TO STUDY THE CLINICAL PROFILE, MANAGEMENT AND OUTCOME OF THE PATIENTS WITH POSTPARTUM RELATED ACUTE RENAL FAILURE -DESCRIPTIVE STUDY

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CERTIFICATE

This is to certify that this dissertation titled **'TO STUDY THE CLINICAL PROFILE, MANAGEMENT AND OUTCOME OF THE PATIENTS WITH POSTPARTUM RELATED ACUTE RENAL FAILURE'** submitted by **DR.V.SUNDARARAJ** to the faculty of General Medicine, The Tamil Nadu Dr.M.G.R. Medical University, Chennai in partial fulfilment of the requirement for the award of MD degree Branch I (General Medicine) is a bonafide research work carried out by him under our direct supervision and guidance.

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I, **Dr.V.SUNDARARAJ**, solemnly declare that the dissertation titled **'TO STUDY THE CLINICAL PROFILE, MANAGEMENT AND OUTCOME OF THE PATIENTS WITH POSTPARTUM RELATED ACUTE RENAL FAILURE'** has been prepared by me.

This is submitted to the Tamil Nadu Dr.M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of MD degree Branch I (General Medicine).

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CONTENTS

INTRODUCTION

1. INTRODUCTION

Acute Renal Failure (ARF) may be defined as a sudden decrease in renal function which is usually reversible, over a period of several hours to days sufficient enough to result in retention of nitrogenous waste products (e.g. blood urea nitrogen [BUN] and Serum Creatinine) and failure to maintain fluid and electrolyte homeostasis in the body.

Acute renal failure is the most challenging medical clinical problem when it occurs during post partum period. In recent years , the incidence of postpartum acute renal failure has decreased in developed countries but still continues to be common in developing countries.

Delay in diagnosis and late referral is associated with increased mortality. ARF is usually multifactorial in postpartum period and many factors can influence the outcome of ARF

All factors that can cause ARF in a nonpregnant woman can theoretically cause renal failure in a postpartum woman, including volume depletion, bleeding and sepsis. It is a rare complication of postpartum period with due to virtual disappearance of septic delivery and better puerperal care.

However, the care of women diagnosed with acute renal failure is a challenge for nephrologist and his team.

Pregnancy related acute renal failure is on the decline from 14.5% reported in 1987 to 4.3% in 2005 in India. The majority of pregnancy related acute renal failure cases were seen in the postpartum period around 75.61%

Acute renal failure is one of the most serious complications of pregnancy. ARF that is severe enough to require dialysis is quite rare in industrialized nations; its incidence is 1:20000 or less of all gestations. These statistics show significant improvement as compared to the situation in 1950s and 1960s when as many as 22% of all cases of acute renal failure were of obstetrical origin with mortality rate ranging from 20% to 48%.

This achievement in industrialized nations is most likely due to liberalization of 100% hospital delivery, improved postpartal care and better management of maternal complications potentially leading to ARF. The incidence of obstetrics related ARF in developing countries has not changed significantly.

There is no such local data available in the past to compare with. Only few scanty available articles which showed postpartum related ARF. Mortality in Obstetric ARF depends on underlying renal lesion and associated complications. It is high when associated with HELLP syndrome, acute fatty liver of pregnancy, HUS, Sepsis, DIC.

2. REVIEW OF LITERATURE

A noted professor of physiology once stated, “to say the function of the kidney is to produce urine is like saying the function of a steel mill is to produce slag”. While it true that the primary function of the kidney is the regulation of fluid volume, electrolyte and acid base status of the body, a certain amount of “slag” as urine must be produced daily to provide this regulation.

It requires an understanding of normal physiology of kidney during pregnancy and postpartum period and the natural history of different renal diseases when pregnancy occurs. During puerperium significant alterations are seen in renal blood flow and glomerular filtration, resulting changes in normal renal laboratory values when these normal renal adoption are coupled with postpartum induced complication or pre-existing renal dysfunction.

2.1. KIDNEY CHANGES IN PREGANCY AND POSTPARTUM

Both anatomical and functional changes occur in kidney during the period of pregnancy and postpartum.

2.1.1. ANATOMICAL CHANGES

The kidney size increases approximately 1 cm in length during normal pregnancy and postpartum period, it will return to normal size usually 2 to 3 weeks after delivery. More striking, however, are the anatomical changes in the calyces, renal pelvis and ureters, which dilate markedly, often giving the erroneous impression of obstructive uropathy. Ultrasound studies suggest that renal parenchymal volumes also increase during this period.

These anatomical changes have important clinical implications.

- Dilatation of the urinary tract may lead to collection errors in tests based on timed urine volume.
- Urinary stasis within the ureters may predispose to develop acute symptomatic pyelonephritis.
- Acceptable norms of kidney size should be increased by 1 cm if estimated during pregnancy or up to 3 weeks of postpartum period.
- Rarely the changes may be extreme and precipitate the ‘over distension syndrome’.

2.1.2. PHYSIOLOGICAL CHANGES

The hemodynamic changes affecting renal blood flow are coincident with and partially causative of some of the general cardiovascular changes of puerperium. Very early decreases in peripheral vascular resistance in pregnancy are due in large part to decreased renal vascular resistance that may be related to the effects of maternal hormones such as relaxin.

This arteriolar under filling is thought to lead to a systemic response including marked increases in cardiac output (approximately 50% above nonpregnant baseline) and plasma volume (approximately 40% above baseline).

There are also changes in glomerular filtration rate (GFR) and renal plasma flow. Both increase during the first half of pregnancy and subsequently level off, with increases on the order of 40–65% for GFR and 50–85% for renal plasma flow. Because GFR increases less than renal plasma flow (RPF) during early pregnancy, the ratio of GFR/RPF, the filtration fraction (FF) decreases. Late pregnancy and postpartum period is associated with an increase in FF to values similar to the non pregnant norm.

Creatinine clearance increases by 25% to 45% in early pregnancy, during the third trimester a consistent and significant decrease towards non pregnant values occurs preceding delivery, and daily measurements postpartum have suggested a small transient increase during the first few days of the puerperium.

Normal laboratory index in pregnancy and postpartum period

Index	Change and range in pregnancy	Changes and range in postpartum
GFR	Increase about 40% over baseline	Transient increase first few days
Creatinine clearance	Increase about 25% over baseline	Transient increase first few days
BUN	9 - 10 mg/dl	8-11mg/dl
Creatinine	0.5 – 0.8 mg/dl	0.5-1.0mg/dl
Urine protein	less than 300 mg in 24 hours urine	<500 mg in 24 h0urs urine
Plasma osmolality	decrease about 19 mosm/kg	Decrease about 8-10 mosm/kg

The ‘hyper filtration’ in these states is associated with sustained increases in glomerular blood pressure, and the glomerular hypertension is thought to play a primary role in the initiation of kidney damage.

There is certainly an increase in total protein excretion and a small increase in albumin excretion particularly during the third trimester and early puerperium - changes related to altered tubular and not glomerular function. A change in tubular function with increased glucosuria also occurs. In addition, a reset in the osmostat occurs, resulting in increased thirst and decreased serum sodium levels (by approximately 5 mEq/L) compared with nonpregnant females.

Similarly, in the initial part of pregnancy, increased levels of progesterone enhance relaxation of the arterial smooth muscles and thus decrease peripheral vascular resistance. Therefore, a blood pressure fall of approximately 10 mm Hg occurs in the first 24 weeks of pregnancy. The blood pressure gradually returns to a pre pregnancy level by term; thus, a consistent normal or pre pregnancy blood pressure may suggest the presence of a condition that predisposes patients to hypertension.

As a rule, all the physiologic changes maximize by the end of the second trimester and then start to return to the prepartum and early postpartum period, whereas changes in the anatomy take up to 3 months postpartum to subside.

2.2 ACUTE RENAL FAILURE IN POSTPARTUM PERIOD

ARF was diagnosed by documenting oliguria (urine output <400 ml/d) or mounting azotemia in the presence of normal urine output (Serum creatinine >2 mg %).

2.2.1. CAUSES OF ARF IN POSTPARTUM PERIOD

- PRERENAL AZOTAEMIA
  - ✓ Water and electrolyte depletion;

    Vomiting, Diarrhea, Diuretics.
  - ✓ Oedema-forming status;

    Postpartum eclampsia, Heart failure.
  - ✓ Postpartum hemorrhages.

- RENAL AZOTAEMIA
  - ✓ Acute interstitial nephritis;

    Pyelonephritis, Septicemia, Drugs.

- ✓ Acute tubular necrosis;

  Postpartum eclampsia

  HELLP syndrome

  Acute fatty liver

  Hemorrhages

  Drugs

  Septic shock

  Haemoglobinuria

  Prolonged intrauterine fetal death

  Amniotic fluid embolism

- ✓ Microangiopathic syndromes;

  Hemolytic uremic syndrome

  Thrombotic thrombocytopenic purpura

- ✓ Bilateral renal cortical necrosis

  Septic or hemorrhagic shock

➢ POSTRENAL AZOTAEMIA

- ✓ Obstruction;

  Nephrolithiasis

  Gravid uterus

  Tubular obstruction (uric acid)

  Broad ligament hematoma

PRE RENAL AZOTAEMIA

Functional renal failure is easy to recognize: the patient is oliguric, the urinary osmolarity is increased, the urine/plasma ratio for urea exceeds 10, and urinary concentration of sodium is usually less than 10mmol/l, implying that tubular reabsorptive function is preserved, differentiating it from acute tubular necrosis. The response to restoration of the blood volume is prompt. Blood loss, diarrhoea, and use of diuretics may also cause (or contribute to) functional renal failure. Prompt diagnosis and correction of volume depletion is essential.

ACUTE PYELONEPHRITIS

The incidence of acute pyelonephritis is increased in postpartum; it occurs in 1to 2 per cent of women without previous bacteriuria, and in up to 40 per cent of patients with untreated bacteriuria.

Acute pyelonephritis does not usually affect renal function in nongravidas, unless there is associated obstruction. By contrast, it may be particularly severe in pregnancy and puerperium. It is often associated with a marked decline in renal function and multiorgan failure. The incidence of ARF is relatively low.

Acute pyelonephritis accounts for 3to5 per cent of cases of ARF in postpartum women in a French study (***Grunfeld et al. 1980***), whereas it is reported as high as 45 percent in others (***Ventura et al. 1997***). Moreover, drug nephrotoxicity may be an additional factor in such patients.

HELLP SYNDROME

This syndrome was first described in 1982 (***Weinstein 1982***). It was originally thought to be a rare complication of severe pre-eclampsia, occurring in 4 to10 per cent of these patients (***Sibai 1990***). More systematic attention is paid to liver enzymes and platelet count showed that it is more frequent than initially believed, and that it can occur in the absence of hypertension and proteinuria (20 per cent of cases). Its significance is controversial, but it is undoubtedly associated with a poor prognosis.

The most frequent presenting symptoms are epigastric or right upper quadrant pain (65 per cent), nausea and vomiting (36 percent), and headache (31 percent). Visual changes, bleeding, jaundice, and diarrhoea are less frequent (***Sibai et al. 1993***). All these symptoms are relatively non-specific, so the disease may be misdiagnosed. Characteristic laboratory findings include mild haemolytic anaemia, thrombocytopenia of variable severity, a moderate increase of transaminases and lactic dehydrogenase,

while alkaline phosphatase and bilirubin are most often in the normal range. This allows clear differentiation from acute fatty liver.

