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A dissertation presented in part fulfilment for the degree of Master of Medicine in Internal Medicine in the University of Nairobi (Kenya).

DECLARATION

I hereby certify that this dissertation is my own original work, and has not been presented for a degree in any other University.

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This dissertation has been submitted for examination with my approval as University Supervisor.

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SUMMARY

47 patients with acute renal failure without a background of chronic renal failure were studied between January 1984 to December 1985. The aim was to study several aspects of the presentation and management of Acute Renal Failure at Kenyatta National Hospital.

The mean age of presentation was 27.8 years and the peak age was between 21 and 40 years. The mortality rate was 40.4%. There were 21 males and 26 females. Most patients came from Nairobi and the surrounding areas within easy reach of Kenyatta National Hospital. There were few patients from distant areas.

The mean duration of stay in hospital was 20.1 days. The mean period from the start of the disease to the onset of oliguria was 2.8 days, and the mean period of oliguria was 13.4 days. The mean period of diuresis was 14.4 days.

Most patients (46.8%) had medically orientated problems but obstetrical and gynaecological problems had a high mortality.

Common medical causes of acute renal failure were acute glomerulonephritis and malaria, while obstetric hemorrhage due to various causes was the most common cause in obstetrics. Surgical patients were very few.
Complications that were associated with a high mortality were neuropsychiatric, and infections. Bleeding diathesis, pulmonary edema and anemia were also associated with increased mortality. Pericarditis was a rare complication, as was pulmonary embolism. Hyperkalemia was associated with a high mortality, but a high blood urea nitrogen was associated with a prolonged stay in the ward. There was a high incidence of anemia.

Dialysis was generally started when the levels of potassium and blood urea nitrogen were high but was associated with good prognosis.
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ACUTE RENAL FAILURE AS SEEN AT KENYATTA NATIONAL HOSPITAL (K.N.H.)

This is a prospective study of patients seen at KNH between 1st January 1984, and 30th December 1985, considering their presentations, aetiologic factors, complications, managements, and their morbidity and mortality.

INTRODUCTION

Acute Renal Failure (ARF) is defined as an abrupt decline in renal function, with retention of nitrogenous wastes, which is not reversible with manipulation of extrarenal factors. Although histologic evidence of tubular necrosis occurs in a minority of cases, the terms acute tubular necrosis, (ATN) and acute renal failure are used interchangeably in clinical practice. The causes of such deterioration include renal hypoperfusion of various aetiologies, (pre-renal) obstructive causes (post renal) and intrinsic renal diseases such as acute glomerulonephritis.

Acute Renal Failure is a medical emergency with high morbidity and mortality, though there is always potential for full recovery of renal function. It is mandatory that great effort be made in the management of all patients if prognosis is to be made better with a drop in morbidity and mortality.
This study intends to analyse all aspects of the presentation of acute renal failure, including aetiologies, clinical presentation, complications, management, natural course of the disease, and prognosis, as well as several factors that have an influence on its outcome.

REVIEW OF LITERATURE

CAUSES OF ACUTE RENAL FAILURE

The causes of acute renal failure can be grouped into one of 3 classes: inadequate perfusion of the kidneys or pre-renal azotaemia, obstruction or post renal azotaemia, and intrinsic renal disease. Such a classification shows the need for rigorously excluding both pre-renal and post-renal disorders, which have specific therapies which are often curative. Intrinsic renal disease do not normally have immediate specific treatment.²

INADEQUATE RENAL PERFUSION

This is a common and frequently unrecognised cause of renal insufficiency. Oliguria occurs as a result of reduced glomerular filtration rate coupled with enhanced salt and water reabsorption, reflecting the normal response of the renal tubules to a deficit of effective extracellular fluid volume.² Intravascular volume depletion with little or no external fluid
losses also may lead to oliguric renal failure as may occur during rapid "third space" accumulations as extensive bowel surgery, intestinal infarction, peritonitis, or acute pancreatitis. Prerenal azotemia is usually associated with a high urine specific gravity (1020 - 1030), low urine sodium concentration (less than 5mEq/l) and an unremarkable urine sediment.\(^2\)

**OBSTRUCTION.**

Urinary tract or bladder outlet obstruction, due to Benign prostatic hypertrophy especially in older men and congenital anomalies in infants may cause sudden and often total cessation of urine output. Bladder outlet obstruction is characterised by both anuria (urine output of 0-50ml/Day) and rapid large swings in urine outputs. Upper tract obstruction is a much less common cause of oliguria since simultaneous obstruction of both ureters is an unlikely event. However, bilateral upper tract obstruction e.g. by retroperitoneal fibrosis and unilateral obstruction of a single kidney as in pelvic malignancy, must be considered in a patient with unexplained oliguria, and relevant investigations should be instituted.
INTRINSIC RENAL DISEASES

These are of diverse types and include vascular diseases that exert their effects by a vasculitis such as malignant hypertension, thrombotic thrombocytopenic purpura, and arterial and or venous occlusion. Other causes include glomerulonephritis and antiglomerular basement membrane diseases, as well as interstitial nephritis due to drugs, the penicillin, especially methicillin, hypercalcemia and diffuse infarction. Acute tubular necrosis is one of the most prevalent causes of acute renal failure due to intrinsic renal diseases, and its pathogenesis is discussed below.

ACUTE TUBULAR NECROSIS.

This follows a period of shock or haemorrhage resulting in renal ischaemia, or it may be caused by direct renal damage. The clinical features and the pathology of the condition vary only slightly with aetiology. Although spontaneous recovery often occurs, the mortality is still considerable and Kennedy et al (1974) in surveying 250 cases, found a mortality of 44%. Minuth et al (1976) found an overall mortality of 57%. The mortality in the group caused by nephrotoxic drugs was the lowest (36%). Robert Anderson et al in 1977 found a mortality of 50% in oliguric acute renal failure and 26% in non-oliguric acute renal failure.
in their study of 92 patients with acute renal failure. Over the past decade between 1970 and 1980 the mortality from 6 series of 636 patients reported an overall mortality of 47% in Oliguric acute renal failure and 32% in non oliguric acute renal failure.

The nephrotoxic and the ischaemic categories of acute tubular necrosis frequently overlap, thus in such states as mismatched transfusion, there is a combination of shock and a possible toxic effect of free haemoglobin, and even of immunological damage from altered red cell stroma. The hypotensive situation results in a greatly diminished renal blood flow and glomerular and tubular damage. The toxic conditions may cause injury to specific portions of the renal tubule.

Some of the causes include traumatic shock associated with hypotension burns and crush injuries, post operative shock and surgical trauma, obstetric shock, abortion, septicaemia, acute peritonitis and pancreatitis, poisons, 'nephrotoxins' e.g. inorganic and organic mercury compounds and conditions causing acute haemoglobinuria, as mismatched blood transfusions and disseminated intravascular coagulation.

Oliguria, indicating a reduced urine output of 500ml/day is a fairly constant, but not invariable feature of the syndrome.
Four clinical phases are described:

The first phase usually lasts between 1 and 10 hours, and extends from the time of the initial insult to the onset of oliguria.

The oliguric phase may last a variable duration, ranging from 2-28 days. Rarely is there true anuria. The urine is of low specific gravity, often dark, and the ratio of urinary urea and plasma urea is reduced. These indicate depressed tubular function. Proteinuria is usually absent or slight, Urinary sodium and chloride are not unduly depressed, while the sodium value is often greater than 30 mmol/l.

The diuretic phase lasts from 2 - 14 days. The urine resembles glomerular filtrate and there may be a considerable electrolyte loss.

The recovery phase starts gradually with the urine being more concentrated, as tubular function returns. The tubular concentrating defect may persist for years following the acute insult.

PATHOPHYSIOLOGY

The precise pathogenesis of acute renal failure remains uncertain, although many of the theories were introduced 50 years ago.

Current pathogenic theories of acute tubular necrosis suggest either a tubular or vascular basis for the lesion. Dible and Bull in 1953, put forward the theory that leakage of glomerular filtrate occurred through the damaged tubule into the interstitial tissue. Consequently there is a rise
in intrarenal tension, this in turn was considered to cause obstruction to renal blood flow followed by a reduction in glomerular filtration rate and consequent oliguria. However, measurements of intrarenal tension in experimental animals have shown no difference in intrarenal pressures, between normal kidney and those with acute tubular necrosis.\textsuperscript{13}

Mechanical obstruction of the tubules by casts or by swollen tubular cells has been invoked as a cause of anuria. This is wrong, as when recovery occurs, the casts are removed, and provide no obstruction to urine flow. Furthermore, there is no evidence of dilatation of those parts of the nephron proximal to the casts or the swollen tubular cells, as would be expected if they were causing obstruction. Sevitt\textsuperscript{14} argued against the theory that oliguria and uraemia were related to back diffusion of glomerular filtrate through the tubules. He showed that tubular necrosis was more common than renal failure in patients at risk, and also that uraemia could appear following trauma, without tubular necrosis. He proposed that renal failure was precipitated and maintained by a low glomerular filtration rate resulting from renal ischaemia. Some experimental work has tended to support this view, as did the clinicopathological study of Finckh, Jeremy and Wliyte\textsuperscript{15} in 1962 that supported the theory that oliguric renal failure is the result of diminished renal blood flow and reduction in glomerular filtration rate.
rate. These authors noted a lack of correlation between the clinical feature, and the lesions seen in the tubules. They considered that the majority of the tubular lesions were due to ischaemia, but that the formation of casts was related to the slow rate of glomerular filtration, especially when there was a heavy pigment load and that lesions in the distal tubules and interstitial inflammation were a reaction to impacted casts.

The role of Renin-Angiotensin system in the initial cortical and glomerular ischaemia following some forms of shock may be important, Brown et al 1970\textsuperscript{16}. The hypotension causes release of renin into the blood from the kidney leading to the formation of angiotensin I, then angiotensin II. The latter causes preglomerular arteriolar constriction, and thus decreased glomerular filtration rate. It is not clear how the renin angiotensin system is activated in cases where there is no severe hypotension, as the initial insult. It also does not explain the prolonged oliguria or anuria that occurs after the blood pressure has returned to normal.

