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A Survey on Etiopathogenesis of Acute Renal Failure

Archana Meshram

Department of Zoology, Dr. Ambedkar College, Deekshabhoomi, Nagpur
Email: drarchie4517@gmail.com

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ABSTRACT

ARF is the most serious and challenging clinical problem in human being, which requires prompt recognition, evaluation, treatment and management. It is a syndrome characterized by rapid decline in GFR and retention of nitrogenous waste products such as BUN and creatinine. ARF includes glomerular nephritis and interstitial or ATN clinically, the process is complex and poorly understood pathophysiological event, which occurs in response to a series of insults. Renal hypoperfusion and certain nephrotoxins, such as radiocontrast media, cause intra- renal vasoconstriction which may lead to parenchymal ischemia and the development of acute tubular necrosis. GFR falls due to vasoconstriction.

Keywords: Acute Renal Failure, Dialysis, Glomerular nephritis, erythropoiesis

INTRODUCTION

Renal failure is a condition where the kidneys loose their ability to function a normal way. This may be due to various factors including infections, auto-immune disease, diabetes and other endocrine disorders, cancer and toxic chemicals. The vasoconstriction causes a decrease in the rate of glomerular filtration. Tubular obstruction as a result of parenchymal edema, desquamated epithelial cells and casts and precipitation of salts such as oxalates occur back leak of glomerular filtrate through abnormally permeable tubular epithelia can then result. The lungs are responsible for maintaining the composition of extra cellular fluid with respect to O_2 and CO_2 in all organisms, the duty for maintaining the composition of this fluid in respect to other constituents develops on the kidneys, and they are the master chemists of our body. Kidney is unique among the organs of the body in its ability to undergo virtually complete recovery of structure and function after damage,

The composition of the body fluid is determined on the retention of the kidneys of the ingested food material it is worth to mention that the bones, muscles, glands and even our brains perform only one kind of the allotted physiological function, but our kidneys play a vital role in performing an innumerable variety of operations such as maintenance of

normal BP., erythropoiesis, activation of vitamin D and other endocrine functions. Erythropoiesis is regulated by the erythropoietin production by renal tubular, peritubular or mesangial cells. Vit. D is important for calcium and phosphorus metabolism. All these functions are deranged in kidney failure.

The severity of the problem

ARF occurs in about 30% of critically ill patients. Loss of renal function in such patients leads to increased morbidity. Increased length of stay and consequently increased costs, with a mortality rate of about 65%. The causes of ARF commonly include post-operative hypovolaemia, congestive cardiac failure, radiocontrast media-induced injury and amino glycoside therapy. It has been estimated the 20% of episodes of ARF are drug-induced and 55% are potentially avoidable episodes as a result of fluid or drug mismanagement. (Hou *et al.* 1983). The mortality on patients who develop renal failure through hypoperfusion is much higher than, for example, in patients who recover an overdose of amino glycosides. Mortality is higher in patients who develop ARF in hospital. (Shusterman *et al.* 1987).

Which patients are likely to get ARF?

1) Poor renal perfusion pressure (sodium depletion, diuretic therapy, hypovolaemia, low cardiac (indino
2) Pre-existing renal dysfunction
3) Diabetes mellitus
4) Sepsis
5) Vascular disease
6) Liver disease

Patients with pre-existing poor renal function are more likely to develop ARF since, the solute load to individual nephrons is higher due to nephron loss. Oxygen consumption increases with the increase in nephron work and there is often damage to juxtamedullary glomeruli, such that functional reserve is severely depleted, thereby making the patient less able to compensate for drug induced effects. Impaired drug elimination may also result in toxic drug levels with consequent positive feedback on the nephrotoxic process. Patients with chronic hepatic disease are likely to metabolize drugs abnormally within the liver. These patients often have altered intrarenal hemodynamics with pronounced salt retention which potentiates any nephrotoxicity. Diabetic patients are especially at risk of ARF.