In a series of 442 pregnancies complicated by this syndrome, the onset was ante partum in 70 per cent and postpartum in 30 per cent. There was no pre-eclampsia in 20 per cent of cases. Disseminated intravascular coagulation occurred in 21 per cent of cases, abruptio placentae in 16 per cent, and ARF in 8 per cent (***Sibai et al. 1993***). There was a strong correlation between abruptio placentae and ARF. Perinatal death occurred in 34 per cent of cases, and maternal mortality was 1.1 per cent. ***Selcuk et al. (2000)*** found the HELLP syndrome associated in 36 per cent of pregnancy-related ARF.

It is not clear whether ARF is a specific component of the HELLP syndrome itself, or a complication of a particularly severe multisystem condition. **Haddad et al. (2000**) found that disseminated intravascular coagulopathy is significantly associated with abruptio placentae and ARF.

In the same series, renal histology showed acute tubular necrosis in virtually all the patients with ARF and only one showed cortical necrosis. In subsequent pregnancies, patients who have had the HELLP syndrome have an increased risk of postpartum complications. The recurrence risk of the syndrome itself is low (3-5 per cent).

ACUTE FATTY LIVER

Acute fatty liver is a rare but life-threatening complication which occurs in the third trimester of pregnancy and early postpartum. Hypertension is present in 20 to 50 per cent of cases. Marked hyperbilirubinaemia with only minimally elevated transaminases are considered a hallmark of this disease, allowing differentiation from the HELLP syndrome and other hepatic diseases.

Pathological liver changes are characterized by microvacuolar fatty infiltration (steatosis) predominantly in the centrolobular areas. Ultrasonography and Computed tomography (CT) scans of the liver have been proposed as alternative diagnostic procedures. Their accuracy remains disputed.

The incidence of ARF in this condition is 60 per cent or more. Nevertheless, renal failure is usually mild and does not require dialysis. The high maternal mortality is due to hepatic failure. In the most recent series (***Castro et al. 1999***), gestational age at diagnosis ranged from 28 to 39 weeks, the disease occurring postpartum in a very few patients.

Almost all patients have renal impairment, but the serum Creatinine is not markedly increased, ranging from 97 to 317 µmol/ l and dialysis is not

required. Acute fatty liver of pregnancy does not usually recur in subsequent pregnancies. No recurrence was found by ***Watson and Seeds (1990***) in 21 patients with 25 pregnancies, but one case of recurrence was reported by ***Usta et al. (1994).***

The renal histopathological findings are not consistent. Kidney structure may be within normal limits, but in other cases there are focal lesions of acute tubular necrosis. Glomerular lesions or intraglomerular thrombi have occasionally been reported. A few patients have lesions quite similar to those observed in the haemolytic uremic syndrome.

The pathogenesis of ARF in acute fatty liver of postpartum is unexplained. Shock is not a constant finding, nor is pancreatitis. Acute fatty liver of postpartum exhibits some similarities with Reye's syndrome observed in children, but ARF is rare in this syndrome. Coagulation abnormalities suggestive of disseminated intravascular coagulation have often been reported. In fact, many of its features resemble the so-called ‘Hepatorenal syndrome’ described in association with various hepatic disorders, and hemodynamic changes may play the prominent role.

SEPTIC SHOCK

Septicaemia associated with postpartum is commonly due to pyelonephritis, chorioamnionitis and puerperal sepsis occur less frequently, but still it is common due to unsterile delivery.

There are many reasons why acute renal failure should be associated with puerperal septicaemia. The patient is both dehydrated and hypotensive – a combination that leads to considerable renal ischemia. Haemoglobinuria (due to haemolysis) and DIC are often present. Most of the puerperal sepsis are due to Gram-negative bacteria and clostridial infection.

The presentation can be quite dramatic, especially in case of E.coli and clostridia infection. There is an abrupt rise in temperature (up to 40 degree C) often associated with myalgias, vomiting and diarrhoea, the last occasionally bloody. There is laboratory evidence of severe anaemia due to haemolysis with hyperbilirubinaemia of the direct type. There are also alterations in clotting factors that suggest DIC. Leucocytosis and thrombocytopenia are observed. Hypocalcaemia, severe enough to provoke tetany, can also occur

The patient then develops dyspnoea, hypotension, and jaundice, followed by the shock syndrome. Many patients respond well to antibiotics and volume replacement but may go on to develop ARF. The period of

anuria may last 3 or more weeks, requiring dialysis and usual intensive care procedures. Although renal function often recovers completely, bilateral cortical necrosis is a not infrequent consequence

HAEMORRHAGE

Uterine haemorrhage and hypotension are directly responsible for acute tubular necrosis in some cases, the frequency ranging from 7 per cent to 39 per cent in one study (***Alexopoulos et al. 1999***).

However, it must be remembered that hypovolaemia due to haemorrhage is involved in many cases of ARF related to postpartum pre-eclampsia, HELLP syndrome. About 90 per cent of patients in ***Sibai's*** series had postpartum haemorrhage. Therefore, this factor is more often additional than causal.

HAEMOLYTIC UREMIC SYNDROME (IDIOPATHIC POSTPARTUM ACUTE RENAL FAILURE)

The first cases of so-called 'idiopathic postpartum renal failure' were reported in the 1960s. Since then, more than 200 cases have been described. In some series, this syndrome accounts for 5 to10 percent of pregnancy related acute renal failure. The disease occurs typically between

a few hours and 8 to 10 weeks postpartum, but delays of several months have been reported occasionally.

In most cases, pregnancy and delivery have been uneventful and most patients are multiparas. It begins with influenza-like symptoms, ARF developing after a few days. Moderate proteinuria and microscopic hematuria precede the development of anuria, which is usual. Hypertension develops in half of the cases. Microangiopathic anaemia occurs in most, but not all, cases. Numerous schizocytes are seen on the blood film; there is a high reticulocyte count, low haptoglobin concentration, and a low platelet count. The inconstancy of microangiopathic anaemia leads some authors to maintain the distinction between ***'idiopathic postpartum renal failure'*** and the typical haemolytic uremic syndrome.

Apart from anaemia and thrombocytopenia, other signs of deranged clotting include prolonged thrombin time, decreased fibrinogen, and an increase in fibrin degradation products, considered diagnostic of disseminated intravascular coagulation, but intra renal consumption and fibrinolysis have also been proposed.

Histopathological examinations reveal the involvement of small arteries, afferent arterioles, and glomeruli. The changes in arteries and arterioles include intimal swelling, suboptimal deposits, concentric intimal

fibrosis, and fibrin thrombi. Fibrinoid necrosis is prominent in severe cases. The glomerular tufts are enlarged, with endothelial swelling and large sub endothelial fibrin deposits giving a ‘double contour’ appearance. Deposits may lead to capillary obstruction, and occlusive thrombi are seen. The mesangial areas appear prominent.

The cause of idiopathic postpartum ARF remains unknown. Deficiency of a specific plasma protease responsible for the physiological degradation of von Willebrand factor seems to be involved in a limited number of cases (***Furlan et al. 1998***), contrary to thrombotic thrombocytopenic purpura. Various triggers have been described for the syndrome, pregnancy being only one of them.

Endothelial damage from release of bacterial endotoxins or vasoactive amines leading to consumptive coagulopathy has been suggested. Various viral infections and drugs have also been reported as triggers (***Elliott and Nichols 2001***). Some patients with late postpartum ARF were found to have been taking the oral contraceptive at the time of onset.

A role for such agents as triggers of the syndrome is quite possible since occurrence of the haemolytic uremic syndrome has also been reported in non-postpartum women patients taking the oral contraceptive. A generalized endothelial disorder is probably the key feature of this syndrome, as it is in other forms of pregnancy-related ARF.

Fresh plasma infusion or plasma exchange, with or without antiplatelet therapy are now considered the therapy of choice ***(Elliott and Nichols 2001***). Intravenous immunoglobulin has also been advocated. The clinical course is usually rapid. Other methods of treatments, including prednisolone, aspirin, dipyridamole, heparin, immunoglobulin, vincristine and splenectomy, should be considered only as adjuncts to plasma exchange.

The maternal mortality, formerly as high as 50 per cent (***Weiner 1987),*** but lower, has decreased since conservative treatment improved ***(Elliott and Nichols 2001)*** Renal failure was previously thought to be irreversible but ***Finkelstein et al. (1974)*** reported the first cases of milder and reversible renal failure, and complete or partial recovery was shown to occur in about 30 per cent of cases.

THROMBOTIC THROMBOCYTOPENIC PURPURA

In pregnant women, TTP occurs most often during the second or third trimester, with a mean of 23 weeks and its occurrence postpartum is very uncommon. The classic clinical picture is one of microangiopathic haemolytic anaemia, thrombocytopenia, neurological abnormalities, fever, and renal dysfunction. TTP has a high maternal and perinatal mortality, and the condition is not improved by delivery.

A specific von Willebrand factor cleaving protease (a zinc metalloprotease) has been isolated from normal human plasma, and severe deficiency of this protease has been found in most patients with TTP. Protease deficiency is mainly due to the presence of a circulating inhibitor, which has been reported to be an IgG antibody. In the rare congenital and familial forms, there is no inhibitor, but a constitutional deficiency of the enzyme, related to mutations in the ADAMTS13 gene (***Levy et al. 2001***).

Therapeutic measures proposed for the treatment of TTP are plasma exchange with fresh frozen plasma. Immunosuppressive therapy has been advocated on the basis of a protease inhibitory antibody.

BILATERAL CORTICAL NECROSIS

Bilateral renal cortical necrosis accounts for about 6 per cent of cases of ARF and for 20 to 30 per cent of pregnancy-related ARF. Patchy cortical necrosis, with complete or partial recovery of renal function, may be missed if investigations are incomplete. Bilateral cortical necrosis is most often a complication of abruptio placentae (36 per cent in the series of ***Chugh***), and all the other conditions associated with disseminated intravascular coagulation.

Bilateral renal cortical necrosis presents as acute renal failure in other conditions too but, unlike acute tubular necrosis, total and persistent anuria is almost constant. Ten per cent of patients had hypertension, 36 per cent had fluid overload, and 14 per cent had hyperkalaemia. The platelet count and plasma fibrinogen are lower in patients with cortical necrosis than in patients with acute tubular necrosis. The diagnosis can be established either by renal biopsy or, better, by selective renal angiographic scans, Magnetic resonance imaging (MRI) has been more recently reported to provide strongly suggestive evidence like lack of enhancement of the renal cortex, enhancement of the medulla, and poor or absent renal excretion on CT scan. Later, cortical calcification can be seen on standard radiographs or CT scans.

OBSTRUCTION

Renal tubular obstruction by uric acid has been reported in a few patients. Serum uric acid exceeded 700 mol/l and was unexplained, but dehydration contributed. Tubular deposition was attributed to an increased concentration of tubular fluid uric acid and acidic urine. Renal failure resolved after hydration, alkalinisation of the urine, and mannitol administration. Urethral obstruction by broad ligament hematoma after caesarean section delivery has been reported to cause acute renal failure in rare instance.