Clarkson et al 1970\textsuperscript{17} found deposits of fibrin and platelets, consistent with intraglomerular capillary thrombosis. Serial studies on coagulation and
fibrinolysis showed abnormalities to be present in
the oliguric phase with elevation of fibrinogen
degradation products, plasma fibrinogen, soluble
fibrin monomer complex and platelet factor 4.
Recovery of renal function was associated with
resolution of intraglomerular coagulation, correction
of coagulation abnormalities and excretion of large
quantities of fibrinogen degradation products in
urine. The obstruction of glomerular capillaries by
fibrin platelet aggregates was considered a possible
major factor in oliguria.

A fundamental objection to the theory that lack
of glomerular filtration is the prime cause of anuria,
is based on radiological evidence, where Chamberlain
and Sherwood (1966)\textsuperscript{18} induced in rats, acute renal
failure due to temporary renal ischaemia, by injecting
mercuric chloride intraperitoneally. All the animals
had a dense nephrogram on pyelography indicating
glomerular filtration was occurring.

A dense nephrogram also occurs in patients with
acute tubular necrosis, and in contrast to normal
subjects, it persists unchanged for hours or days.
Since the nephrogram depends on the contrast medium
being filtered by the glomerulus and being present
in the tubules, these observations favour the back
diffusion theory of oliguria, in which the oliguria
is due to complete reabsorption of glomerular filtrate
into the renal venules through the damaged proximal tubular epithelium, and is probably dependent on an osmotic effect. This theory is also compatible with the ultrastructural observations on the renal tubules where focal areas of cell necrosis are seen. This is the theory of back leak of glomerular filtrate. The theories that have been reviewed above include tubular obstruction, efferent arteriolar constriction sufficient to reduce glomerular capillary pressure, decreased glomerular membrane permeability, activation of the renin angiotensin system and cell swelling.

The precise pathogenesis of acute renal failure thus remains uncertain. Several experimental models of acute renal failure have been studied and these have shown that the pathogenesis is multifactorial and a comprehensive theory to account for the pathophysiology of hypofiltration in all phases of all models of acute renal failure cannot be provided. The models studied have included the uranyl nitrate model, the mercuric chloride model and the aminoglycoside model. These models have incriminated the already mentioned factors in pathogenesis of acute renal failure, viz, reduction in renal blood flow, intratubular obstruction, back-leak of filtration makers and reduction in the filtration constant of the glomerulus.
Whatever the pathogenesis, acute renal failure is a life threatening, generally reversible illness whose outlook is otherwise excellent if managed appropriately. The outcome depends on the meticulous attention to detail and keen supportive and aggressive treatment like employment of dialytic therapy. This is further reviewed in the section on management of acute renal failure.

New categories of medical patients with acute renal failure have been recognised and have received considerable attention over the past few years. These include non-traumatic rhabdomyolysis/myoglobinuria induced by seizure, myopathies, strenous exercise, and coma from drug overdosages. Nephrotoxicity caused by aminoglycosides also contribute to the aetiology of acute renal failure. These drugs have formed cornerstones of therapy in gram negative bacillary infection. The toxicity is greatest when there is interaction between cephalosporins and aminoglycosides.

Radiocontrast induced acute renal failure has also appeared in the literature as well as acute renal failure following cardiac surgery. Although the potential for nephrotoxicity by radio contrast has been recognised for more than two decades, the increased attention in literature of acute renal failure following radio contrast studies has been both due to an increased awareness and an increased incidence of the problem. It is particularly associated with
diabetes mellitus and multiple myeloma, and is more apt to occur in patients with preexisting renal disease. The diabetic patient is susceptible to renal failure due to vascular disease while in multiple myeloma, the complication is due to obstruction of the renal tubules by protein casts formed by precipitation of Bence Jones proteins by contrast medium. Protein binding is an important determinant of Bence Jones protein precipitation, and this explains why modern contrast media, such as diatrizoate cause renal failure less frequently than the now discarded acetrizoates. Dehydration is particularly hazardous in these patients and must be avoided.

Some of these patients may have hypercalcemia with hypercalciuria and hyperuricosuria/deposits of calcium and uric acid crystals in the kidney.

Preexisting renal disease predisposes to acute renal failure in radiocontrast media studies, firstly by the higher dose of radiocontrast required in the presence of uremia, secondly by the diminished functional reserve of the kidneys making them susceptible to injury and thirdly by the prolonged plasma half-life of contrast media in renal failure. Contrast media may cause the precipitation of Tammy-Horsfall protein in the tubular lumen, resulting in intrarenal obstruction.

Rhabdomyolysis, long known to occur in crush injuries, burns and inflammatory states of muscle, also occurs in a variety of settings in which muscle blood flow, oxygen supply or metabolism is disturbed.
or muscle energy consumption is increased. Grossman, R.A. et al have emphasised the occurrence of non traumatic rhabdomyolysis secondary to myopathies, seizures, strenous exercise or coma from drug over dosage. Symptoms include muscle pain and weakness, muscle swelling and tenderness. The diagnosis is supported by a constellation of laboratory findings including orthotolidin positive urine dipstick of RBC free supernatant, pigmented granular casts, and elevated muscle enzymes. Other characteristic abnormalities include rapid rises in serum creatinine, potassium, uric acid and phosphates.
RENAL FAILURE IN THE TROPICS

Reversible acute renal failure.

The commonest cause is as elsewhere, acute tubular necrosis. Experience of MSR Hutt and A.J. Wing based on clinical and post mortem studies suggests that it is much commoner than is evident from literature. This is partly due to the pattern of disease in tropical areas and partly due to the late hospitalisation of patients with precipitating factors, due to problems of distance and transport availability. Important causes include traumatic haemorrhage and the crush syndrome, post abortal and post partum haemorrhage, hemorrhage from oesophageal varices and incompatible transfusion.

Acute primary and secondary infections, if untreated in the early stages, is associated with acute tubular necrosis often as a preterminal events e.g. in Typhoid, lobar pneumonia, septic abortion, dysentery, gastro-enteritis. While these are diseases also found in temperate countries, they are particularly serious in the tropics.

Occasionally, cases of acute tubular necrosis of uncertain origin occur in patients who have been treated with herbal medicine which may be containing nephrotoxic substances.
The occurrence of acute tubular necrosis in association with acute infections may also be related to high incidence of G6PD deficiency in some areas. Lwanga & Wing 1970. The incidence is about 15% in some parts of East Africa where malaria is endemic and 20% among the Yorubas of Nigeria. Gilles & Ikene 1970. Fulminating infections may themselves cause hemolysis in patients who have the defect, and the hemolytic effect of some antimalarials, which all patients with fever are likely to receive in the tropics, is known to be enhanced by bacterial infection. Intravascular hemolysis is a potent factor in the development of acute tubular necrosis, due possibly to liberation of thromboplastic factors from lysed RBC, and the activation of intravascular coagulation, Rodriguez Edmann 1965.

The glomerular alterations in plasmodium malaria infection have been well described, especially in nephrotic African children. Glomerular changes are considered to be of immune complex nephritis. Plasmodium falciparum infection is also known to cause an acute transient reversible glomerular lesion developing 1-3 weeks after infection. At this time, antigen, immunoglobulins and complement (BIC-globulin) are detected in glomerular deposition. This lesion responds to antimalarial therapy and the abnormalities disappear in a vast majority of cases. It can be a cause of acute renal failure. Tubular lesions due to plasmodium falciparum, are a more recognised
cause of acute renal failure, and will be discussed later.

Nephrotoxicity by the action of poisons is uncommon but toxic chemicals of various identities have been incriminated. Snake bites by snakes which produce hemolytic toxins can also cause acute renal failure. This can also result from disseminated intravascular coagulation, caused by some venoms.

Sickle cell anaemia will also cause acute renal failure in situations of intravascular sickling leading to massive hemolysis similar to disseminated intravascular coagulation and the hemolytic uremic syndrome.

DIAGNOSTIC APPROACH TO ACUTE RENAL FAILURE

History and physical examination often provide significant clues in evaluation of patients with acute azotemia. Pre-renal and post renal causes are usually easy to diagnose.

Important historical and physical evidence in pre-renal azotemia include recent blood or fluid loss, past history and signs of cardiac disease, diuretic therapy, and lack of appropriate fluid ingestion. Volume depletion will be further attested to by evidence of increased sympathetic activity and hypotension. Hypoperfusion will conversely be evidenced by no change in weight, or even an increase, as when intravascular volume falls in generalised oedema.

Post renal azotemia may be the cause when there is a history of recent surgery involving the urogenital tract. Past history of nephrolithiasis, haematuria
(obstruction by clots), granulomatous disease (causing retroperitoneal fibrosis) are all important including symptoms of bladder outlet obstruction. Sloughed papillae in patients susceptible to papillary necrosis (Diabetics, alcoholics, analgesic abusers) may be the cause of obstruction. Pain may or may not be present in acute obstruction.

Acute glomerulonephritis will be suspected as a cause of intrinsic renal, acute renal failure in the hypertensive, proteinuric, hematuric child or adult where there is smokey urine and red blood cell casts. The symptoms are less obvious in the adult. Drug induced vascular inflammation, as a cause of acute allergic interstitial nephritis, will be evidenced by fever, rash and eosinophilia in addition to the urinary findings which include proteinuria, haematuria and eosinophiluria. Acute tubular necrosis develops in the presence of vascular instability, sepsis, ingestion of a variety of toxins and drugs, and rhabdomyolysis.

**URINARY FINDINGS.**

Although oliguria is commonly considered the hallmark of acute tubular necrosis, urine volume is unreliable for diagnosis due to the occurrence of non oliguric acute renal failure. Anuria is a rare occurrence, having been found in 1 of 85 patients in a series by Swann and Merrill 1953. It often is
associated with bilateral cortical necrosis, acute bilateral renal arterial or venous occlusion, or severe acute glomerulonephritis. Positional change of a stone or tumour will cause wide fluctuations in daily urine volume.

The urine volumes of acute tubular necrosis is usually in the range of 100-400 ml daily.