ARF: Review of earlier work

The current concept of ARF was better understood during the bombing of World War II in London. This is evident from the observations of Bywaters and Bealk, (Bywaters and Bealk D, 1941) on erush injury cases and the subsequent description of similar abnormal-lities by Swam and Mernil (Burton, 1994) these were based on diverse etiological factors like mismatch blood transfusion, abortion, cardio-vascular collapse, sepsis and nephrotoxic substances. Acute Renal Failure has been known for many years but it was rediscovered later. It was described in the first world war casualties in the German literature. (Cameron, 1986) The world war and II has been responsible for its rediscovery. Thadani *et al.* 1996 in their review article have highlighted the epidemiology, general causes and evaluation of ARF in adults. Expanding on the pathophysiology of ischemic ARF. They have discussed in detail the rationale for both current and future therapies and considered replacement therapies in the light of recent studies. Galley (2000) suggested the correction of salt and volume depletion to be paramount in the prevention of renal damage. They suggested that measures which stimulate intense filtration of glomeruli in ARF, such as the use of arterial natriuretic peptide analogs, thiophyline, dopamine, or growth factors should be regarded with caution, since these all increase metabolic workload in the outer medulla and hence aggravate medullary hypoxia. Corinne Bagnis, *et al.* (2001) suggested that Erythropoietin is a growth factor whose synthesis mainly takes place in the kidney. Erythropoietin has been shown to support the growth not only of erythr-oid progenitor cells but also of certain other cell types. They had attempted to establish whether erythropoie-tin enhances the recover from ARF induced by cisplatin. Usha *et al.* (2002) investigated 82 cases of pregnancy related ARF (PR- ARF) who were admitted in the Dept. of Nephrology, Osmania General Hospital, Hyderabad These studies showed that the overall incidence of ARF had decreased from 20.3% to 12.2. The epidemiology and pathophysiology of ARF has been well discussed in a review article by Schrier *et al.* (2004) It includes useful information on the vascular tubular and inflammatory perturbations and elabora-tely describes the clinical evaluation of the ARF and its implications for potential future therapies to decrease the high mortality rate. Lobo *et al.* (2004) have carried out an investigation on the mortality of critically ill patients who developed ARF in the 1CCU setting. They found that the mortality is extremely high (50-80%).

According to them any mode of renal replacement therapy chosen should be able to achieve solute and water clearance while maintaing hemodynamic stability, have positive effect on nutrition, and have low complication rates. The clinical investigations by Gill *et al.* (2005) in critical care units accounted for about 7.6% of ARF cases and indicated that despite of the introduction of hemodialysis > 30 yrs ago, the mortality rates from ATN in hospitalized and ICCU patients are about 37.1% and 78.6% respectively. The present prospective investigation was carried out in Central India at Amravati Division from Dec. 2003 to May 2007. The study was commenced after obtaining the informed written consent from the patients who were enrolled in the study. All the enrolled patients underwent a detailed history and various biochemical, hematological, physical examinations. The study included the patients suffering from ARF with various etiological factors. A total of 39 patients were studied out of which 27 were male and 12 were female. The patients whose complete records were lacking were excluded from this study. The comprehensive data so obtained included clinical presentation; biochemical parameters, hemograms, radiological investigations, the predisposing conditions, risk factors and complications with its outcome were studied. Treatment modalities such as conservative management and dialysis (hemodialysis).

Investigations of the renal diseases

The investigations included the following

Presenting symptoms, Physical examinations, Hematological examinations, Biochemical examinations, Urine examinations, MP, Widal, HbsAg. Oligouria, anuria, hematuria, dysuria, are again the prominent symtptoms in ARF. Dry history like NSAID canprecipitate ARF, quinolones, aminoglycosides, indigenous drugs, antibiotics, rifampicin are the most common agent to produce ARF. Preaclampsia, PLH, eclamsia are the most evident causes of ARF.Detailed physical examinations included Pulse rate, B.P, temperature, skin, edema, peripheral pulsation, scratch mark, purpura, pallor, hair loss, butterfly rash on face, pigmentation on skin, hepatosplenomegaly, chest examination, joint examination, neurological and ophthalmic examination. Hematological examinations included CBC like Hb, TLC, Platelets. All these were estimated with the use of Beckman's Coulter. Biochemical examinations like Serum creatinine, sodium, and potassium, BUN. were estimated with the help of MERCK and AVL 9180 Electrolyte Analyzer. Urine examinations were carried out to detect the proteins, Pus cells and hematuria.