2.2.2. PATHOPHYSIOLOGY: UNIFYING CONCEPTS

Pathologically, the spectrum of postpartum related ARF includes:

1. Acute tubular necrosis in the setting of severe pre-eclampsia and/or HELLP syndrome.
2. Microangiopathic diseases, typically idiopathic postpartum renal failure, hardly distinguishable from the haemolytic uremic syndrome, and from TTP.
3. Bilateral renal cortical necrosis, which may be associated with any clinical picture.

Acute tubular necrosis is related to ischemia and/or vasoconstriction in the renal cortex, causing direct damage to proximal tubular cells. However, it has been shown recently that the medullary thick ascending limb of Henle may be more susceptible to ischaemic and/or hypoxic injury. Tubular insult is greatly increased by prostacyclin deficiency, low nitric oxide delivery, or excess endothelin production, which are hallmarks of the pre-eclampsia syndrome.

2.2.3. PHASES OF ACUTE RENAL FAILURE

Traditionally there are three consecutive phases and these have important management implication.

OLIGURIA

Urine volumes are < 400ml in 24 hours, this phase may last from a few days to several weeks. Complete anuria is uncommon in acute tubular necrosis and is usually a manifestation of massive acute cortical necrosis or complete obstruction. Non oliguric forms of acute cortical necrosis may be occasionally encountered in which urine volumes seem adequate but renal function is severely impaired.

POLYURIA

Urine volumes increase markedly and can be up to 10L in 24 h. the polyuria may last from several days to 2 weeks. The urine is dilute and, despite the large volumes excreted, metabolic waste products are not efficiently eliminated. Consequently plasma urea and creatinine levels will continue to rise for several days in parallel with increased urine output. Profound fluid and electrolyte losses can endanger survival if not adequately replaced.

RECOVERY

Urine volume decreases towards normal. Renal function gradually improves, nearing the level before the acute renal failure developed.

2.2.4. DIAGNOSIS AND INVESTIGATION

A carefully taken history may reveal a background of haemorrhage, sensitization to drugs or incompatible blood transfusion. Once the diagnosis has been entertained, the possible causes must be pursued and, in conjunction with a nephrologist, a full initial assessment should be undertaken.

The assessment should include the following;

1. Blood specimens; complete blood count, urea, electrolyte and osmolality, glucose, liver function tests, amylase, plasma proteins, coagulation indices and acid-base status.

2. Urine specimens; specific gravity osmolality, electrolyte concentrations and protein excretion

3. Bacteriological assessment; blood cultures(aerobic and anaerobic)

4. ECG; in hyperkalaemia, peaked T waves with QRS prolongation and then disappearance of P waves with deformation of QRS complex.

5. Assessment of fluid balance: The bladder should be catheterized and continuous drainage allowed so that hourly volumes can be recorded. A CVP line should be established.

6. Ultrasound abdomen

7. Renal biopsy

2.2.5. GENERAL MANAGEMENT

PRERENAL FAILURE OR VASOMOTOR NEPHROPATHY

The basic principle is to replace blood and fluid losses adequately and maintain blood pressure at levels that will permit adequate renal perfusion. Volume correction should always precede the use of diuretics.

VOLUME AND METABOLIC CONTROL

Fluid intake and output must be determined daily. Insensible fluid losses cannot be measured directly, but weighing the patient every day is

helpful in assessing the state of hydration, and haematocrit and total protein determinations are additional evaluators.

Volume balance is achieved by allowing the non-febrile patient an intake of 500 ml of fluid plus the total output of the preceding 24 hour. If the patient is febrile, 200 ml of additional intake is needed for each 1degree C increment. If fluid volume is in balance then body weight should decrease approximately 0.3- 0.5 kg per 24hours because of tissue catabolism. Over hydration must be avoided and it must be promptly corrected. Continuous careful supervision and replacement of urinary fluid electrolyte losses is even more important in the polyuric phase, as 50% of deaths occur during this time

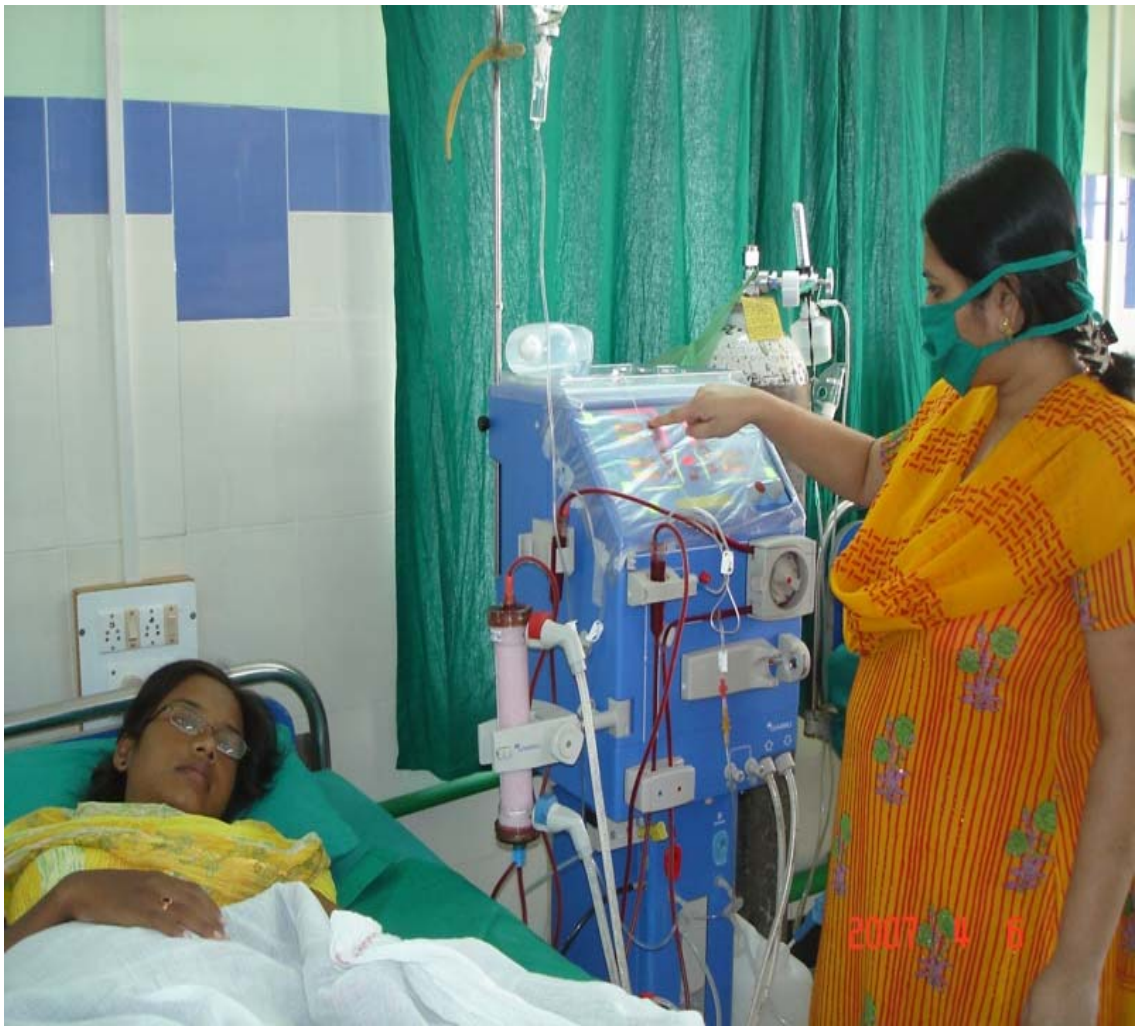
There is a need for oral or paraenteral administration of at least 1500 calories of a protein-free fat /carbohydrate combination, which is low in potassium and sodium. Carbohydrate calories are important for decreasing gluconeogenesis from protein, thus retarding the development of azotemia / acidosis.

PERITONEAL DIALYSIS

Peritoneal dialysis has many advantages and can be used in recently delivered women. It is easily available, simple, and inexpensive and has a

relatively low complication rate. There are no absolute contraindications and it can be used even in the presence of pelvic peritonitis. Relative contraindications are intra peritoneal adhesions, open wounds or drains in the abdomen and recent retroperitoneal operations. Occasionally peritoneal dialysis may be complicated by peritonitis or trauma to the intra abdominal viscera.

Fig.1. Hemodialysis Unit



HEAMODIALYSIS

Haemodialysis depends on an adequate shunt blood flow and has limited usefulness in the presence of hypotension. It is contraindicated in the actively bleeding patient, because even well-controlled regional heparinization does not ensure the safety of the procedure.

AIMS AND OBJECTIVES

3. AIMS AND OBJECTIVES

- To study the clinical profile of acute renal failure in postpartum period
- To find out the various risk factors and etiology for postpartum related ARF
- To study the dialysis requirement in postpartum related ARF
- To observe the management and outcome of the postpartum related acute renal failure patients

MATERIALS AND METHODS

4. MATERIALS AND METHODS

4.1. SELECTION OF SUBJECTS:

All patients were referred from the Department of Obstetrics to the Department of Nephrology in Government Rajaji Hospital with deterioration of renal function following delivery. 40 patients were included in our study.

4.2. PERIOD OF STUDY:

This descriptive study was conducted on cases of postpartum related acute renal failure between September 2008 and August 2009 at the nephrology department.

4.3. INCLUSION CRITERIA:

Postpartum women who were previously healthy and developed ARF were diagnosed with oliguria (Urine output <400 ml/d) and for mounting azotemia (Serum Creatinine >2mg %).

4.4. EXCLUSION CRITERIA

1. Evidence of renal disease prior to pregnancy (glomerulonephritis, renal insufficiency from any cause)
2. History of hypertension or diabetes before gestation
3. History of renal stone diseases
4. Renal scarring on ultrasonography
5. Small size of the kidneys
6. Elevated serum creatinine prior to gestation

Thus, women with no history of oliguria or renal disease prior to gestation, normal sized kidneys on ultrasound and no urological complications were included in the study.

4.5. HISTORY

Detailed history about obstetric and antenatal period were obtained from all the patients were made regarding the mode of delivery, term status during delivery, postpartum events, clinical presentation of acute renal failure, need for blood transfusion and surgical intervention.

4.6. INVESTIGATIONS:

The following investigations are done in all patients;

1. Haemoglobin
2. Blood urea
3. Serum creatinine
4. Blood sugar
5. Serum electrolytes
6. Liver function test and serum proteins
7. Complete hemogram and peripheral smear
8. Blood and urine culture
9. HBsAg
10. ECG
11. Ultrasonography

RESULTS

5. RESULTS

5.1. PROFILE OF THE STUDY:

Between September 2008 and august 2009, 8672 delivery were conducted in the obstetric labour ward, among them 40 patients (0.46%) were referred to nephrology department for postpartum related acute renal failure. These patients, who were healthy previously and had developed ARF were diagnosed in oliguria (Urine output <400 ml/d) and for mounting azotemia (Serum Creatinine >2mg %) after delivery. The following results were obtained in our study,

5.2. CHARACTERISTICS OF CASES STUDIED

Table 1: Age Distribution:

AGE GROUP(yrs)	NO. OF CASES	PERCENT
18-20	8	20
21-25	22	55
26-30	8	20
31-35	1	2.5
>35	1	2.5
Total	40	100

In our study, patients were between the age group 18-40 years. The mean age of patients with postpartum related ARF was 23.6 years. The youngest was 18 years old and the oldest 36 years old.