Non oliguria, or high output acute tubular necrosis has been recognised since 1943. It mostly occurs in burn patients and following treatment with nephrotoxic antibiotics, but may develop in acute tubular necrosis of any cause. Urinary volumes usually range between 600 to 1500 ml daily. Robert J Anderson et al studied prospectively 92 patients with acute renal failure, 54% of whom were non-oliguric. They had a lower mortality and morbidity (26% mortality in non oliguric as opposed to 50% in oliguric patients). The probable decrease in morbidity and mortality in non-oliguric acute renal failure has been explained on the basis of higher glomerular filtration rate and relative preservation of tubular function when compared to the oliguric state. Problems related to volume overload and hyperkalaemia are ameliorated and more latitude is allowed in the treatment of acidosis with bicarbonate. Post furosemide non-oliguria may confer the same benefit as the spontaneous variety, hence large doses of furosemide should be administered
early in the course of oliguric acute renal failure, to convert it to a non-oliguric state with the attendant improved prognosis. However, the prophylactic value of furosemide is still far from established, but it is known that furosemide use especially in well hydrated patients is useful. This was shown by KleinKrechc who found that, compared to controls not treated with diuretics, patients given large doses of furosemide (up to 3,200mg per day) had a higher urine output, suffered a shorter duration of oliguria (5.7 vs 15 days) needed less dialysis (2.8 treatment vs 8.81 and showed a more rapid decline in serum creatinine and blood urea nitrogen. Differential urinary chemical profile is useful in the diagnosis of acute renal failure. When patients with prerenal azotemia were compared with those patients who had renal acute renal failure, the preserved tubular function of the former was evidenced by elevation of urine osmolality urine/plasma urea Nitrogen and creatinine, and by a low urine sodium 20umol/l. The patients with acute renal failure failed to demonstrate such tubular functional intergrity.

Assessment of the urinary sediment is also of great value in the differential diagnosis of acute renal failure. The pH ranged from 5.5 to 7 in acute
tubular necrosis. A strongly acidic urine is suggestive of prerenal azotemia or acute glomerulonephritis. Evidence of pathogenesis of hematuria by a positive dipstick reaction is common in any of the intrinsic renal injuries or post renal ARF and signifies the presence of pigments. The absence of red blood cells suggests hemoglobinuria or myoglobinuria. The finding of one to two positive proteinuria is compatible with acute tubular necrosis. This is non-selective.

Microscopy in prerenal insufficiency may reveal increased numbers of hyaline and finely granular casts. The sediment in acute tubular necrosis contains large numbers of tubular cell, tubular cells casts and coarsely granular pigmented casts. White cell and white cells casts are encountered in glomerular or interstitial nephritis. Large number of uric acid, calcium oxalate, or triple phosphate crystals may suggest the aetiology of the acute renal insufficiency.

Renal biopsy has a potentially important place in the diagnosis of acute renal failure. Criteria include the following: no obvious causes for acute renal failure, oliguria or anuria lasting longer than 3 weeks, physical or laboratory findings suggestive of a primary renal disease, vasculities or systemic disease, and exposure to drugs known to be associated with tubulo-interstitial disease. Investigations may be required which will tell whether hydronephrosis
is present, or whether renal arterial or venous flow is impaired. A plain abdominal film may reveal radio-opaque stones, or abnormalities in renal size. Intravenous pyelogram is a known cause of acute renal failure as mentioned earlier and the need for their use in acute renal failure should be weighed carefully. Hydronephrosis will cause characteristic changes in the nephrogram. Oliguric intrinsic renal acute renal failure causes several characteristic nephrographic patterns in this disease. An immediate dense and persistent nephrogram is characteristic but not pathognomonic.

Ultrasonography is a non-invasive investigation which can accurately document the presence of one or both kidneys, measure renal size and define the morphology of the collecting system. The role of the computerised axial tomography requires further definition as adequate data can be gathered by the preceding radiologic procedures. We do not have this facility at Kenyatta National Hospital.

Contrast studies of the renal vasculature are invaluable in selected clinical circumstances as when renal cortical necrosis is suspected. It will also make the diagnosis of vascular occlusion, especially in excluding renal arterial thrombosis in acute renal failure following transplantation.
Radionuclide perfusion scanning may have a role in post transplant patients with acute renal failure who require arteriography.

**CLINICAL COURSE OF ACUTE RENAL FAILURE**

Mention has already been made of the four phases of the clinical course of acute renal failure viz the initiating phase, the oliguric phase, the diuretic phase and the recovery phase. Recognition of the initiating phase is very important, since early correction of the underlying cause can theoretically prevent the development of the subsequent phases, thus prerenal and post-renal factors should be corrected. This phase however commonly escapes the clinician. Oliguria starts shortly after the initiating event and takes an average of 10 - 14 days. The usual range is 2-28 days though it can last as long as 6-8 weeks. Prolonged oliguria occurs commonly in the elderly with underlying vascular disease. Oliguria lasting more than 4 weeks is more commonly associated with diffuse cortical necrosis, rapidly progressive glomerulonephritis and renal artery occlusion and renal vasculitis.

Two factors are important in causing the severity of resultant abnormalities in blood chemistry viz whether the patient is oliguric or
non-oliguric, and the catabolic state of the patient. In the febrile catabolic oliguric patient with acute renal failure, the daily increment in blood urea nitrogen and serum creatinine average 10-20 mg/dl and 0.5-1.0 mg/dl respectively. In catabolic patients with fever, sepsis or extensive trauma, daily increment in BUN and serum creatinine may be as high as 40-100 and 2-5 mg/dl respectively.
MANAGEMENT

Management of acute renal failure.

Acute renal failure is a medical emergency. The first principle of therapy is to exclude causes of deterioration in renal function which are potentially remediable. A search of pre-renal factors, obstructive uropathy, glomerulonephritis, renal vascular and interstitial disease should be made.

There are interesting pathophysiologic aspects in the use of diuretics in acute renal failure. The vasodilators like bradykinin and secretin which can be elaborated during acute renal failure have got protective effect on the kidney, comparable to prostaglandins, especially prostaglandin E2 ($\text{PGE}_2$). $\text{PGE}_2$ increases renal blood flow and solute excretion. However, bradykinins as opposed to secretin, increases blood flow, solute excretion and urinary flow. Both are vasodilators. Mannitol and furosemide have these function, i.e. increase of renal blood flow and solute
excretion and increased urine flow. Other diuretics like thiazide diuretics, are vasoconstrictors, and may decrease urinary flow. Furosemide and mannitol are thus recommended in the initiation phase of oliguric acute renal failure to increase urinary flow.

There should be anticipation of complications of acute renal failure and strenous efforts to avert them. Infection still remains the leading cause of death in acute renal failure. Vigilance in the maintenance of IV lines, attention to pulmonary hygiene and avoidance of indwelling bladder catheters are essential. Gastrointestinal hemorrhage is another serious cause of mortality and judicious use of cimetidine is indicated in the high risk patients.

Judicious water and salt restriction depending on well kept input - output charts are needed in the oliguric patient and adequate supplies of fluids should be allowed in the non-oliguric patients.

Serum electrolytes, BUN, creatinine, fluid intake and output with weight charts should be carefully monitored. High serum phosphates should be lowered with aluminium hydroxide (aludrox) resin. Nephrotoxic antibiotics and other drugs must be used with caution, depending on the level of serum creatinine. Adequate caloric intake must be ensured with needed dietary protein to decrease wasting, improve general clinical condition, and prevent uraemic toxicity.
Dialytic therapy is used to prevent rather than treat uremic complications, such as hyperkalemia, cardiac arrhythmia, and fluid overload. This has been shown through several studies that show that prophylactic dialysis is associated with less morbidity and mortality and less uremic complications. Aggressive dialytic therapy is also associated with less mortality than the non-aggressive treatment. Serum creatinine should be kept at 8-10 mg% and BUN at less than 100 mg%. Early dialysis gives an improved environment for total resistance to infection, and better wound healing. It also prevents or lowers the uremic complications that are more easily prevented than treated. Dialysis also allows the administration of fluids and/or protein to ensure adequate nutrition, and early alimentation.

The hypercatabolic state exerts an immuno suppressive effect on wound healing, and immune competence. It also reduces muscle mass. The situation is worse when there is malnutrition which may occur in such very ill patients. Thus adequate calories and suitable positive nitrogen balance best suited to the abnormalities of acute renal failure, should be provided. Parenteral nutrition is sometimes used as most patients are too ill to feed orally.
Infusions are used with essential amino acids, and hypertonic glucose. This is especially useful in oliguric acute renal failure with pneumonia, gastrointestinal hemorrhage and generalised sepsis.

Peritoneal dialysis is adequate for most patients. A few in hypercatabolic states may need hemodialysis. Peritoneal dialysis is popular in the developing countries because it is less expensive and requires highly skilled personnel. It also does not require large blood supplies and frequent transfusions as does hemodialysis.

Disposable peritoneal catheters and commercially prepared dialysis fluids have made this technique convenient and the dialysis can usually be started within the hour from deciding it is needed.

Absolute indications for dialysis include symptomatic uremia usually manifested by central nervous system and or gastrointestinal symptomatology, development of resistant hyperkalaemia, severe acidemia, or fluid overload not responsive to medical therapy, and pericarditis. In addition, many centres attempt to keep predialysis levels of BUN and creatinine less than 100 and 8mg% respectively because above these levels the complications are severer like haemorrhagic, colitis and pericarditis.
AIM AND OBJECTIVES.

This study aims at improving the management and outlook in acute renal failure at the Kenyatta National Hospital. The patients are usually referred to the renal unit by all wards at the hospital. Many of these patients have been referred from outlying referral units as district hospitals, and provincial hospitals as these units do not have facilities for dialytic therapy. A good number of patients, are primarily from the different departments of the Kenyatta National Hospital Complex.

The study relates the prognosis of patients with acute renal failure to several factors and also attempt to determine the significance of these factors to the outcome.

These include:

1. Age of patients at onset of acute renal failure
2. Sex of the patients.
3. The presentation of the patients, including the duration of illness before presentation to hospital, and the interval between onset of acute renal failure and consultation of the renal team.
4. The causes of acute renal failure.
5. Findings of physical examination and their significance in relation to prognosis.
6. Complications and their severity.

Particular attention is paid to occurrence of pulmonary edema, hyperkalemia and bleeding tendencies,
pericarditis, embolic phenomena, sepsis, and neuropsychiatric complications as fits, asterexis and confusion.