In recent years the investigations on etiopathogenesis of ARE have attracted wide attention from the researchers all over the world, particularly in medical sciences. This is primarily because ARF is a syndrome not a disease, as it has numerous causes and occurs in a wide range of patients. This makes the evaluation of prognosis a daunting task. Survival of the patients is marked limited by the nature of the associated conditions and by the severity of the renal failure itself. In those who survive, clinical recovery is the rule. However, residual defects in renal structure and functions may be present. In a few patients renal failure does not improve temporarily or may not improve at all and may lead to CRF.The recent literature survey showed the paucity of systematic ARF etiopathogencesis data developed in basic science dboratories, in Central India and in general in Amravati division in investigation on ARF in human patients in Amravati region so that a comprehensive clinical ARF data could be evaluated for the better understanding and final diagnosis and consequent appropriate

RESULTS

The present investigation-as carried out in 39 patients of varied etiologies, admitted to the nephrology unit and were studicd during the period of December 2003 to May 2007, out of these 39 patients, 26 were males and 13 were females respectively. Their mean age was found to be 15 years to 92 years.

Table 1: Demographic Profile of patients.

Demographic Features		
Sex	Male	n=26
	Female	n=13
Age (Years)	Mean ±SD	51.28±19.699
	Range	15 yrs to 92 yrs

n=denotes the numbers of patients

Table 2: Severity of Creatinine at presentation:

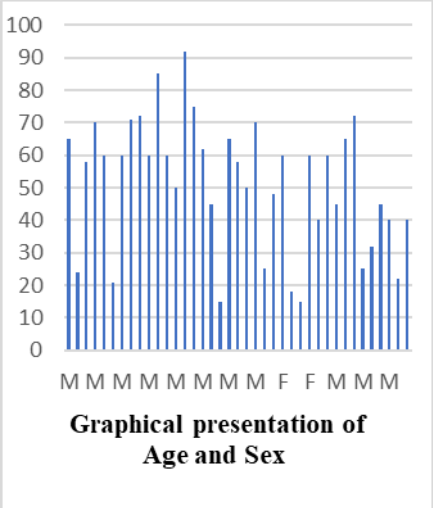
Mild ARF (Serum Creatinine <3 mg %)	30.76%
Moderate ARF (Serum Creatinine 3- 6 mg %)	33.33%
Severe ARF (Serum Creatinine > 6 mg %)	35.89%

Table 3: Severity of Creatinine at discharge

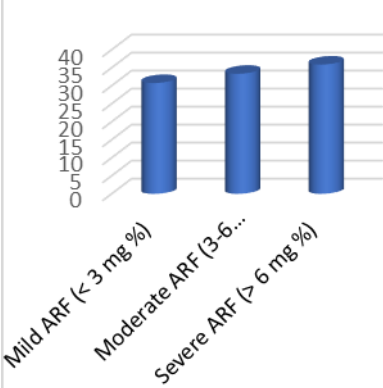
Mild (<3 mg%)	74.35%
Moderate (3 -6 mg %)	15.38 %
Severe (>6 mg %)	0%

Table 4: Creatinine levels (mg %) at presentation and at discharge.

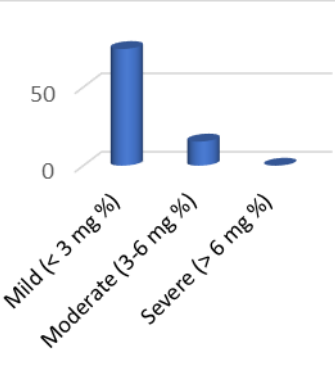
Creatinine at presentation (mg %)		Creatinine at discharge (mg %)
Mean±SD	Mild 2.14 ± 0.3057 Range: 1.6-2.56	1.7769±1.2909 Range 0-5.5
	Moderate 4.4231 ± 0.9814 Range: 3-6	
	Severe 9.46 ± 2.7621 Range: 5.2-14.14	