Fig.1. Age Distribution of the patients:

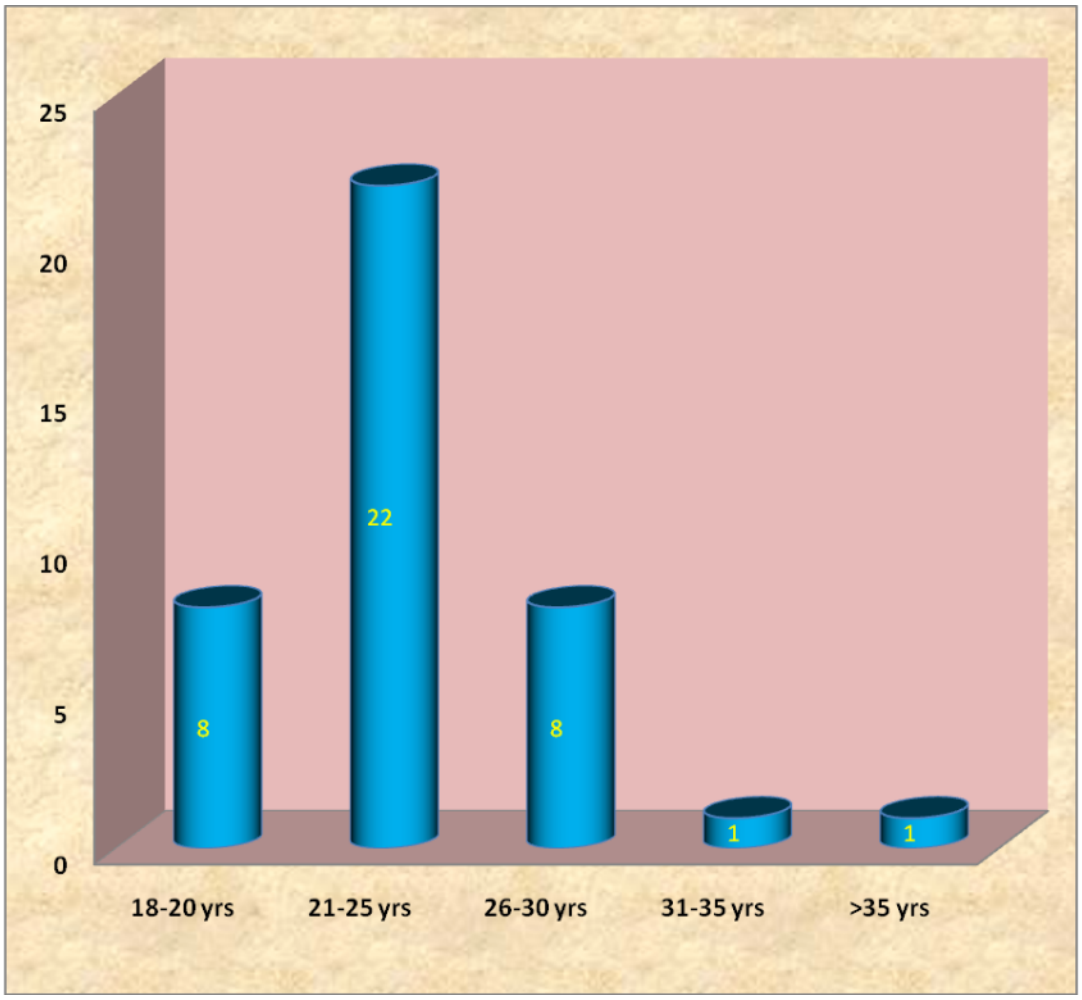


Table 2: Gravid status of the patient:

The following table shows gravid status of the patient in our study.

Gravid status	Cases	Percent
Multiparous	16	40.0
Primi	24	60.0
Total	40	100.0

In this study group 60% of patients were primi and rest were multiparous patients

Table 3.Booked status of the patient

BOOKED STATUS	CASES	PERCENT
BOOKED	36	90.0
UNBOOKED	4	10.0
TOTAL	40	100.0

In this study group, 90% of patient were booked cases, remaining 10% patient were unbooked and had not taken any antenatal care

Fig.2.Gravid status of the patients:

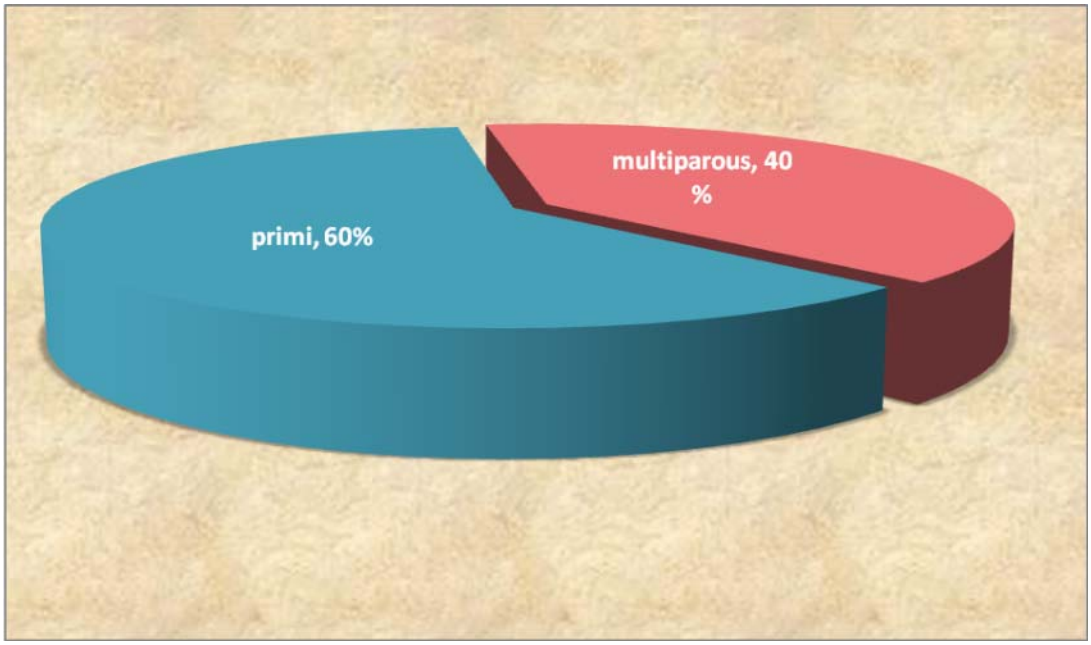


Fig.3.Booked status of the patients:

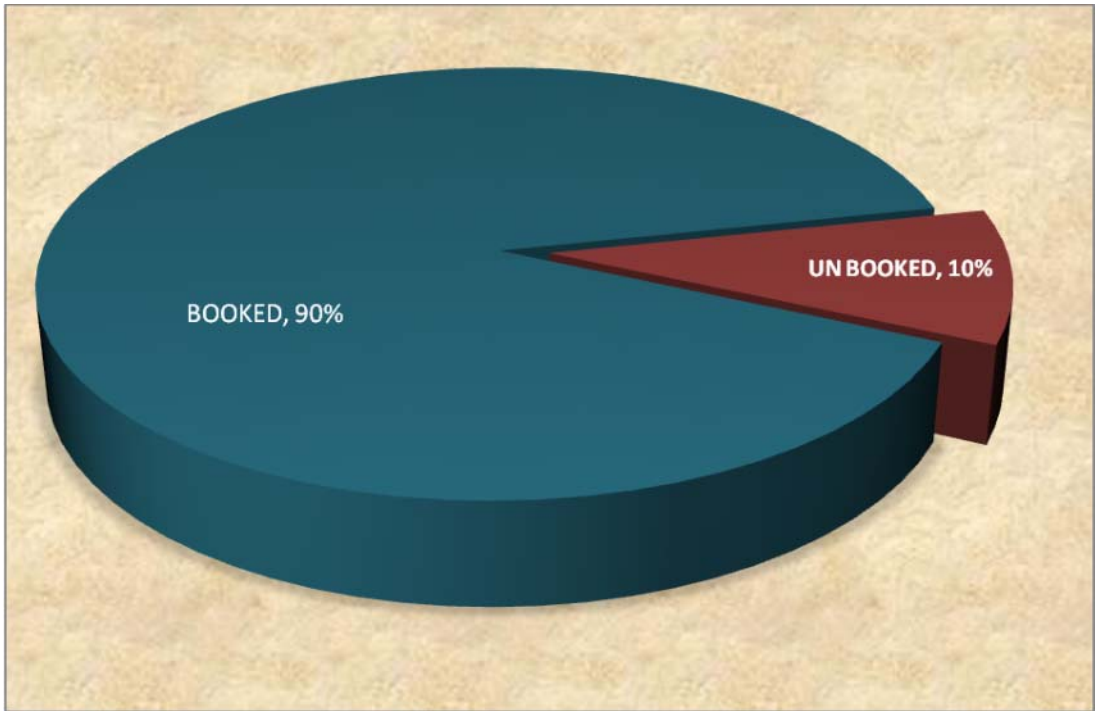


Table .4: Term status of the patient

TERM STATUS	CASES	PERCENT
POST TERM	6	15.0
PRETERM	4	10.0
TERM	30	75.0
TOTAL	40	100.0

In our study group, 75% of patient delivered at full term, 10% patients delivered preterm and remaining 15 % of patients were post term delivery. In post term 2 patients out of 6, delivered IUD baby.

Table.5. Mode of delivery

MODE OF DELIVERY	CASES	PERCENT
LSCS	12	30.0
VAGINAL	28	70.0
TOTAL	40	100.0

In this study, 70% of patients had normal vaginal delivery and remaining 30% had LSCS delivery.