7. Laboratory data are tabulated. Particular attention is paid to urinalysis, potassium and urea levels.

8. Infection as a cause or complication of ARF is closely monitored.

9. The clinical type of ARF is analysed, e.g. oliguric or non-oliguric, together with their prognosis.

10. The management of the patient, with respect to
   
i. Duration of conservative management.
   
ii. Prognosis with dialytic therapy.
   
iii. Type of dialytic therapy.
   
iv. Level of BUN & K⁺ at which dialysis started.
   
v. Duration of peritoneal dialysis, with or without hemodialysis and reason for change.
   
vi. Duration of illness before death.
   
vii. Cause of death.
   
viii. Duration of oliguria/anuria.
   
ix. Duration of diuretic phase.
MATERIALS AND METHODS

This is a prospective study on all patients with acute renal failure seen between 1 January 1984 and 30th December 1985, at Kenyatta National Hospital. These were patients referred to the renal unit for expert management of acute renal failure. All these patients were reviewed by Renal Registrars who are senior house officers, doing Master of Medicine course with the University of Nairobi in Internal Medicine and Paediatrics, and are attached to the renal unit for an average of three months. All patients were also reviewed by renal physicians who are attached to the departments of paediatrics and medicine.

Excluded from the study were the following categories of patients:

1. Patients in whom the acute renal failure was on a background of chronic renal failure, as identified by appropriate clinical and laboratory evaluation.

2. Patients in whom the acute renal failure was so mild that referral to the renal unit was withheld, and therefore escaped my attention.

3. The patients with uropathy as caused for example by Benign prostatic hypertrophy, posterior urethral valve and strictures were excluded from the study because many of them had some degree of chronic renal disease due to the obstruction, and would therefore not qualify to be treated as having acute renal failure.
Included were all patients with acute renal failure who were seen by the Renal team during the whole of 1984 and 1985, most of whom I participated in their management.

Laboratory data were assessed in the main hospital laboratories. Blood urea nitrogen and serum sodium and potassium levels were assessed by Sequential Multiple Analysis (SMA) machine Technician 2. Several specimens needed urgently at night and during the weekends were tested with Urostat strips for blood urea nitrogen and flame photometer for serum sodium and potassium levels in the renal unit laboratory. Many specimens were also similarly tested for BUN, sodium and potassium in the department of medicine by Mr. Ndirangu and myself.

Liver function tests as SGOT, bilirubin and alkaline phosphatase were measured by SMA Machine Technicon 1.

Hemoglobin and white cell counts were assessed by the Coulter Counter Model S, while the differential white cell count, platelet count and peripheral blood film were done manually.

Blood glucose was assessed by the glucose oxidase method.

Urinalysis was done for hematuria, proteinuria, pyuria, casts, crystals and urine culture and sensitivity. Blood culture and sensitivity was also done in selected cases. Peritoneal dialysis fluid culture and sensitivity was also included where appropriate.
RESULTS

A total of 47 patients were seen in the study. There were 20 males and 27 females. Their ages ranged from 6 months to 60 years with a mean of 27.8 years. Most patients seen were between the ages 20 and 40 years with the peak of 20-30 years. The Paediatric population were mostly in the age range 0 - 10 year. There were only 2 patients in the age range 11 - 20 years. This is shown in figure 1.

Figure 1:

Figure showing age distribution in years, at presentation and the number alive and dead.

Key: 
- □ Alive
- ◯ Dead
There were 9 patients below 21 years of age, 21 patients between 21 and 30, and 17 patients above 50 years, as shown in table 1. The mortality within these age groups did not differ significantly.

Table 1: The Table showing age distribution at presentation, and mortality in the age groups.

<table>
<thead>
<tr>
<th>Age</th>
<th>Alive</th>
<th>Dead</th>
<th>Total</th>
<th>% Dead</th>
</tr>
</thead>
<tbody>
<tr>
<td>21</td>
<td>4</td>
<td>5</td>
<td>9</td>
<td>56</td>
</tr>
<tr>
<td>21-30</td>
<td>13</td>
<td>8</td>
<td>21</td>
<td>38</td>
</tr>
<tr>
<td>30</td>
<td>11</td>
<td>6</td>
<td>17</td>
<td>33</td>
</tr>
</tbody>
</table>

$X^2_1 = .82 \quad P = .20 \quad$ Not significant

The sexes were evenly represented in the different age groups, and the mortality between the sexes was almost the same; 40% for males and 40.7% for females. See figure 2.
Fig 2.

Figure showing Age and Sex Distribution

<table>
<thead>
<tr>
<th>AGE YEARS</th>
<th>0-10</th>
<th>10-20</th>
<th>20-30</th>
<th>30-40</th>
<th>40-50</th>
<th>50-60</th>
</tr>
</thead>
<tbody>
<tr>
<td>FEMALE</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MALES</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
There were 16 patients seen in 1984 and 31 patients seen in 1985. The 1984 mortality was significantly greater than the 1985 mortality vis 62.5% and 29% respectively. P=0.0339. Overall mortality for the 2 years of observation was 40.4% as seen in table 2.

**Table 2: Table of period of observation and mortality**

<table>
<thead>
<tr>
<th>YEAR</th>
<th>ALIVE</th>
<th>DEAD</th>
<th>TOTAL</th>
<th>MORTALITY</th>
</tr>
</thead>
<tbody>
<tr>
<td>1984</td>
<td>6</td>
<td>10</td>
<td>16</td>
<td>62.5</td>
</tr>
<tr>
<td>1985</td>
<td>22</td>
<td>9</td>
<td>31</td>
<td>29.0</td>
</tr>
<tr>
<td>TOTAL</td>
<td>28</td>
<td>19</td>
<td>47</td>
<td>40.4</td>
</tr>
</tbody>
</table>

P=0.0339  
Significant Relationship

Table 3 shows that many patients were from the Kikuyu as well as the Akamba and the Luo ethnic groups. The Somalis and Luhyas were also well represented. Many other communities were not represented.
### Table 3
**ETHNIC DISTRIBUTION OF THE PATIENTS**

<table>
<thead>
<tr>
<th>TRIBE</th>
<th>NO. OF PATIENTS</th>
<th>PERCENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kikuyu</td>
<td>20</td>
<td>42.2</td>
</tr>
<tr>
<td>Kamba</td>
<td>8</td>
<td>17.0</td>
</tr>
<tr>
<td>Luo</td>
<td>4</td>
<td>8.3</td>
</tr>
<tr>
<td>Somali</td>
<td>3</td>
<td>6.4</td>
</tr>
<tr>
<td>Luhya</td>
<td>3</td>
<td>6.1</td>
</tr>
<tr>
<td>Ugandan</td>
<td>2</td>
<td>4.2</td>
</tr>
<tr>
<td>Kalenjin</td>
<td>2</td>
<td>4.2</td>
</tr>
<tr>
<td>Masai</td>
<td>1</td>
<td>2.1</td>
</tr>
<tr>
<td>Kuria</td>
<td>1</td>
<td>2.1</td>
</tr>
<tr>
<td>Turkana</td>
<td>1</td>
<td>2.1</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td><strong>47</strong></td>
<td><strong>100</strong></td>
</tr>
</tbody>
</table>

**Table 4: TABLE OF PATIENTS BY PROVINCES OF REFERRAL**

<table>
<thead>
<tr>
<th>PROVINCES</th>
<th>NO. OF PATIENTS</th>
<th>PERCENTAGES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nairobi</td>
<td>13</td>
<td>27.7</td>
</tr>
<tr>
<td>Central</td>
<td>12</td>
<td>22.5</td>
</tr>
<tr>
<td>Rift Valley</td>
<td>11</td>
<td>23.4</td>
</tr>
<tr>
<td>Eastern</td>
<td>7</td>
<td>14.9</td>
</tr>
<tr>
<td>North Eastern</td>
<td>2</td>
<td>4.2</td>
</tr>
<tr>
<td>Coast</td>
<td>2</td>
<td>4.2</td>
</tr>
<tr>
<td>Nyanza</td>
<td>0</td>
<td>0.0</td>
</tr>
<tr>
<td>Western</td>
<td>0</td>
<td>0.0</td>
</tr>
</tbody>
</table>

Table 4 shows the provinces from which patients were referred.
The duration of hospital stay ranged from 1 to 80 days. The mean was 20.1 days. Most patients who died, died within the first 5 days of admission. No death occurred after a stay over 30 days in the wards. Most survivors spent 11 to 30 days in the hospital as seen in figure 3. There is a significant relationship between duration of stay and outcome, \((P=0.0001)\)

Fig 3. Figure of Duration of stay in Hospital Related to outcome.
Causes of Acute Renal Failure

The causes of ARF among the 47 patients studied are shown in table 5. 21 patients seen came with medical problems. Of these, the most frequently seen were acute glomerulonephritis and malaria, followed by pyelonephritis and cholera. Other causes included the hemolytic uraemic syndrome and hepatorenal syndrome. 33.3% of patients from medicine died, contributing to 36.8% of all deaths.

Mortality in paediatrics, 57.1%, contributing to 21% of all deaths. Obstetric problems were seen in 16 patients.

Post abortal sepsis and eclampsia, together with different causes of hemorrhage in pregnancy were the major causes. 31% had ARF related to abortion. These were associated with a mortality of 40%. All were due to post abortal sepsis following criminal abortion. There was no patient with ARF due to hemorrhage during abortion. 11 patients had ARF in the second half of pregnancy, and these had a mortality of 36.4%. The mortality in Obs/gynae was 42.9% and these contributed to 31.6% of all deaths. Four of the 16 patients from obs/gynae developed ARF while on treatment at KNH. Two of these died and two survived. 12 patients were referrals from peripheral health units. Four of these died and eight survived. Thus 66.71% of the deaths in obstetrics and gynaecology were among patients referred from outside Kenyatta National Hospital.