Graphical presentation of Age and Sex



Severity of Creatinine at presentation



Severity of Creatinine at discharge

DISCUSSION

The present investigation was carried out in 39 patients of varied etiologies, admitted to Nephrology Unit and were studied during the period of Dec.2003 to May 2007, out of this 26 patients were male (66.66%) and 13 were female (33.33%) respectively. The mean age of these patients was found to be 51.28 19.69 ranging from 15 years to 92 years. This age range basically indicates that none of the age group has any inclination or risk for the development of ARF. This result carborates with the finding of Stevens *et al.* (2001) which showed that their 12 month prospective study of initial management of patients with acute

renal failure in East Kent had the mean age as 73 years. The age range was found to be 14 years to 96 years, whereas Prakash *et al.* (2006) reported 46 (3.79%) patients of ARF with the mean age of 44.9 ± 17 years and out of them 56.5% were males. The investigations undertaken by Panda *et al.* (2003) showed the age range of renal population from 16-70 years with the male preponderance which broadly co-relates with our study as we found male dominance with a 66.66% where as the present study revels 33.33% females to have been affected by ARF. While Jai Prakash, *et al.* 1997 observed that patients of diverse etiologies when presentation. All these patients were followed till the time of creatinine < 3mg% in 30.76% patients. Moderate ARF was seen in ARF with creatinine level of > 6mg% was noted in 35.89% at discharge and it was found that these levels of serum creatinine were 33.33% patients with the creatinine level of 3 6mg % and severe classified as elderly ARF with the mean age studied over a period of 9 years showed that 96 (15%) patients were of 72.5 years. Creatinine is produced from creatine, a molecule of major importance for energy production in muscles. Approximately about 20% of the bodies creatine is converted to creatinine every day. Creatinine is transported through the blood stream to the kidneys, which then filter out most of the creatinine and dispose it off in the urine, thus, maintaining the blood creatinine in a normal range. As the kidneys become impaired the creatinine level in the blood rises and the abnormal high levels of creatinine thus warn the possible malfunction or failure of the kidneys. This study presented mild ARF with a serum creatinine<3mg% in 30.76% patients. Moderate ARF was seen in 33.33% patients with the creatinine level of 3 -6mg% and severe ARF with creatinine level of >6 mg % was noted in 35.89% at presentation. All these patients were followed till the time of discharge and it was found that these levels of serum creatinine were well managed to decrease by the time of discharge and the patients showed the mild range of creatinine in 74.35% patients, moderate levels were seen in 15.38% patients and the severe ones were brought down to nil. This was possible due to the conservative treatment given and with the help of dialysis given. Thus, the mean level of creatinine at discharge was 1.7769 + 1.2909 (range 0 - 5.5), with a mild level as 2.14±0.3057 (range 1.6 2.56) and mode-rate level of creatinine with 4.4231 0.9148 (range 3 6), severe with 9.46 + 2.7621 (range 5.2 - 14.14) at the time of admission of the patients. While a dissertation of DM Nephrology from the university of Mumbai in july 1999 showed 19 patients (90.47%) who had severe ARF with a creatinine level of> 6mg% and 2 patients had moderate ARF with a serum creatinine level of 3 6mg% whereas no mild ARF i.e. creatinine level <3mg% was seen in any of the patients. The kidney is a dominant organ regulating the excretion of the sodium. Each day, approximately 25,000meq of sodium is filtered through the glomerulus. The kidneys normally reabsorbs 99% of the sodium and the remaining 1% of the filtered sodium load is excreted under regulation. Proximal tubule is the major site of sodium absorption. Anderson and Schrin (1997). The cell concentrates about 90% of the potassium within it and this cell, when damaged releases the potassium into the blood. Potassium plays the vital role in the nerve conduction, muscle function and helps to maintain the acid-base balance and the osmotic pressure. The elevated potassium levels (hyperkalemia) can be found in oliguria, anemia, urinary obstruction, renal failure due to nephritis or shock, metabolic or respiratory acidosis, renal tubular acidosis with the K-/H- exchange of hemolysis of the blood. Whereas low potassium levels (hypokalemia) can be found in excessive loss of potassium through diarrhea or vomiting, inadequate intake of potassium, malabsorption, severe burns and increased secretion of aldosterone. High or low potassium levels may cause changes in muscle irritability, respiration and myocardial functions. Potassium that is filtered at the glomerulus is nearly completely reabsorbed in the proximal tubule, while the potassium that appears in the urine is secreted by the distal nephron, particularly from the tubular cell into the lumen is passive and is dependent mainly on the electronegativity of the tubule lumen, which is a consequence of active sodium reabsorption. (Wardener, 1986) All these functions are altered in ARF.