Fig.4.Term status of the patients:

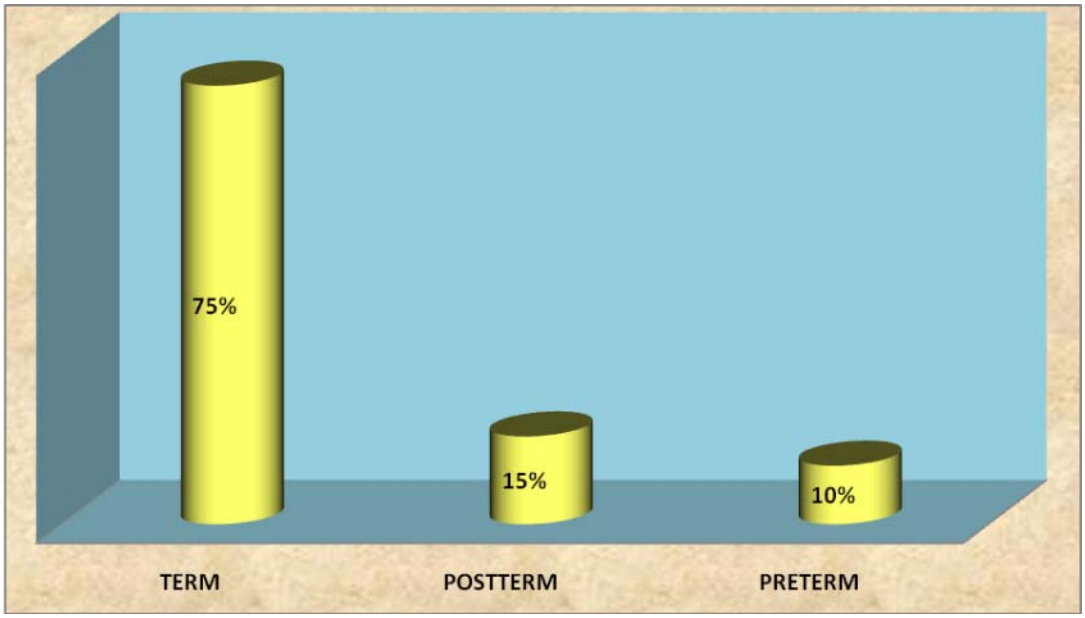


Fig.5.Mode of delivery of the patients:

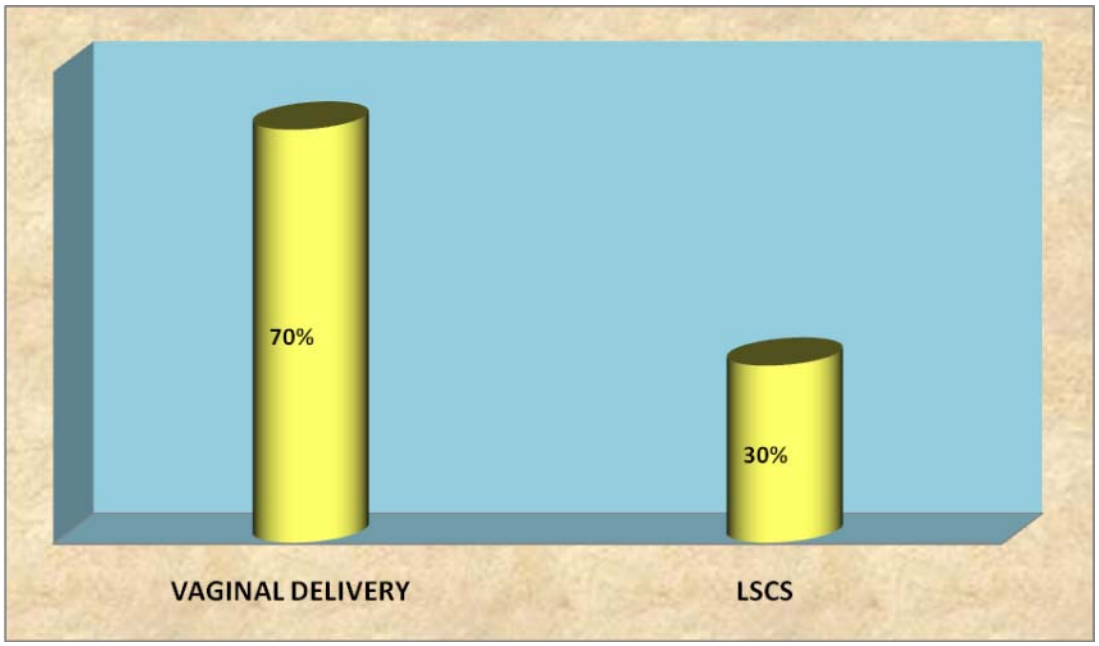


Table .6: Urine volume status of the patients

URINE VOLUME STATUS	CASES	PERCENT
ANURIA	13	32.5
NON OLIGURIA	2	5.0
OLIGURIA	25	62.5
TOTAL	40	100.0

In our study, twenty-five patients (62.5%) presented with oliguria, while 13 (32.5) with anuria and only two (5.0%) patients had non oliguric renal failure

Table.7: Antenatal events of the patients

ANTENATAL EVENTS	FREQUENCY	PERCENT
NO EVENTS	19	47.5
PIH	15	37.5
APH	3	7.5
HYPEREMESIS	3	7.5
TOTAL	40	100.0

37.5% of patients had history of PIH and 7.5% of patients had hyper emesis during the antenatal period

Fig.6. Urine volume status of the patients

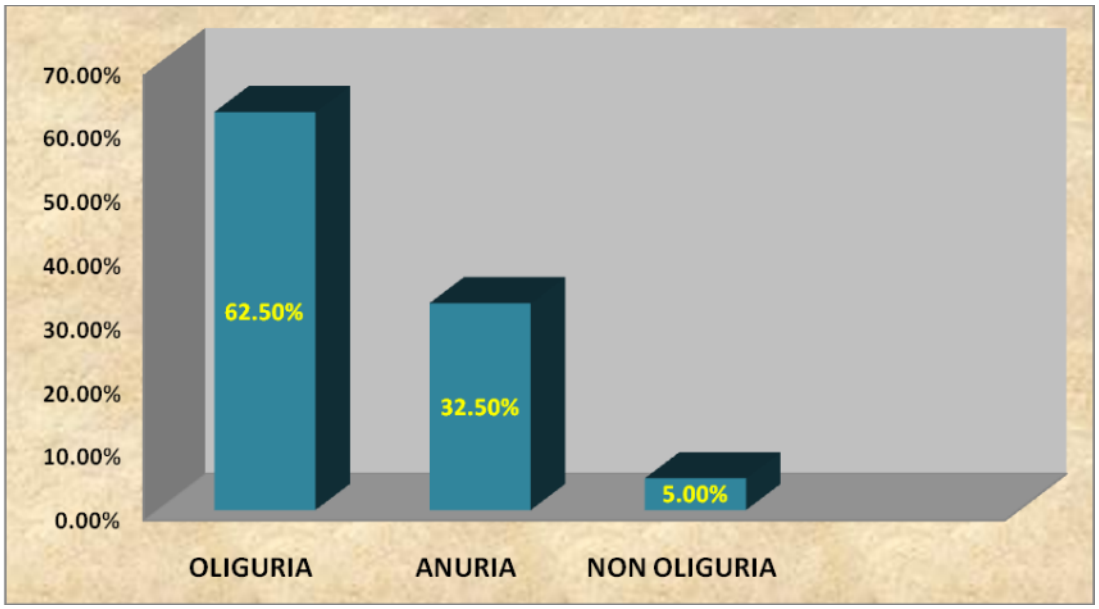


Fig.7. Antenatal events of the patients

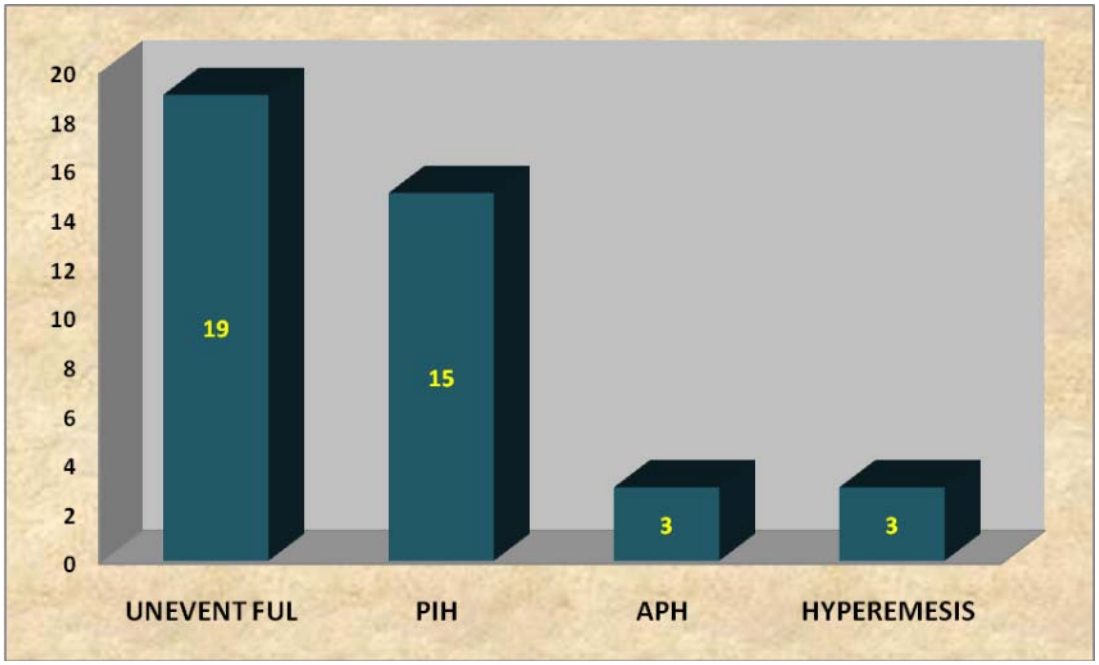


Table.8.Clinical presentations of the study group

CLINICAL FEATURES	NO.OF CASES	PERCENT
Anaemia	37	92.5
Pedal oedema	35	87.5
Fever	8	20.0
Hematuria	6	15.0
Jaundice	5	12.5
Vomiting	4	10.0
Dyspnoea	2	5.0

Most patients presented with more than one symptom and sign, Anaemia and pedal oedema are more common. 12.5% of patients presented with jaundice. Fever is the main symptom in 20% of patients who presented with septicaemia.