A notable surgical cause of ARF was a case of snake bite, referred from Turkana district. The mortality in
surgery was 66.71%, though the numbers seen was small. The surgical deaths contributed to 10.6% of all deaths.
Table 5: Aetiological factors in ARF

<table>
<thead>
<tr>
<th>Specialty</th>
<th>Disease</th>
<th>No. of patients</th>
<th>Alive</th>
<th>Dead</th>
<th>% of all Deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medicine &amp; Paediatrics</td>
<td>Acute Glomerulonephritis</td>
<td>9</td>
<td>6</td>
<td>3</td>
<td>15.7</td>
</tr>
<tr>
<td></td>
<td>Malaria</td>
<td>9</td>
<td>6</td>
<td>3</td>
<td>15.7</td>
</tr>
<tr>
<td></td>
<td>Cholera</td>
<td>2</td>
<td>0</td>
<td>2</td>
<td>10.5</td>
</tr>
<tr>
<td></td>
<td>Pyelonephritis</td>
<td>3</td>
<td>3</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Haemolytic Uraemic syndrome</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>5.3</td>
</tr>
<tr>
<td></td>
<td>Gastroenteritis</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Drug Reaction</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Transfusion reaction</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Obstetric and Gynaecology</td>
<td>Hepatorenal syndrome</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>5.3</td>
</tr>
<tr>
<td>Obstetric and Gynaecology</td>
<td>Typhoid</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>5.3</td>
</tr>
<tr>
<td>Obstetric and Gynaecology</td>
<td>Cardiogenic shock</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>5.3</td>
</tr>
<tr>
<td>Obstetric and Gynaecology</td>
<td>Post abortal sepsis</td>
<td>3</td>
<td>1</td>
<td>2</td>
<td>10.5</td>
</tr>
<tr>
<td>Obstetric and Gynaecology</td>
<td>Eclampsia</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>5.3</td>
</tr>
<tr>
<td>Obstetric and Gynaecology</td>
<td>Placenta praevia</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>5.3</td>
</tr>
<tr>
<td>Obstetric and Gynaecology</td>
<td>Pre eclampsia</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Obstetric and Gynaecology</td>
<td>Post caeserian section sepsis</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>5.3</td>
</tr>
<tr>
<td>Obstetric and Gynaecology</td>
<td>Abruptio Placenta</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>5.3</td>
</tr>
<tr>
<td>Obstetric and Gynaecology</td>
<td>Litra caeserian section shock</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Obstetric and Gynaecology</td>
<td>Retained Placenta</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Obstetric and Gynaecology</td>
<td>Post Partum Hemo-</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Obstetric and Gynaecology</td>
<td>orrhage of undetermined cause</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Surgical</td>
<td>Perforated Duodenal ulcer</td>
<td>2</td>
<td>0</td>
<td>2</td>
<td>10.5</td>
</tr>
<tr>
<td>Surgical</td>
<td>Snake Bite</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>TOTAL</td>
<td></td>
<td>47</td>
<td>28</td>
<td>19</td>
<td>100</td>
</tr>
</tbody>
</table>
The case fatalities for diseases which had more than 2 patients is shown in table 6. Though the number of patients are few, post abortal sepsis is shown to have a high case fatality. The mortality for type of acute renal failure is also shown in table 6. Acute renal failure due to intrinsic renal lesions was the most frequent, but pre-renal causes had a significantly high mortality \( P=0.912 \).

Table 6: Mortality by Disease and by Type of ARF.

<table>
<thead>
<tr>
<th>DISEASE</th>
<th>ALIVE</th>
<th>DEAD</th>
<th>TOTAL</th>
<th>MORTALITY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute Glomerulonephritis</td>
<td>6</td>
<td>3</td>
<td>9</td>
<td>33.3</td>
</tr>
<tr>
<td>Malaria</td>
<td>6</td>
<td>3</td>
<td>9</td>
<td>33.3</td>
</tr>
<tr>
<td>Pyelonephritis</td>
<td>3</td>
<td>0</td>
<td>3</td>
<td>0.0</td>
</tr>
<tr>
<td>Post abortal sepsis</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>66.7</td>
</tr>
<tr>
<td>Eclampsia</td>
<td>3</td>
<td>0</td>
<td>3</td>
<td>0.0</td>
</tr>
<tr>
<td><strong>TYPE OF ARF</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intrinsic Renal</td>
<td>24</td>
<td>12</td>
<td>36</td>
<td>33.3</td>
</tr>
<tr>
<td>Pre-renal</td>
<td>4</td>
<td>7</td>
<td>11</td>
<td>63.7</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>28</td>
<td>19</td>
<td>47</td>
<td></td>
</tr>
</tbody>
</table>
CLINICAL FEATURES

The period from onset of the disease process causing acute renal failure, and the onset of acute renal failure, varied considerably, depending on disease process. The range was 0 - 15 days, and the mean was 2.8 days. Acute renal failure started on the same day as the aetiologic disease process in 34% of patients. 46.8% of patients had a period lasting between 1 and 5 days. The interval was shortest in paediatric patients (mean 1 day) and longest among surgical patients (mean 5.5 days). The mean duration was 2.15 days for medical patients and 3.8 days in obstetrics and gynaecology patients.

The period of oliguria also depended on the cause of ARF. The mean was 13.4 days and the range was 2 to 36 days. The longest period of oliguria, was found in a patient who had abruptio placenta, and the shortest was found in a patient who had transfusion reaction as a cause of the ARF. Patients who survived had a mean duration of oliguria of 13.4 days, with a range of 3-32 days. All patients who died, had had an average duration of oliguria of 7.3 days, with a range of 2-32 days. 5.45% of patients had a period of oliguria lasting between 2 and 10 days. Figure 4 shows the period of oliguria and associated mortality.

Only 2 patients (4.2%) developed Non-oliguric ARF. One had acute glomerulonephritis, and the other had mitral valve regurgitation due to rheumatic heart disease.
Graph of period of oliguria vs number of patients.

Fig 4
The most common clinical signs were palor, 70.2% dehydration 53.2% edema 59.6% and kussmaul breathing 51.1%. Less common were uraemic frost and jaundice 21.3% and 19.1% respectively. Most patients who died had palor dehydration and kussmaul breathing. However, the signs in the presence of which death was most likely to occur, were uraemic frost and jaundice while the signs in the presence of which death was least likely to occur was edema, followed by fever. Table 7 shows the frequency of clinical signs.

Table 7: Frequency of clinical signs

<table>
<thead>
<tr>
<th>Clinical signs</th>
<th>No. of patients</th>
<th>Percentage of all Pt.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Palor</td>
<td>33</td>
<td>70.2</td>
</tr>
<tr>
<td>Edema</td>
<td>28</td>
<td>59.6</td>
</tr>
<tr>
<td>Dehydration</td>
<td>25</td>
<td>53.2</td>
</tr>
<tr>
<td>Kussmaul breathing</td>
<td>24</td>
<td>51.1</td>
</tr>
<tr>
<td>Fever</td>
<td>22</td>
<td>46.8</td>
</tr>
<tr>
<td>Uraemic frost</td>
<td>10</td>
<td>21.3</td>
</tr>
<tr>
<td>Jaundice</td>
<td>0</td>
<td>19.1</td>
</tr>
</tbody>
</table>

Most patients 55.3% had 3 to 4 signs. 21.3% of patients had less than 3 signs and 23.4% of patients had more than 4 signs. Mortality was not significantly related to the number of signs a patient had. Patients who had more than 4 signs had a mortality of 27% while those who had less than 3 signs had a mortality of 44%. Patients who had 3-4 signs had a mortality of 46%.
Complications were fairly common, and only 3 patients did not manifest any complication. Most patients, 53.2% had 4 to 6 complications and 48% of them died. 12.8% of patients had greater than 6 complications and this group had a mortality of 33%. Of the patients who had less than 4 complications, (34.0%), the mortality was 31%.

48.9% of patients (23) had pulmonary oedema of whom slightly less than half died (43.5%). Of the patients who developed pulmonary oedema, 65.2% also had oedema of the pre tibial region. The presence of hypertension did not correlate with the occurrence of pulmonary oedema neither did the highest level of BUN in these patients. Thus the highest level of BUN in patients who developed pulmonary oedema was 37.3 umol/l, while in patients who did not develop it was 40.5umol/l. About 68.4% of patients who survived developed pulmonary oedema. This complication was most frequent in obstetric and gynaecological patients (56.3%) followed by Medical patients and Paediatric patients (46.4%). Only 1 of the 3 surgical patients, 33.3% developed it. The frequency of complications is shown in table 8.

<table>
<thead>
<tr>
<th>Complications</th>
<th>Total No.</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infection</td>
<td>31</td>
<td>66.0</td>
</tr>
<tr>
<td>Neuropsychiatric</td>
<td>30</td>
<td>63.8</td>
</tr>
<tr>
<td>Pulmonary oedema</td>
<td>23</td>
<td>48.9</td>
</tr>
<tr>
<td>Bleeding Diathesis</td>
<td>18</td>
<td>38.8</td>
</tr>
<tr>
<td>Pulmonary Embolism</td>
<td>4</td>
<td>8.5</td>
</tr>
<tr>
<td>Pericarditis</td>
<td>1</td>
<td>2.1</td>
</tr>
</tbody>
</table>
Bleeding diathesis was manifested mostly as gingival bleeding, hematemesis, undue per-vaginal bleeding, frank haematuria, ecchymoses and melaena stool. It was detected in 18 (38.8%) patients. There was little difference in the mean of the highest level of BUN between patients who developed bleeding diathesis (40.0 mmol/l) and patients who died did not develop it (35.5 mmol/l). 47.4% of patients who died did not develop it, while 32.1% who lived, had it.

Clinically evident gastrointestinal bleeding occurred in 7 patients (14.9%) of whom 4 died (57.1%).

Pericarditis was found in 1 patient who had mitral regurgitation due to Chronic rheumatic heart disease. The patient died.

Neuropsychiatric complications were fairly common occurring in 30 (63.8%) patients. These included confusion seizures, asterixis delirium, hallucination, agitation and myoclonus. Half of these patients died and half survived. 78.9% of patients who died had this complication and 53.6% of survivors had it. The highest level of BUN in those who developed it (59.9 mmol/l) was not different from the level in those who did not develop it. Obstetric and gynaecology had the highest number of patients with neuropsychiatric manifestation 75%.

The incidence of pulmonary embolism as a complication of ARF was low occurring in 4 (8.5%) patients.

A high percentage (66%) of patients developed infection during the course of the illness or was associated with the cause of ARF.
The highest rate was found in obstetric and gynaecology (87.5%) followed by medicine, paediatrics (46.9%). All patients from surgery developed infection.

The frequency of infection and associated mortality are seen in table 9. The most frequent infection was urinary tract infection, though it was associated with a low mortality of 23.6%. Pneumonia was fairly frequent, and was associated with a high mortality, as was septicaemia, in both of which half of the patients died. The occurrence of peritonitis pelvic inflammatory diseases, or pelvic abscess, and wound sepsis, were not associated with a high mortality among those affected.