The study also showed various symptoms like nausea, vomiting, fever, oliguria, dysuria, hematuria, gastroenteritis, polyuria, pain in abdomen along with the conditions associated with ARF such as trauma, HT, DM, NSAID. Conditions in which ARF is most likely to occur such as Pancreatitis, Pre-renal azotemia, Glomerular disease, obstruction, pregnancy, drug induced ARF were also noted. As per symptometric presentation was concerned 21 patients had nausea and vomiting (53.85%) 24 patients complained of having fever (61.52%) from mild to severe degrees. 25.64% patients i.e. 10 patients had oliguria. 5 patients had significant amount of edema on feet (12.82%). As a presentation of dysuria 12.82% of patients had

preceding symptoms of urinary burning and all these patients had microscopic hematuria as well. Only 4 patients had dyspnea at the time of presentation (10.26%), which later progressed to renal dysfunction, but all these patients had complete clinical recovery. The 15 patient (38.46 %) presented with pain in abdomen at the time of hospitalization for which the exact cause could not be pointed out, when the patients were clinically examined and evaluated we observed associated hypertension in 13 patients (33.33%) who included preeciding history of hypertension and development of HT during the cause of medical illness probably due to salt and water retention or hypokalemia. Polytrauma or vehicular accident complicated to renal failure was observed in one patient only. All the patients after clinical evaluation were investigated according to the probable cause of the disease. Each patient had undergone urinary examination of which 58.97% patients had active urinary sediments i.e. urine albumin positive with macroscopic hematuria and pyuria. Similar results were observed by Ravi Thadani, *et al.* 1996. The author suggested that ARF is characterized by a deterioration of renal function over a period of hours to days, which results in the failure of the kidney to excrete nitrogenous waste products and to maintain the fluid and electrolyte homeostatis. After these serological examinations, hematological examinations were done which revealed malarial parasytemia in two patients. i.e. 5.12% out of which one had Plasmodium vivax positive and the other showed Plasmodium falciparum positive. In the P.vivax positive patient the probable cause of renal failure was pre-renal azotemia because of dehydration and the *P. falciparum* positive presented with proteinuria, microscopic hematuria, DIC gm negative septicemia with renal failure discharged with creatinine of 1mg% whereas (Prakash et al 1997) studied 637 patients with ARF of found that ARF due to diverse etiologies over a period of 9 years an *P. falciparum* was seen 7 patients only. In the same way (Panda et al, 2003) analyzed two thousand and two hundred proved cases of falciparum malaria from the year 1994 2003 and found that 770 cases had the evidence of renal failure and concluded that falciparum malaria was one of the leading cause of ARF in the Southern Orissa (Berhampur) whereas (Chug and Sud, 1998) states that the past decade had seen a resurgence of falciparum malaria in many parts of India. He further added that in the north-eastern state of Assam it accounts for 38% of all cases of ARF.

One patient who had hepato-renal syndrome was HBSAB positive. A 70-year-old man who had presented with the pain in abdomen in right side had congenital left small kidney with impacted calculus in the right ureter. The patient had an acute shut down of kidney leading to elevated serum creatinine of 12.5mg%, after the removal of the stone the creatinine dropped down to 1.5mg%. The patient was not given dialysis. Similarly, Sural, *et al.* 2000 stated nthat ARF associated with liver disease is a commonly encountered clinical problem of varied etiology and high mortality. They prospectly analysed patients with liver disease and ARF to detect the etiology, clinical spectrum, prognosis and factors affecting the outcome and found that other than hepatorenal syndrome 66 patients developed ARF secondary to various liver disease like cirrhosis.