Fig.8. Clinical presentations of the study group

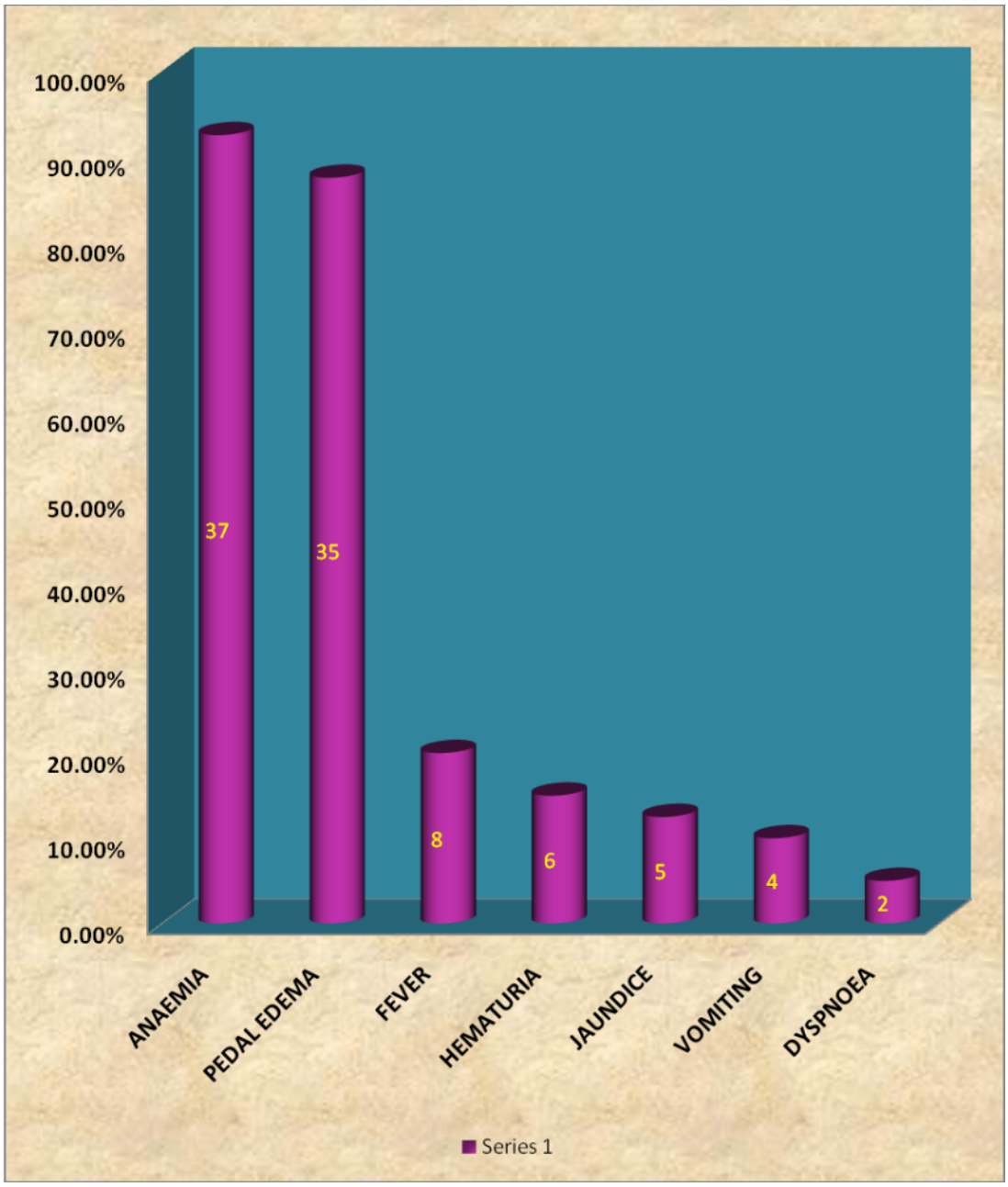


Table.9: Abnormal laboratory findings in postpartum ARF

This table shows various laboratory findings in our study,

Laboratory findings	No. of cases	Percent
Anaemia	37	92.5
Leucocytosis	12	30.0
Thrombocytopenia	23	57.5
Hypernatremia	5	12.5
Hyponatremia	16	40.0
Hyperkalaemia	12	30.0
Hypokalaemia	8	20.0
Abnormal LFT	11	27.5
Hypoalbuminaemia	23	57.5
Blood culture positivity	12	30

Fig.9. Abnormal laboratory findings in postpartum ARF:

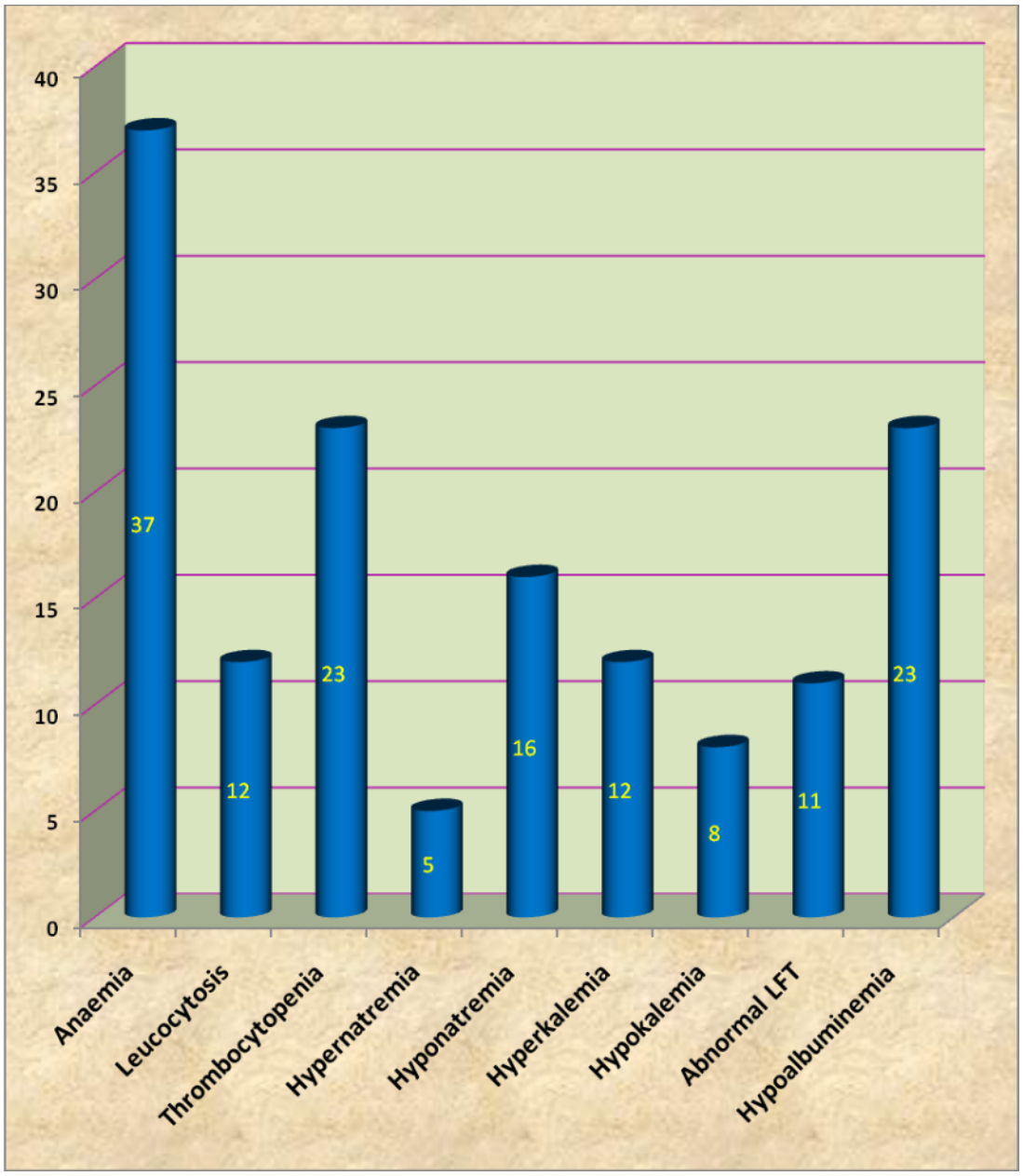
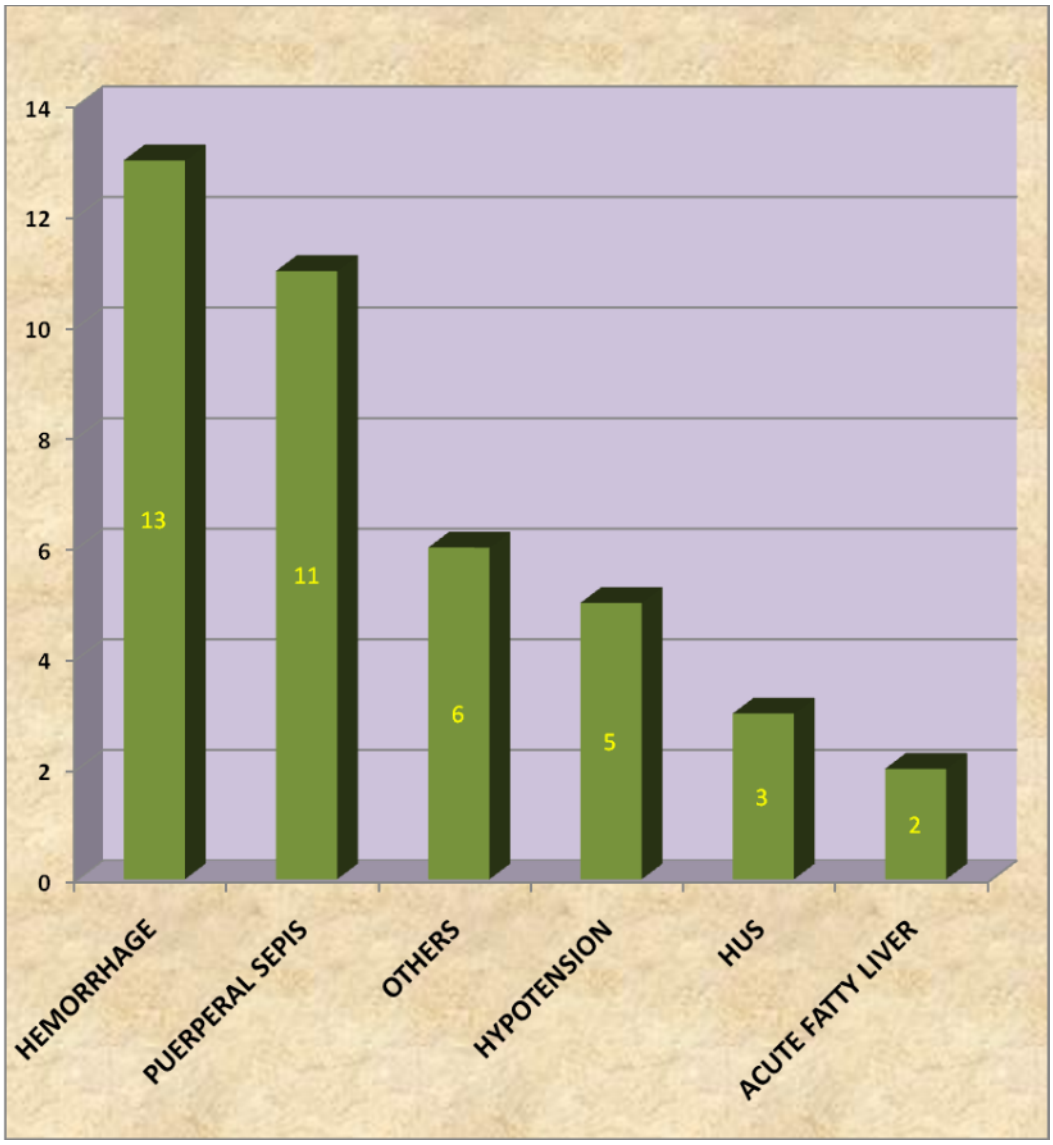


Table.10.Aetiology of postpartum renal failure

AETIOLOGY	NO. OF CASES	PERCENT
ACUTE FATTY LIVER	2	5.0
HAEMORRHAGE	13	32.5
HYPOTENSION	5	12.5
SEPSIS	11	27.5
HUS	3	7.5
OTHERS	6	15.0
TOTAL	40	100.0

In our study, various conditions produce postpartum acute renal failure. Among them haemorrhage and puerperal sepsis are the common causes. In 32.5% of patients, postpartum haemorrhage was the common etiological factor leading to ARF, while in 27.5% of patient puerperal sepsis was the cause.

Fig.10. Aetiology of postpartum renal failure



Hypotension as the aetiology for ARF was present in 12.5 % of the patients, haemolytic uremic syndrome in 7.5 % and acute fatty liver in 5.0% of patients. Acute pyelonephritis, broad ligament hematoma, RHD, CVT with hemorrhagic stroke and HELLP syndrome accounted for 15.0% of patients with postpartum related ARF.