Table 9: Frequency of infection and Mortality associated with infections

<table>
<thead>
<tr>
<th>Infection</th>
<th>Frequency</th>
<th>No. Dead</th>
<th>% Dead</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urinary infection</td>
<td>14</td>
<td>4</td>
<td>23.6</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>12</td>
<td>6</td>
<td>50.0</td>
</tr>
<tr>
<td>Septicaemia</td>
<td>10</td>
<td>5</td>
<td>50.0</td>
</tr>
<tr>
<td>Peritonitis</td>
<td>10</td>
<td>2</td>
<td>20.0</td>
</tr>
<tr>
<td>Wound sepsis</td>
<td>9</td>
<td>2</td>
<td>22.2</td>
</tr>
<tr>
<td>Pelvic inflammatory disease/Ab.</td>
<td>6</td>
<td>1</td>
<td>16.6</td>
</tr>
<tr>
<td>Upper respiratory tract infection</td>
<td>6</td>
<td>1</td>
<td>16.6</td>
</tr>
<tr>
<td>Typhoid</td>
<td>2</td>
<td>0</td>
<td>0.0</td>
</tr>
</tbody>
</table>

Peritonitis only occurred in patients on peritoneal dialysis. Pelvic abscesses and pelvic inflammatory diseases
were found in post abortal sepsis and patient in puerperium.

Among the obstetric and gynaecology patients, pneumonia was most frequent, followed by pelvic inflammatory disease, then septicaemia and urinary tract infection. In medicine and paediatric, the common infections were urinary tract infection, wound sepsis and peritonitis. Two cases of typhoid were found, one in a patient who also had Malaria as a cause of the ARF and in the other, S. Typhinirium was cultured in an obstetric patient who had been in the ward for sometime, and could have been hospital acquired.

35 patients had their urine tested for culture and sensitivity and 9 (25.7%) had positive results. The most common organism found was Klebsiella species 7 (77.8%). There was 1 case in which E. Coli was isolated and in 1 case there was a mixed growth. 14 patients had symptoms of urinary tract infection. Pyuria was encountered in 20 patients. The patients with clinical features of urinary tract infection, yet had negative urine culture results had had antibiotics already during their hospital stay before the collection of the urine specimen.

31 patients had their blood cultured. Only 3 of these (9.7%) had positive culture. Patients who were diagnosed clinically as having septicaemia were 10. The anomaly between clinical diagnosis and culture positively could again be due to use of antibiotics before blood specimens were taken for culture. The septicaemia was in
several instances acquired during the hospital stay. 30% of patients with clinically diagnosed septicaemia, were culture positive. *S. Typhimurium* was isolated in 2 patients, and *Klebsiella* species in 1 patient.

Peritonitis was encountered clinically in 10 patients all of whom had been on peritoneal dialysis. A total of 15 patients had peritoneal dialysis as a mode of therapy, hence the infection rate was 66.7%. The clinical diagnosis was based on the presence of cloudy dialysate, abdominal tenderness, leucocytosis, and infected insertion catheter/site. Symptoms to go by included presence of abdominal pain, fever and catheter blockage. Only 4 of these patients were bacteriologically proven by a positive dialysate culture. The organism isolated were *Klebsiella* species (2), *E. Coli* (1) and mixed growths were encountered in 1 case.

**LABORATORY INVESTIGATIONS**

The mean of the highest value of BUN obtained in all patients was 43.2 m Mol/l and the range was 17-80 m Mol/l. 30 patients had their values measured. The remaining 17 patients did not have exact values measured and a figure of greater than 25 mMol/l was what was reported. There was no significant relationship between the highest level of BUN found in patients and mortality, from this study. \( \chi^2 = 0.97 \).

Most patients (19) 40.4% had values of BUN lying between 20.1 and 50 mMol/l. 4 patients had BUN values of greater than 70.1 mMol/l, and 3 of them survived.
A higher level of the highest BUN value obtained in a patient, however is shown in Figure 5 to be associated with a longer stay in the ward for survivors. No relationship can be made between the highest level of BUN and duration of stay in the ward before death for non-survivors.

Figure 5: Showing mean duration of stay in the ward in relation to highest BUN level and outcome.
The mean of the highest serum potassium values found in patients was 6.0 mmol/l, with a range of 3.8 to 10.0 mmol/l. 

41 patients had their serum potassium values assessed. The remaining 6 could not be assessed because they died before samples could be taken, and no serum potassium values had been taken in the hospital from which they had been referred due to technical reasons. 19 patients (46.3%) had serum potassium values of greater than 6.0 mmol/l and 22 patients (53.7%) had values less than 6.0 mmol/l of which 18.2% died as compared to a mortality of 57.8% among patients who had a serum potassium level of greater than 6.0 mmol/l. This difference is statistically significant P=0.0115 and mortality is related to the highest serum potassium level recorded. Almost all patients who had serum potassium levels greater than 8.1 mmol/l died. Figure 6 shows the outcome of patients in relationship to the highest potassium level obtained.

Figure 6: Figure of outcome in relation to highest K⁺ level obtained.

![Figure 6: Figure of outcome in relation to highest K⁺ level obtained.](image-url)
Malaria was encountered in 9 patients as a cause of acute renal failure. 4 of them died, hence a mortality of 44.4%. Four of the patients had deep jaundice. Liver function tests were measured in all the patients, and the values of SGOT, SGPT, Alkaline phosphates, and serum bilirubin, were moderately elevated in the 4 patients who had jaundice. Urinalysis was not remarkable in the patients who had malaria, except for 3 patients who had concurrent urinary tract infection, all of whom had proteinuria, pyuria and haematuria. The mean haemoglobin for the patients who had malaria was 9.0g% with a range of 6.8g% to 10.2g%. Peripheral blood film examination showed polychromasia in the 4 patients with jaundice, reflecting hemolysis. In the remaining 5 patients, the peripheral blood film was normal. The mean period from onset of malaria to onset of oliguria was 2.9 days with a range of 0 to 7 days, and a mode of 2 days. The mean period for survivors was 9.8 days. The range being 4 to 20 days. The mean duration of diuresis for survivors was 11.6 days, ranging from 10 to 15 days.

4 patients with malaria were treated by peritoneal dialysis, and of these, 3 survived. No patient had hemodialysis. The mean duration of peritoneal dialysis was 11 days with a range of 6 to 25 days.

Haemoglobin values were assessed in 35 patients, 23 females and 12 males. It could not be assessed in the remaining 12 patients due to technical reasons. 33 patients (70.2%) were clinically pale. 19 of the 23 females
assessed had haemoglobin values less than 11g% and 11 of the 12 males assessed had hemoglobin values less than 12.6g%. Thus 30 patients were anaemic. Of these, 12 died (40%). The mean haemoglobin value at admission was 9.1g% with a range of 5.3 to 16.2g%. There was a significant drop in mortality as haemoglobin values increased. 

\[ X^2_1 = 4.25, \, P=0.05. \] However, as death was not due to a single cause in the patient, this finding should be seen in its right perspective. No patient with haemoglobin values at admission of greater than 11.1g% died. More females had low haemoglobin values than males. These features are seen in figure 7.

Figure 7: Figure of Haemoglobin at Admission Vs outcome by sex.

![Figure of Haemoglobin at Admission Vs outcome by sex.](image)
Peripheral blood films were examined in 23 patients. 9 patients (39.1%) had normal films. Another 9 had hypochromia, anisocytosis and poikilocytosis reported. 2 patients (8.7%) had polychromasia and 1 patient had pancytopenia. Platelets were adequate in all 23 patients examined, except the one who had pancytopenia.

Random blood sugar was tested in 30 patients and all had normal values. There was no case of diabetic Ketoacidosis as a cause of acute renal failure, and there was no case of acute renal failure occurring in diabetic.

TREATMENT

Hemodialysis was carried out in 10 patients. These were very sick hypercatabolic patients who had the following conditions:

1. Acute renal failure following caeserian section complicated by DIC and a transfusion reaction was suspected.
2. Post abortal sepsis (2 patients)
3. Eclampsia
4. Hypovolaemic shock at
5. Abruptio placentae (2 patients)
6. Severe Gastroenteritis with shock
7. Pyelonephritis
8. Cerebral Malaria

8 of these had gynaecological problems, 2 had medical problems. The mean duration of HD was 15 days, with a range of 5-30 days.
6 patients had hemodialysis alone, and the remaining 4 had hemodialysis following an initial attempt of peritoneal dialysis. This was because of the following reasons:

1. Patients very sick and in a catabolic state.
2. Presence of complications like pelvic disease.
3. Results of HbsAg being awaited before hemodialysis could be started.
4. Absence of vessel tips or concentrate for prompt institution hemodialysis, hence peritoneal dialysis was used while these were being sought.
5. Presence of peritonitis or pulmonary edema.

The mean BUN at a start of hemodialysis was 32.3 mmol/l.

All the patients had HbsAg negative serum, except 1 patient who had AON as cause of the acute renal failure.

The number of patients who died while on hemodialysis was 2. Thus mortality with hemodialysis was 20%. Mortality with peritoneal dialysis was thus 26.7%. The number of patients who had no form of dialytic therapy was 25. Of these, the number of patients who died was 13. This gives a mortality without dialysis of 52%. The mean duration of peritoneal dialysis alone was 12.9 days with a range of 5-26 days. The total number of cycles of peritoneal dialysis done alone was 129.8 cycles while the range was 42 to 349 cycles. The mean level BUN at which peritoneal dialysis was started was 47.6 mmol/l. The range was 20 to 86.5 mmol/l. The mean level of serum potassium at
which peritoneal dialysis was started was 6.3 mmol/l and the range was 5.0 to 8.4 mmol/l. The mean duration of conservative treatment alone in survivors (i.e. survivors who did not require dialytic therapy) was 22.1 days, with a range of 7.51 days.

In patients who eventually required dialysis the mean duration of conservative management was 7.7 days, with a range of 1-47 days.

Peritoneal dialysis was started at relatively high levels of blood urea nitrogen and potassium values.
DISCUSSION

This series is composed of 47 patients who had acute reversible renal failure, from causes spanning all disciplines of medicine, during the period from January 1984 to December 1985.

Both sexes are represented in this study, in almost equal proportions of 44.7% and 55.3% for males and females respectively. The age range of 20-40 years is the group most frequently affected, with a mean age of 27.8 years. Studies carried out in the developed countries show higher mean ages, with the highest mean age being found in the surgical group. Thus A.C. Kennedy et al. in 1972 found a mean age of 43 years in his study of 251 cases of Acute Renal Failure. Our younger mean range could be explained on the age distribution in our society that tends to be much younger than in the developed countries. There is a major contribution of obstetric and gynaecology as well as paediatric causes that tend to have younger age groups.