In a group of obstructive uropathy one patient had carcinoma of bladder who was discharged against medical advice and died at home. The present investigation revealed that twelve patients were diabetic and were taking medication for the same. Although proteinuria had been demonstrated in diabetic patients since the eighteenth century (Rollo, 1798). It was Bright who in 1836 postulated that albiminuria could reflect a serious renal disease specific to diabetes. (Bright, 1836.) Multimorbid diabetic patients with nephropathy are particularly prone to develop ARF very often when serum creatinine is already elevated. In the Heidelberg program, 27% of patients with ARF had diabetes (Schevenger *et al.* 2001) Nakazawa *et al.* 1996 reported a patient with rhabdomyolosis which was secondary to hyperosmolar nonketotic diabetic coma, this patient had progressed to acute renal failure. He was a 43-year-old male with diabetes mellitus and was admitted to hospital for three years because of loss of conciousness.

NSAID as a sole cause of ARF was seen in three patients. The most commenest etiology for ARF due to NSAID is either ATN or tubular interstitial nephritis. The analgesic drug can sometimes cause severe amount of gastritis, which leads to dehydration and mild-renal dysfunction. Griffin *et al.* (2000) states that renal prostaglandins by NSAIDS may decrease renal function, especially under conditions of low effective circulating volume. Thus, concluded that NSAIDS represent a relatively uncommon but avoidable. cause of ARF in trail elderly persons.

As mentioned earlier in the Review (Ejaz *et al.* 2004) also mentions that NSAIDS which are a commonly used drugs have adverse effects, mediated via inhibition of prostaglandin synthesis from arachidemic acid by non-specific blocking of the enzyme cyclo-oxygenase leading to vasoconstriction and reversible mild renal impairment in volume contracted states. Further they add that in patients who are on long term NSAIDS without CRF or ARF are found to obtain subclinical renal dysfunction such as reduced creatinine clearance and impaired urine concentrating ability. Although this sub-clinical dysfunction is reversible, on the withdrawal Of NSAIDS, persistent residual dysfunction was reported.

ARF is the most serious and challenging clinical problem when it occurs in pregnancy, which requires prompt recognition, evaluation and treatment to protect both mother and the fetus. (Krane, 1988). The anatomy and physiology of the kidney is altered in pregnancy (Sheehan and Lynch, 1973) (Bailey and Rolleston, 1971) (Cietak and Newton, 1985) A more remarkable change is the increased capacity of the dialated renal collecting system, also known as the physiological hydronephrosis of pregnancy (Fried, *et al.* 1982) (Rasmussen and Nielson, 1988). The calyces, renal pelvis and ureters all dilate accompanied by hypertrophy of urethral smooth muscle and hyperplasia of its connective tissue, often giving the erroneous impression of obstructive uropathy. (Conrad, 1992).

In the present investigation, out of 39 only 1 patient had ARF complicating pregnancy. This patient had intra-uterine death of the baby with discriminated intravascular coagulation who had a creatinine of 10mg at presentation. Similar results were observed by (Brady *et al*. 1996). The author observed that the incidence of pregnancy bad dropped over a recent decade as a result of improved antenatal care and the virtual elimination of post-aboral sepsis. According to them recently the incidence has dropped down to 1 in 10,000 pregnancies. Similarly, (Smith *et al.* 1968) states that pregnancy related ARF is one of the commonest entities, but the incidence is dramatically reducing because of good anti-natal care and proper management of early septicemia.

