-	N	C	Rec.
24	+	Epi 6-8	48	14.8	2.4	3.3	32.4	Po	-	N	PD - once	Rec.
25	+	Epi 6-8 Pus 2-5	18	50	0.3	0.4	39.6	Pr.	-	N	PD - once	Rec.
26	Nil	Epi 6-8 Pus 2-4	78	38.8	1.5	2	30.1	R	-	N	C	Rec.
27	+	Pus:2-4 Epi: 4-8 RBC 2- 4	52	7.6	4.9	6.7	24.2	R	-	N	HD - Thrice	Rec.
28	+	Epi: 6-8 RBC 2- 4 Pus:4-6	42	14.2	2.4	3	25.8	R	-	N	C	Exp.
29	Nil	Epi 2-4	28	47.1	0.5	0.6	42.9	Pr.	-	ND	C	Rec.
30	+	Pus: 3-4 Epi 2-3 RBC: 1-2 BS +ve BP +ve urobilinogen 0.7	52	4.9	8	10.7	17.2	R	Sr. Bili: 28.1 (Total) Direct : 16.4 Indirect: 11.7 SGOT: 46 SGPT: 52 Alk Phos: 181 MAT (for Leptospira): +ve	N	PD - once	Rec.
31	Nil	Epi 2-4	28	39.5	0.6	0.7	32.8	Pr.	-	ND	C	Exp.
32	Nil	Epi 4-6 Pus 2-4	28	29.3	0.7	0.9	41.4	Pr.	-	ND	C	Rec.
33	+	Epi 8-12 RBC 6-8 Pus 2-4	60	6.4	7.3	9.5	20.2	R	-	N	PD - once	Exp.
34	Nil	Pus 2-3	18	61.1	0.2	0.3	43.3	Pr.	-	N	C	Rec.
35	Nil	Pus 2-4	60	83.3	0.5	0.7	29.2	Pr.	-	N	PD - once	Rec.
36	+	Plenty of Pus cells + RBC / HPF	54	65.5	1.8	2.5	37.3	R	-	N	C	Rec.

MASTER CHART

37	+	Epi 4-6 RBC 2-4	24	42.7	0.4	0.6	24.1	Pr.	-	ND	C	Exp.
38	Nil	Epi 1-2	20	41	0.4	0.5	38	Pr.	-	N	C	Rec.
39	+	Epi 8-12 Pus 4-6	162	5.4	21.5	29.8	15.7	R	-	N	C	Exp.
40	+	RBC 6-10 Epi 8-12 Pus 2-4	22	16.9	1.1	1.3	30.3	R	-	N	PD - once	Rec.
41	+	Epi 6-10 RBC 4-6 Pus 2-4	40	3.0	10.1	13.4	19.4	R	-	N	PD - once	Rec.
42	+	Epi 4-6 RBC 2-4	33	12.1	2.1	2.7	28	R	-	N	PD - once	Rec.
43	Nil	Epi 2-4	25	92	0.2	0.3	30	Pr.	-	N	PD - once	Rec.
44	+	Epi 4-6 RBC 2-4	35	36	0.7	0.9	35.4	Pr.	-	ND	C	Exp.
45	+	Epi 5-10 RBC 4-6	40	11.3	2.7	3.5	30	R	-	N	PD - once HD - Twice	Exp.
46	Nil	Epi 2-4 Pus 1-2	30	60	0.4	0.5	43.1	Pr.	-	ND	C	Rec.
47	+	Pus 2-4 Epi 4-6	41	35.6	0.8	1.2	28.6	Pr.	-	ND	C	Rec.
48	Nil	Epi 2-4	28	27.9	0.8	1.0	46	Pr.	-	N	C	Rec.
49	Nil	Pus 4-6	35	87.7	0.3	0.4	30.7	Pr.	-	N	PD - once	Rec.
50	+	Pus 4-6 Epi 4-8	96	77.1	0.9	1.2	41.4	Pr.	-	N	C	Rec.
51	Nil	Epi 2-4 RBC 2-4	35	37.1	0.8	1.0	21.7	Pr.	-	N	C	Rec.
52	+	Pus 2-8 RBC 1-2 Epi 1-2	32	7	3.5	4.6	20.4	R	-	N	PD - Twice	Exp.
53	+	Epi 4-6	32	18	1.4	1.8	29	R	-	N	C	Rec.
54	Nil	Epi 4-6 Pus 2-4	16	6.1	2.4	2.7	17.1	Po	-	N	PD - once	Rec.
55	+	RBC 4-6 Epi 6-8	54	19.6	2.1	2.8	17.7	R	-	N	PD - once	Rec.
56	+	RBC 2-4 Epi 5-10 Pus 4-6	35	16.7	1.6	2.1	46.7	R	-	N	C	Rec.
57	Nil	Epi 2-4	50	72.9	0.5	0.7	47.1	Pr.	-	N	PD - once HD - once	Rec.

MASTER CHART

58	+	Pus 4-6 Epi 8-10	33	1.6	15.7	20.9	20.6	R	-	N	PD - Twice HD - Twice	Rec.
59	+	Epi 4-6 RBC 2-4	26	36.9	0.5	0.7	38.3	Pr.	-	ND	C	Rec.
60	Nil	Pus 1-2 Epi 2-4	110	120.9	0.6	0.7	54.5	Pr.	-	N	PD - once	Rec.
61	+	RBC 8-10 Epi 5-10 Pus 4-6	140	8.6	12.5	16.2	11.7	R	-	N	PD - four HD - once	Exp.
62	+	Epi: 6-8 RBC: 2-4 Pus 2-4	96	70.9	1.1	1.4	23.1	R	-	N	C	Rec.
63	+	Epi 8-12 RBC 4-6 Pus 5-10	110	19.7	4.2	5.6	19.7	R	-	N	HD - four	Rec.
64	Nil	Pus 4-8 Epi 3-5	28	39.5	0.6	0.7	32.8	Pr.	-	N	C	Rec.
65	+	Epi 8-10 RBC 2-4 Pus 4-6	105	32.5	2.5	3.2	22.5	R	-	N	PD - once	Rec.
66	+	Pus 2-4 Epi 1-2	25	52	0.4	0.5	38.7	Pr.	-	N	C	Rec.
67	Nil	Epi 2-4	48	63.6	0.6	0.8	37.9	Pr.	-	ND	C	Rec.
68	+	Epi 6-10 RBC 5-10 Pus 2-4	68	5.6	9.2	12.1	39.4	R	-	N	PD - Twice	Rec.
69	Nil	Pus 2-4	34	85.3	0.3	0.4	34.7	Pr.	-	N	PD - Once	Rec.
70	+	Epi 5-10 Pus 2-4 RBC 4-6	82	16.1	3.9	5.1	32.7	R	Sr. Bili: 1.2 SGOT: 12 SGPT: 28 Alk Phos: 84 prot: 5.3	N	C	Rec.