The mortality in the age group 0-10 years is the highest 57.1%. The poor representation of the surgical specialty also contributes to the lower mean age as this group have the highest mean age in most series.
The overall mortality rate was 40.4%. This figure agrees well with figures reported by authors who/attended to larger numbers of patients, thus L.W. Blueve et al. in his analysis of 100 cases of acute renal failure in 1959 had a mortality rate of 50% while A. Kennedy et al., had a mortality rate rising from 35% to 50% between the years from 1968 to 1970. The mortality rates in large series of patients range from 30 to 60%. The mortality rates are highest in postoperative or traumatised patients (50-70%) intermediate in patients with acute renal failure encountered in a medical setting (30-50%) and lowest in acute renal failure observed in the obstetric setting (10-20%). Our figures showed a relatively high mortality of 37.5% in the obstetric setting. This enviable mortality in developed countries in acute renal failure due to obstetric causes could be due to the excessive/preoccupation with maternal mortality in obstetrics, leading to early referrals to renal units in situations of acute renal shut down. In our series obstetrics and gynaecology had a high specialty death rate of 37.5%. We can attribute this to late referral of patients from peripheral health units, as most deaths in obstetrics were of patients referred from outside Nairobi. This in turn could be due to poor transport facilities and possibly, also lack of awareness of the need for early referral by the doctors manning these units. This is an area that
could be improved on by distributing more obstetric specialists to the peripheral units. Most patients who died in obstetrics were from peripheral health units, and not Kenyatta National Hospital. This constituted 66.7% of deaths in obstetrics. 12 out of the 16 obstetric patients (75%) came from peripheral health units, and the remaining 4 (25%) came from Kenyatta National Hospital.

The mortality in a medical setting was 37.9% (i.e. 31.8% for Internal medicine and 57.1% for paediatrics), this fits well within the 30-50% mortality range found in large series.

In a survey over a 1 year period in 1968-1969, at Mulago Hospital the mortality rate was 79.2%. Ejogwu et al found a mortality of 82% in surgical patients, 61% in medical patients and 17% in obstetrics patients.

A distinctive feature of our series is the low mortality rate found in 1985 of 29% compared to the high mortality in 1984 of 68.8%. This may be attributable to the fact that the establishment and operation of our renal unit became well known in 1985 when renal transplantations were effectively commenced. Referral from peripheral health units, before that were of only very sick patients, who fared badly on admission, hence the high mortality. After our unit had been popularised, referrals were fairly early with effective therapy, hence the lowered mortality. However, a larger series could give a true picture of the mortality rate in the Kenyatta National Hospital renal unit.
Kenyatta National Hospital has the only renal unit in the country. Most patients seen in this series came from areas close to Nairobi, i.e. Kiambu, Machakos, Nyeri and Nakuru, and the ethnic groups managed most frequently were Kikuyu 42.2%, Kambas 17%, and Luos 8.3%. There were no referrals from such vasts population groups as found in Western Kenya, and only 2 patients were referred from the Coast. People who originate from these provinces however, were referred from provinces like Nairobi.

CAUSES OF A.R.F

Malaria due to plasmodium falciparum was an important cause of acute renal failure in this series being a likely cause in 9 medical patients (31%).

Acute renal failure due to a transient reversible glomerular lesion caused by plasmodium falciparum has been described. More commonly however, ARF occurs as a result of tubular pathology caused by P. falciparum. It is usually associated with acute intravascular hemolysis or heavy parasitic infection.

Intravascular hemolysis may be induced by malarial infection or by antimalarial drugs in patients with or without G6PD deficiency. Quinine, chloroquine and pyrimethamine have been incriminated. One mode of presentation is the black water fever. This is a syndrome of severe hemoglobinuria and haemoglobinaemia occurring in association with malaria due to plasmodium falciparum.
Black water fever can be diagnosed only when drug-induced hemolysis is excluded. It need not be associated with quinine or G6PD deficiency as was previously believed. It rarely occurs in the adult indigenous inhabitants of holo-endemic areas. It is seen most frequently in immigrants to an endemic malarious area. There probably is a state of sensitivity that results from partial loss of immunity which causes a sharp hemolysis upon re-infection with P. falciparum. Parasites are usually scanty in the peripheral blood film. Patients with high fever and numerous parasites usually do not develop the Black water fever.

Heavy P. falciparum infection is a more common cause of A.R.F. The patient is acutely ill with fever, and develops a hyper catabolic type of ARF. There is cholestatic jaundice, and mild intravascular hemolysis may be present. ARF may take a few days to several weeks. Non oliguric ARF is not common.

Disseminated intravascular coagulation has been documented in severe malaria, and may play a role in the pathogenesis of renal failure in malaria, though this has not been conclusively verified.

Other factors contributing to the pathogenesis of ARF in P. falciparum infection include hypovolaemia due to increased insensible loss of fluid, sweating, pyrexia, decreased fluid intake and increased capillary permeability due to Kinins. Significant hyperviscosity
of blood also occur and this compromises renal blood flow. The role of jaundice has also been entertained, as the association between renal failure and obstructive jaundice has been well established.

Salmonella typhimurium was isolated from blood in 2 patients. ARF due to salmonellosis has been described by D. Lwanga, who reported a patient in whom acute renal failure was a consequence of massive intravascular hemolysis due to G6PD deficiency. Chloramphenicol may be responsible for the hemolysis in some cases.

There was 1 case of hepatorenal syndrome due to viral hepatitis. The term hepatorenal syndrome was first used by Heyd in 1923 for the development of ARF after surgical relief of severe obstructive jaundice. Obstructive jaundice impairs liver function which in turn, may alter intrarenal hormonal balance in such a way as to impair renal perfusion. There is excess of Angiotensin II and deficiency of bradykinin and prostaglandine which cause a hepatorenal vasoconstriction in as yet unidentified manner. The term hepatorenal syndrome includes entities as primary liver disorders with secondary effects or the kidney e.g. cirrhosis, common disorders affecting the kidney and the liver, e.g. Carbon tetrachloride poisoning, and effects of renal disorders on the kidney, e.g. fatty degeneration of the liver in association with glomerulonephritis.
ARP resulting from herbal and patent remedies is a potentially life threatening condition. The ARP may occur in isolation, or be associated with other features as metabolic acidosis, hypercatabolism, intercurrent infection, DIC, and bleeding, jaundice and neurological abnormalities.

No patient had ARP due to ingestion of herbal medicine in our series, though from our experience this is a difficult fact to verify as most patients tend to hide the fact that they have had treatment with herbs, and patent remedies.

We had 1 case of hemolytic uremic syndrome. This is the coincidence of ARP, hemolytic anemia and thrombocytopenia. It occurs mostly in infants and children, but is also seen in adults. There is a prodrome of respiratory, digestive and systemic symptoms, followed by signs of acute renal failure, central nervous system injury, and gastrointestinal bleeding, there is pathological and biochemical evidence of intravascular coagulation.

Several other causes of ARP that were encountered in our study in the medical setting, have already been dealt with in the section of literature review.
Aetiologies of acute renal failure in obstetrics could be broadly categorised as those due to haemorrhage (42%), sepsis (29%) and toxaemia (29%) of pregnancy. Most of the patients were in late trimester (71%).

In a survey of 31 patients with acute renal failure of obstetrics, only 12 patients (29%) presented in the late trimester, and the mortality was lower than in patients who presented in early pregnancy in which septicaemia and hypovolaemia were the principal pathogenetic mechanism.

Clinical Features and Complications

Signs of severe disease were common and included uraemic, frost and jaundice, kussmaul breathing, and severe dehydration. Their presence should activate a concerted effort to initiate dialytic therapy with keen supportive care.

Neuropsychiatric abnormalities are seen in severe acute renal failure. These include, confusion, disorientation, asteresis, agitation, myoclonus, muscle twitching, and generalised seizures. They are caused by uremia per se, drug administration, metabolic and electrolyte abnormalities, and occasionally, by primary neurological disease. In this series, their presence was associated with a poor prognosis for the patients.
Infection has been shown in many series to be a poor prognostic indicator in acute renal failure. L.W. Blueme in 1959 found that it was the most frequent complication, and also the severest, accounting for more death than any other complication. 80% of patients manifested some evidence of infection in their series. Serious ones included septicaemia, pneumonia, peritonitis, tracheobronchitis, parotitis. In 1973, A.C. Kennedy, found that sepsis occurred in about 80% of the patients, and accounted for 40% of the deaths. Montgomerie et al (1968) in a very extensive review of the problem, found infection to be the commonest cause of death. A considerable proportion of the infection was acquired during the course of the hospital stay. In many series infection developed in 66% of the patients.

This enhanced susceptibility to infection in these patients is due to a variety of changes in leukocyte formation and function. Lymphocytopaenia and atrophy of lymph structures occur, whereas neutrophil production is unimpaired. Leucocytes have decreased chemotaxis with resulting impairment of acute inflammatory response, and decreased delayed hypersensitivity. Patients have less fever in response to infection, hence infection may be more difficult to recognise. Coexisting factors as acidosis may further impair leucocyte function. Dialysis may increase further portals of entry of organisms, as vascular access devices. The underlying illness may also lower resistance to infection. Prophylactic
antibiotics increase the frequency of infection.

The high infection rate in our study was associated with a high mean total white blood cells of $10.3 \times 10^9/\text{l}$. Most of these were neutrophils which had a mean of 71.9% while lymphocytes had a mean of 24.7%.

**LABORATORY FEATURES**

Hyperkalaemia due to decreased renal excretion of potassium, occurring with continued tissue potassium release is a frequent accompaniment of acute renal failure and has grave consequences if left unmanaged. In this study, hyperkalaemia is significantly related to and may be a contributor to mortality, among other causes of the latter. All patients who had maximum $K^+$ level of $\geq 3.1 \text{ mmol/l}$ died and 57% of patients who had $K^+$ level of $\geq 6.0 \text{ mmol/l}$ died, as compared to 13.2% in those with maximum potassium level of $<6.0 \text{ mmol/l}$.

In this study as in other studies, there was no significant relationship between the highest blood urea nitrogen obtained in each patient and mortality. A higher maximum level of BUN was however significantly associated with a longer stay in the ward for survivors.