Kidney secretes a hormone responsible for the formation of RBCS in ARF, this function is detoriated leading to lower level of HB%. It was seen that there was a decrease in the level of Hb% in the patients at the time of admission, this decrease was due to the deficient erythropoietin production. Similar results were shown by (Nielson and Thoysen, 1990) Anaemia is always present in patients having ARF. The hematocrit gradually falls into the 25% to 30% range in the absence of excessive blood loss. Deficient erythropoietin production develops in ARF. Hypovolemia due to hemorrhage should be corrected with packed red blood cells in saline, while isotonic saline is usually appropriate replacement for plasma loss Symptomatic anemia should be treated by blood transfusion says Lieberthal and Levinsky, (1992). This study showed 9 patients 23.07% who had UTI as a sole cause of septicemia leading to renal failure.

UTI is the most common of all bacterial infections, affecting humans throughout their life span. Not only it is common, but the range of possible clinical syndrome it can produce is exceptionally broad, one of them is ARF. 9 patients in this study had ARF due to infection i.e. 23.07 %. Pancreatitis is again one of the most retrimental cause of ARF, the patients suffering from pancreatis had a creatinine level of 1.8mg % at the time of presentation with the normal renal function at the time of discharge.

In the list of drug induced failure 15.38 % i.e. 7 patients suffered from drug induced ARF. NSAIDS, rifampicin and quinolones were the probable cause for drug induced renal failure. (Shaver and Shah, 2002) studied the most important manifestation of aminoglycoside nephrotoxicity is ARF secondary to ATN, the only treatment for aminoglycoside nephrotoxicity is to discontinue the medication and to support the patient during the period of ARF. Amphotericin B is a relatively frequent cause of ARF. Hydration with normal saline before the infusion of amphotericin B decreases the incidence of ARF from this medication. Amongst all these etiological factors 11 patients 28.12 % had pre-renal azotemia because of dehydration, hypovolemia, because of gastritis, entritis, sub-acute intestinal obstruction, improper correction, hydration and cardiogenic shock. While, Shaver and Shah (2005) defines ARF as an abrupt decrease in renal function sufficient to result in retention of nitrogenous waste (azotemia), as measured by an increase in serum levels of blood urea nitrogen and creatinine. Acute renal failure can result from a decrease in renal blood flow (pre-renal azotemia), intrinsic renal parenchymal diseases (renal

azotemia), or obstruction of urine flow (postrenal azotemia). The most common intrinsic renal disease that leads to acute renal failure is ATN.

As a primary cause of ARF glomerulo-nehritis is observed in 3 patients out of which one patient had Wegner's glomerular mitosis (ANCA-C possibility) and one patient had SLE.

The studies carried out by (Chugh *et al.* 1989) indicated that rapidly progressive and post-infectious glomerulonephritis constitutes about ten percent of all the cases of ARF seen in the ropics. According to (Shaha *et al.* 1997) the incidence of ARF associated with the post-infectious form of glomerulonephritis has declined in the west to about one tenth of what was seen in the 1950s. However, it continues to be significant cause of ARF, especially in the pediatric population in tropical countries. As compaired to the western series, patients with rapidly progressive glomerulonephritis are younger and type I (anti-GBM) crescentric glomerulonephritis is less common in patients reported from India.

One patient presented with proteinuria, hematuria, and hypertension. He was diagnosed of having MPGN, and recovered partially at the time of discharge. The list of other causes of ARF were reported as trauma, cardiogenic shock, hepato-renal syndrome, accelerated hypertension contributes one in each case leading to ARF.

As a part of management out of 39, 6 patients 15.38 % required renal replacement therapy in the form of hemodialysis. None of the patients was given peritoneal dialysis. This indicates that the main stream of management remains the correction of etiological factors, proper correction of hydration, control of BP, and treatment of infection without renal replacement therapy. 5 patients 12.82 % were even blood transfusion as the Hb % level decreased. One patient 2.56 % was administered with FFP.