A notable finding in this study is the high percentage of patients who had clinical anaemia (76.2%) and who were proven by investigation to have a haemoglobin of less than $11g\%$ (61.7). In the temperate countries, an important point of differentiation between
acute renal failure and acute on chronic renal failure is the presence of a normochromic normocytic anaemia in the latter, which indicates that the BUN has been persistently more than 100mg% or the creatinine clearance less than 25ml/ml, for some months. In the tropics, anaemia has a lot of other causes and its presence cannot be a criterion for making renal failure without underlying renal damage, an unlikely diagnosis.

Anaemia is a known complication in acute renal failure. There results a normochromic normocytic anaemia occurring following the onset of significant azotemia and the hematocrit usually stabilises between values of 20 and 30 volume percent. It is due to impaired erythropoiesis, as well as shortened red cell survival, other factors include hemodilution, gastrointestinal blood loss, suppressed erythropoiesis due to infection, dialysis and drug administration.

Peripheral blood films were taken in 23 of our patients and in 39.1% of these, there was a normochromic normocytic picture. In 39.1% there was hypochromia with poikilocytosis and anisocytosis. There was hypochromia and microcytosis in 8.7% and a similar percentage had polychromasia. An important contributor to anaemia in our patients could thus be blood loss, either related to the acute renal failure or unrelated. This is an area that requires further study because the diagnosis of acute renal failure in all our patients left very little room for doubt of the existence of an
underlying chronic renal failure, which was an exclusion criterion.

There was a significantly high mortality in association with low haemoglobin levels, thus 40% of patients who had anaemia died while there was no mortality in patients whose initial haemoglobin at admission was greater than 11g%. Causes of death however, were multifactorial and a study needs to be carried out to determine the significance of this finding.

A defect in haemostasis is a well recognised complication of acute renal failure, Swann and Merrill, 1953, and is a significant cause of mortality, when manifested as GIT bleeding etc. Factors that contribute to it include mild degrees of thrombocytopenia due to reduction in bone marrow platelet production. (In our patients there was 1 instance of thrombocytopenia in those who were tested). Other factors include qualitative defects in platelet function. The intravascular coagulation that commonly occurs in the course of acute renal failure and which in certain circumstances is the immediate mechanism leading to bilateral cortical necrosis. Plasma coagulation defects also occur. Bleeding diathesis occurred in 38.8% of our patients, half of whom died. Its contribution to mortality in our study is difficult to delineate however, as there were many other potential causes of fatality in these patients.
The management of the patients was based on the principles already presented in the review of literature. Dialytic therapy was considered in most of the patients, as they were very sick at the time of referral to the renal team. Many however, did not get such therapy because they died shortly after arrival, or the relevant apparatuses were lacking, and dialysis could not be started.

The results with dialytic therapy were quite encouraging. Of the 15 patients who had PD, only 4 died (26.7%), of the 10 patients who had Hemodialysis, only 2 died (20%). This is in comparison to a mortality of 52% among those who neither had hemodialysis nor peritoneal dialysis.

The incidence of clinical peritonitis in the 15 patients who had peritoneal dialysis was 66.7% and the organisms found were klebsiella and E.coli, in the 4 patients from whom dialysate culture was positive. Wairagu found a rate of 54.7% of peritonitis among 108 patients who had peritoneal dialysis at Kenyatta National Hospital, Klebsiella species accounted for 54.3% and staphylococcus aureus accounted for 22.6%. The incidence of peritonitis in the developed world ranges from 0-15%. Golper et al showed that occurrence of peritonitis was higher when glass bottles were used instead of plastic bags, to hold the commercially prepared dialysis fluid. With improvements aimed at reducing the rate of peritonitis, peritoneal dialysis should be the mainstay of therapy of Acute renal failure.
in the developing world where reaching renal units in which facilities for hemodialysis are available is a difficult task. Facilities for peritoneal dialysis should be made available to large provincial hospitals where trained personnel can be concentrated to manage these patients.

Hemodialysis was carried out in 10 patients, and peritoneal dialysis was done in 15 patients. Dialysis was started at relatively high levels of blood urea nitrogen and potassium of 47.6 and 6.3 mmol/l, respectively. In line with prophylactic dialysis in acute renal failure this should be at a lower level.

In this study, survivors who had active management in the form of dialysis stayed a shorter period in the ward than survivors who were not dialysed.
CONCLUSION

This is a prospective study of 47 consecutive patients who presented at Kenyatta National Hospital with acute renal failure, without a background of chronic renal failure, and were referred to the renal team for expert management. The patients were seen over a two-year period between 1st January, 1984 and 31st December, 1985. The study analyses the presentation of ARF, and attempts to relate several factors in the presentation to the outcome.

There were 20 males and 27 females, and their ages ranged from 6 months to 60 years. The mean age was 27.8 years. Most patients seen were between 20 and 40 years. The mortality within different age groups did not differ significantly (P=0.20). The sexes were evenly represented in the different age groups and mortality was not related to the sex of the patients. The mortality for 1984 was significantly higher than that of 1985 (P=0.0339). This was presumed to be due to earlier referrals to the renal unit in 1985, than in the previous year.

The major ethnic groups in Kenya were proportionately represented. Most referrals however, were from regions close to Kenyatta National Hospital.

The duration of hospital stay ranged from one to 80 days, with a mean of 20.1 days. There was a signi-
significant relationship between duration of stay in the ward and outcome \((P=0.0001)\) with most deaths occurring within the first 5 days of admission, and no death occurring after 30 days.

The important medical causes of Acute renal failure were acute glomerulonephritis and malaria. Others were pyelonephritis and cholera. Of the obstetric causes, post abortal sepsis and eclampsia, together with different causes of hemorrhage in late pregnancy, were the principle aetiological diseases. Most deaths in obstetrics were of patients referred from outside Kenyatta National Hospital.

Post abortal sepsis had the highest case fatality. Acute glomerulonephritis and malaria had low case fatalities, while no deaths were recorded with eclampsia and pyelonephritis. Surgical causes of ARF were few, though they had the highest mortality. This was followed by obstetric causes of ARF. Medical causes of ARF had the lowest mortality by discipline.

Although intrinsic renal causes of acute renal failure were the most frequent, pre-renal causes had a significantly higher mortality.

49 patients had oliguric ARF and only 2 patients had non-oliguric ARF. This however, may not reflect the true picture as patients with the latter condition may not have been referred to us as frequently as patients
with oliguric ARF. The mean duration of the oliguric and diuretic phases were found to be within ranges found in larger studies elsewhere. The period of oliguria was not found to be significantly related to mortality. All deaths occurred in the oliguric phase.

Signs of severe disease were common. Mortality was not related to the number of signs a patient had.

Complications were also fairly common, infection and neuropsychiatric complications being the most common. Others were pulmonary oedema and bleeding diathesis. Rare complications encountered were pulmonary embolism and pericarditis. The number of complications a patient had was not significantly related to mortality.

There was no significant relationship between the mean of the highest level of BUN obtained in the patients and mortality. A higher mean level of BUN value obtained was however related to a longer stay in the ward for survivors. The highest value of serum potassium recorded was found to be related significantly to mortality (P=0.0115). A significant inverse relationship also existed between the level of haemoglobin at admission and mortality (P=0.05).

Results with both peritoneal dialysis and hemodialysis were quite good, and mortality in patients who were dialysed were lower than for patients who had no form of dialysis.
RECOMMENDATIONS

1. Large provincial hospitals should be equipped with staff and materials to enable peritoneal dialysis to be carried out there.

2. Train physicians and nurses well versed in peritoneal dialysis, to manage patients with acute renal failure in large provincial hospitals as Mombasa and Kisumu. Only very sick hypercatabolic patients can then be referred to K.N.H. for hemodialysis.

3. Improve transportation services to all corners of the country to facilitate quick referral for dialytic therapy in large provincial hospitals or at K.N.H., for patients with ARF.

4. More obstetrics and gynaecology specialists should man peripheral hospitals to decrease maternal mortality from acute renal failure, mostly due to haemorrhagic causes.

5. Malaria is an important cause of acute renal failure and further study should be carried out on this subject.

6. Legislation on abortion should be reviewed to decrease the incidence of post abortal sepsis and its complication, including acute renal failure.
7. Dialysis should be started early in line with the concept of "prophylactic dialysis" to achieve better results.

8. A longer study on acute renal failure, covering a decade or more should be carried out to determine the trends of acute renal failure in our set up.
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PROFORMA
ACUTE RENAL FAILURE AT
KENYATTA NATIONAL HOSPITAL

1. Name Sex
   Age Tribe
   Place of Birth
   Occupation
   Residence Current
   Date of Admission
   Date of Discharge/Death
   Date of Renal Consultation
   Outcome.

2. Date of onset of oliguria
   Period from onset of aetiological
   Factor to onset of oliguria
   Period of oliguria
   Date of onset of diuresis
   Period of diuresis
   Period from onset of oliguria to
   referral to KNH.
   Period from onset of oliguria to
   consultation of renal team

3. Medical specialty referring the patient
   Disease preceding onset of ARF

4. Significant clinical signs.
   Dehydration Edema
   Kussmaul Breathing Fever
   Uraemic frost ascites
   Others

5. Complications
   Highest recorded Bp
   Duration of Hypertension
Pulmonary Edema

GIT Hemorrhage - Hematemesis
  Maleana stool
  Per rectal bleeding

Bleeding Diathesis? Site:

Pericarditis

Neuropsychiatric
  Delirium
  Depression
  Others

Fits

Hallucination

Pulmonary Embolism

Infection

Septicaemia

Pneumonia

Peritonitis - Before p/dialysis
  - with p/dialysis

wound sepsis

Anemia

6. Date of start of P/D
  Duration of P/A
  Total No of cycles P/D
  Level bun at which P/D started
  Date of start H/D
  Duration of H/D
  Bun at which H/D started
  Reasons for starting H/D
  HBs Ag.
  Duration of conservative Rx
  No of transfusions needed
  Level of Hb at which transfusion started
  Other treatment given
  Treatment of complications.
7. Dates | Bun | Na⁺ | K⁺
---|---|---|---

Hemoglobin
WBC - total
  - differential
Film report
Platelets
PTI

LFT's
Ser. Bilirubin
Urinalysis - Hematuria
  proteinuria
  pyuria
  others.

ultrasound
urine culture
blood culture
Peritoneal dialysate culture
Other specimens culture.