ARF is often preventable, once diagnosed; the causes must be identified and treated. Dialysis is usually considered in the presence of severe hyperkalemia, fluid overload, pulmonary edema, marked acidaemia and pericarditis or encephalopathy attributed to uraemia. The patient who remains oliguric or anuric despite a trial of diuretics and who is receiving multiple intravenous infusions including total parenteral nutrition, is unlikely to escape dialysis. Similarly, patients who present with hyperkalemia and acidosis may be temporized with medical therapy but will likely eventually require dialysis, unless the oliguria can be reversed (Bellomo and Ronco, 1998), Hakim *et al.* 1994 mentions that placement of a hemodialysis catheter carries a significant risk of bleeding, pneumaothorax, and line-associated sepsis. Dialysis with a bio-incompatible membrane may cause additional injury by the activation of complement and neutrophils. Patients on dialysis are shown in Fig. 19.

Before the development of dialytic therapies, the most common ce of death in patients with acute renal failure were progressive uremia, hyperkalemia and complications of volume overload. With the advert of dialysis, the most common cause of death are sepsis, cardiovascular and pulmonary dysfunction, and withdrwal of life- support measures. (Cameron JS, 1986) (Liano *et al.* 1989) (Turney, 1990) (Woodrow and Turney, 1992). In the undertaken study mortality was seen in 3 patients 7.69 % 1 patient could not be followed up. (Lobo, *et al.* 2004) states that the mortality of critically ill patients who developed ARF in an ICU setting is extremely high about 50 - 80%. In another study (Chertow *et al.* 1995) identified various risk factors for increased mortality in patients with ATN, including male sex, advanced age, comorbid illness, malignancy, oliguria, sepsis, mechanical ventilation, multiorgan failure, and severity of illness score. Other factors such as acute myocardial infraction, acute stroke such or seizure, chronic immuno suppression, and metabolic acidosis also have been associated with the relative risk of death after progressive ARF due to ATN as stated by (Chertow *et al.* 1998). Whereas, (Prakash *et al.* 2006) in their study foud 63 % of morality rate. This shows that in the present investigation through proper medication and management the morality rate could be reduced to 7.69%.

CONCLUSIONS

Despite limited data, broad areas of consensus exist for the physiological and clinical principles needed to guide the development of consensus recommendations for defining ARF, selection of animal models, methods monitoring fluid therapy, choice of physiological and clinical end-points, final trials, and the possible role of information technology.

Every effort should be made to prevent further kidney injury and provide supportive measures until recovery has occurred from ARF.

There is no significant prepondance for and age of the patients. Males are seen to be more affected by ARF as compaired to females.

(a) Infection remains the highest etiological factor for ARF
(b) NSAIDS and antibiotics have a significant role to play as a prime cause of ARF
(c) Conservative line of management i.e. without kidney transplantations renal replacement therapy like dialysis has an important role in saving the life of the patient.
(d) Because of early availability of nephrology facilities the total mortality is not very high
(e) Identify and correct pre-renal and post-renal factors.
(f) Optimize cardiac output and renal blood flow Review drugs; stop nephrotoxic agents; adjust doses and monitor concentrations where appropriate
(g) Accurately monitor fluid balance and daily body weight
(h) Identify and treat acute complications (hyperkalaemia, acidosis, pulmonary edema)
(i) Optimized nutritional support: adequate calories, minimal nitrogenous waste production, potassium restriction.
(j) Identify and aggressively treat infection; minimize indwelling lines; remove bladder catheter if anuric.
(k) Identify and treat bleeding tendency: prophylaxis with proton pumps inhibitor or H2 antagonist, transfuse if required, avoid aspirin
(l) Initiate dialysis before uraemic complications emerge.

So, we summarize that, early detection of the disease, proper correction of precipitation g factor and available renal replacement therapies are important in the management of ARF.

The broad conclusions that flow out from the present investigations are:

(a) There is no significant prepondance for and age of the patients. Males are seen to be more affected by ARF as compared to females.
(b) Infection remains the highest etiological factor for ARF
(c) NSAIDS and antibiotics have a significant role to play as a prime cause of ARF
(d) Conservative line of management i.e. without kidney () transplantations renal replacement therapy like dialysis has an important role in saving the life of the patient.
(e) Because of early availability of nephrology facilities the total mortality is not very high
(f) So, we summarize that, early detection of the disease, proper correction of precipitation g factor and available renal replacement therapies are important in the management of ARF.

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