Table.11: Nature of disease in our patients

NATURE OF THE DISEASE	CASES	PERCENT
ATN	24	60.0
HELLP SYN	1	2.5
HUS	3	7.5
UNKNOWN	12	30.0
TOTAL	40	100.0

In this study in 60% of patients acute tubular necrosis was the cause of renal damage.

Fig.11.Nature of disease in our patients

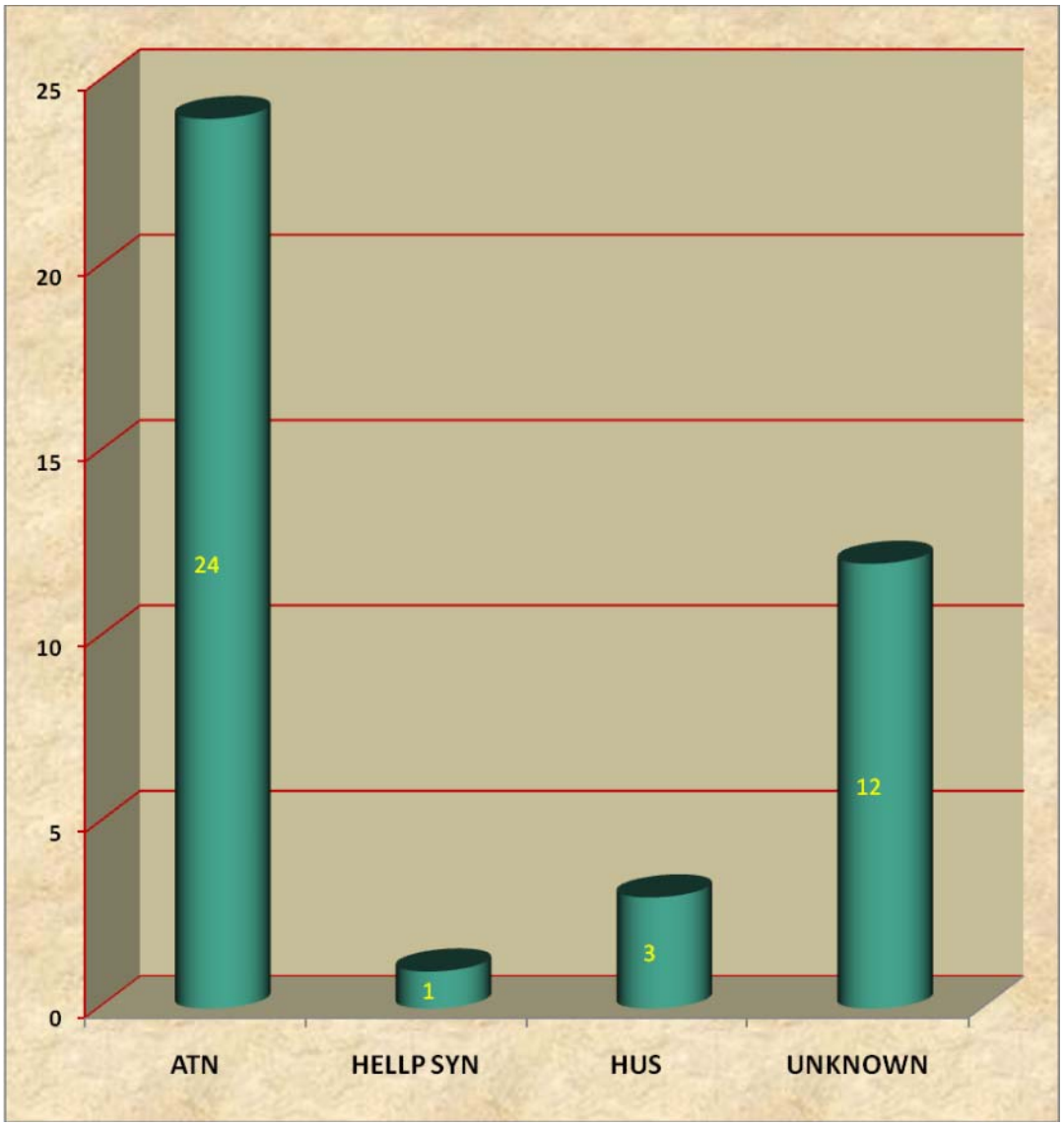


Table.12: Type of renal failure in our patients

TYPE OF RENAL FAILURE	CASES	PERCENT
POST RENAL	1	2.5
PRE RENAL	7	17.5
RENAL	32	80.0
TOTAL	40	100.0

Intrinsic renal ARF was the most common type in our study (80% of the patient), 17.5% of patients had pre-renal ARF and 2.5% of patients had post renal-ARF in our study.

Fig.12.Type of renal failure in our patients

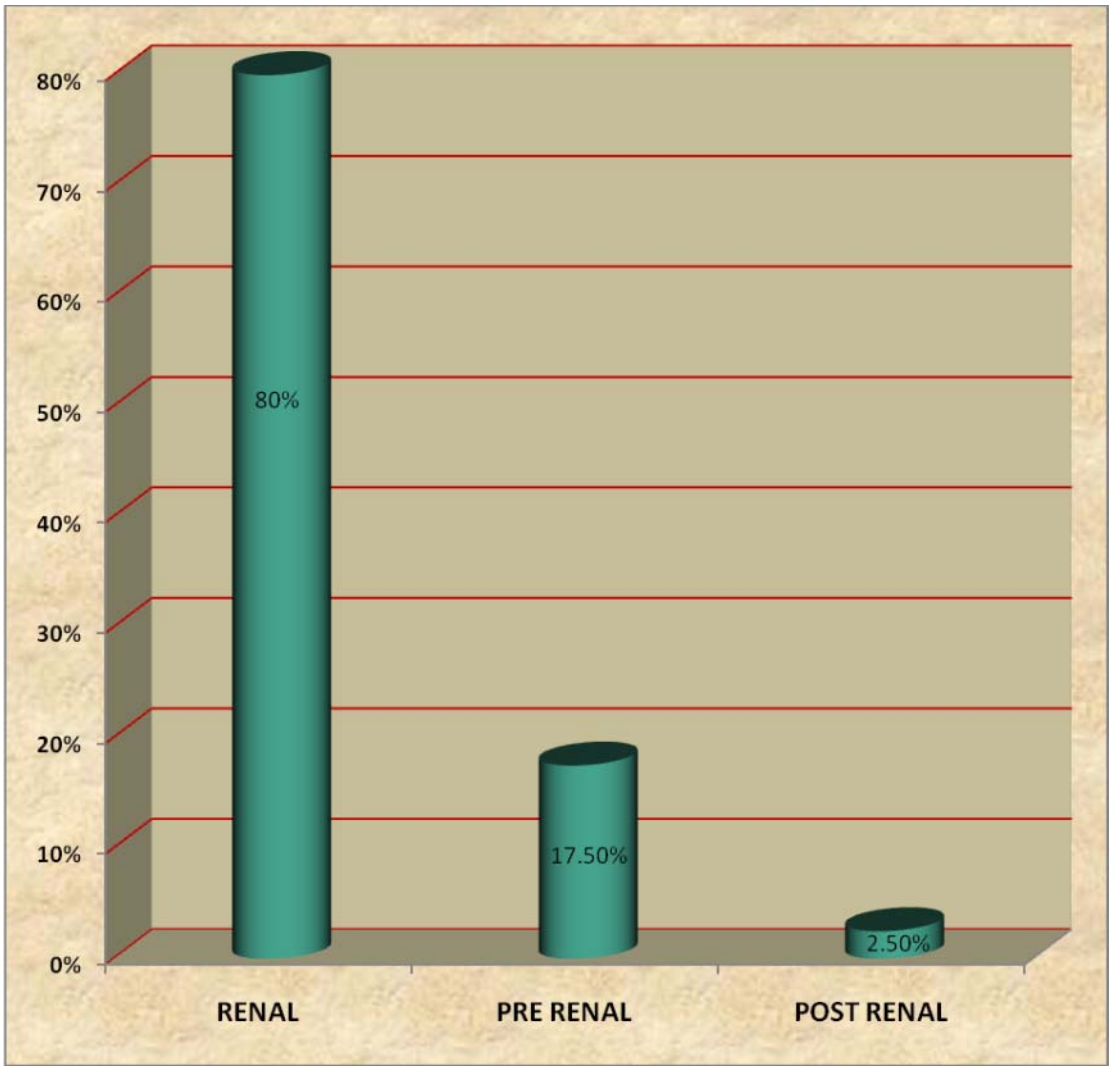


Table.13: Dialysis requirement in our study

Dialysis status	No of cases	Percent
Dialysed	25	62.5
Dialysis not required	15	37.5
Total	40	100.0

Majority of the patients (62.5 %) underwent hemodialysis, while 3 patients who needed mechanical ventilatory support in the intensive care unit, died without hemodialysis. One patient who underwent peritoneal dialysis due to hypotension died later.

Peritoneal dialysis done in 2 patients due to initial hypotension, 1 patient who improved received hemodialysis later.

Fig.13. Dialysis requirement in our study:

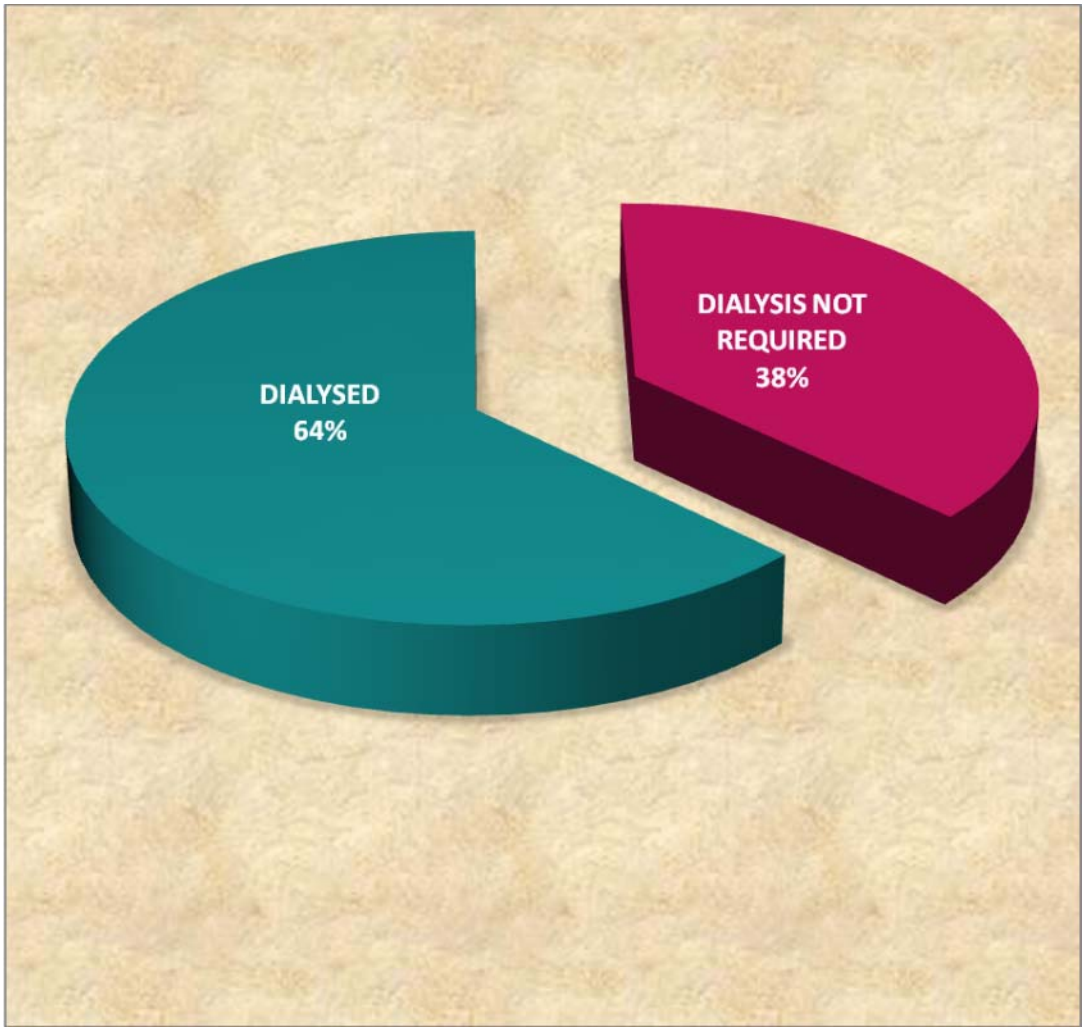
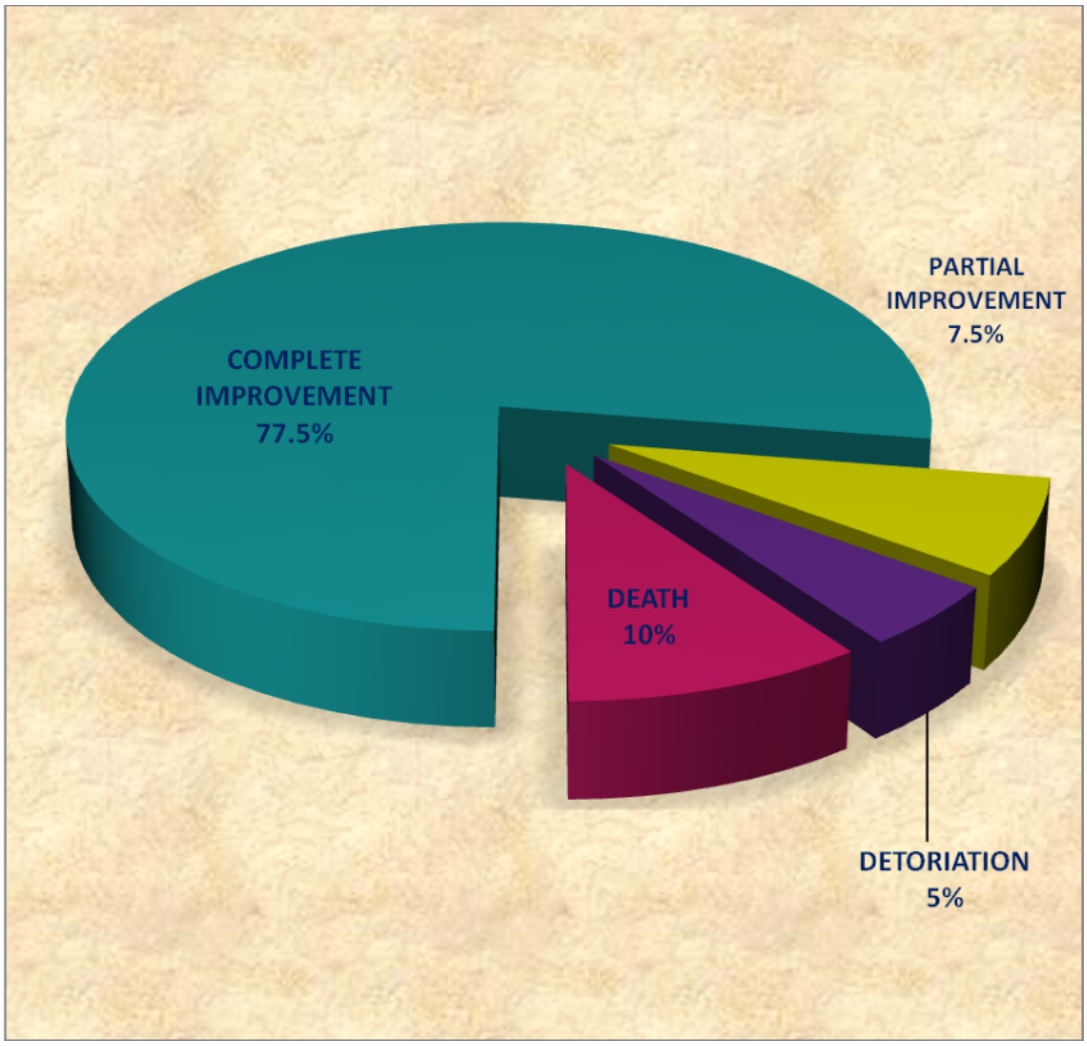


Table .14: Outcome of the study patients:

Outcome	Cases	Percent
Complete recovery	31	77.5
Partial recovery	3	7.5
Detoriation	2	5.0
Death	4	10.0
Total	40	100.0

Of the 40 patients, 31 cases (77.5%) made a complete recovery and 3cases (7.5%) showed partial recovery (i.e. dialysis independent in two patients and one patient discharged against medical advice) and 2 patients detoriated to grade II medical renal disease confirmed by follow up USG abdomen, 4(10 %) patients died, two patients developed ARDS and septic shock; two patients had DIC and hemorrhagic shock.

Fig.14.Outcome of the study patients:



DISCUSSION

6. DISCUSSION

Postpartum related acute renal failure is a rare entity in developed countries but continues to be a major problem in developing countries, resulting in high maternal mortality. The world wide incidence of postpartum related ARF has come down markedly in the past 30 years from 20-40% to less than 10% in more recent studies, largely due to the improvement in obstetric and postpartum care.

Pregnancy related acute renal failure is on the decline from 14.5% reported in 1987 to 4.3% in 2005 in India. The majority of pregnancy related acute renal failure cases were seen in the postpartum period (75.61%).

This study was done for one year period, with 8672 deliveries. Of them, only 40 patients developed ARF in the postpartum period, comprising 0.46%. This is consistent with the other studies in the recent past.

While ARF occurred in about 0.5 per 1000 pregnancies 30 years ago, its incidence is 10 times lower in the latest studies. In two series (***Chapman and Legrain 1979; Lindheimer et al. 1983***), no case of ARF was observed in 12,000 and 20,000 births, respectively. Two studies (***Donohoe***

1983; Stratta et al. 1996) document this decrease of frequency with time. In recent years the incidence of postpartum related ARF was seen in 0.07 % to 0.36% of pregnancies (***sibai et al 1994; firmat et al 1997)***.

In this study, patients were in the age group 18-40years. The youngest patient was 18 years old and the oldest was 36 years old. The mean age of patients with postpartum related ARF was 23.6 years. In other studies the mean age of patients with postpartum related ARF was 25.6 years ***(kumar et al 2006).***

In our study, 75% of full term delivered women had postpartum related ARF, and15% post term delivered women were involved, in other studies 60% of full term and 10%-20% of post term delivered women were involved (***Rani et al: 2002***).

Postpartum related ARF followed normal spontaneous vaginal delivery in 70% of our cases (28), caesarean section in 30% of cases (12). In other studies, postpartum related ARF followed normal vaginal delivery in 50% cases, caesarean section in 50% cases (***kilari et al 2006***).

In this study, 5% of patients had non oliguric ARF, 62.5% had oliguric ARF and 32.5% were anuric. In other studies all patients had oliguric ***(Kumar et al 2006).***

52.5% postpartum related ARF patients in our study had history of antenatal events like PIH, APH and hyperemesis and 47.5 % patients had uneventful antenatal period. Antenatal PIH patients were more prone to develop ARF in the postpartum period. ***(Prakash et al; 1995)***

More number of patients in this study presented with anemia (92.5%) and pedal edema (87.5%). Twenty percent of patients presented with fever and 12.5% of patients with jaundice. In study by ***kumar et al;2006***, fever was the predominant symptom.

In our study, 92.5% of patients (37/40) had anaemia, 6 patients was severe anaemia (Hb < 6 gm%), 30% of patients (12/40) had leucocytosis, 57.5% of patients had thrombocytopenia, 40% of patients (16/40) had hyponatremia, 30% of patients (12) had hyperkalaemia, 27.5% had abnormal liver function test and 30% patients (12) had blood culture positivity of gram negative organism. In ***Kumar et al study***, anemia was seen in 87.80%, leucocytosis in 80.49% and thrombocytopenia in 31.71%. Electrolyte abnormalities were seen in 75.68% and among these hypernatremia was seen in 14.63%, hyponatremia in 36.59% and hyperkalemia in 9.76% of patients.

In the present study, postpartum haemorrhage (32.5%) and puerperal sepsis (27.5%) were the common causative factors, while in other studies

puerperal sepsis (39.2%) was the most common (**Grunfeld JP et al; 2005, *kumar et al; 2006***).

In the remaining cases, postpartum related ARF was secondary to postpartum haemolytic uremic syndrome (7.5 %), acute fatty liver (5.0%), hypotension (12.5%), acute pyelonephritis, broad ligament hematoma, RHD, CVT with hemorrhagic stroke and HELLP syndrome. Intrauterine deaths were noted in 4 patients (10%) in our study, whereas in ***kumar et al; 2006*** study it was 17.7%.

One case of retro peritoneal and broad ligament hematoma was reported after caesarean section delivery. It compressed the ureters and produced post renal ARF and after surgical removal of hematoma, patient completely recovered. Another patient reported with postpartal CVT with hemorrhagic stroke. She developed ARF 2 days followed delivery. In this study, ARF developed in the first week of postpartum period whereas in ***Sibai*** studies, ARF developed commonly in 3rd day of postpartum period.

Renal biopsy was not done in our study and the exact cause of renal damage to one third of the patients could not be ascertained, two third of patient diagnosed acute tubular necrosis by clinical examination, laboratory and radiological examinations.

In our study only one patient (2.5%) diagnosed as HELLP syndrome (Haemolysis, Elevated Liver enzymes, Low Platelet count) was associated with postpartum related ARF, whereas in ***sibai et al 1994*** study , in a series of 442 pregnancies complicated by this syndrome, the onset was ante partum in 70 per cent and postpartum in 30 per cent. ***Seljuk et al. (2000*)** found the HELLP syndrome was associated in 36 per cent of pregnancy related ARF among them, one fourth occurred in postpartum period.

In our study, 2 patients (5%) had acute fatty liver in post partum period. In the more recent series (***Usta et al. 1994; Castro et al. 1999),*** acute fatty liver occurred in the 28 to 39 weeks of pregnancy (90%) and in postpartum in a very few patients (< 10%).

In our study, HUS was noted in 3 cases (7.5%) of postpartum related ARF. All the 3 patients were multiparous and had history of PIH in ante partum period. Among them, 2 patients developed grade II medical renal disease which was confirmed by follow up ultrasound abdomen. In some series, the haemolytic uremic syndrome was noted in 5 to 10 per cent of postpartum-related renal failure (***Grunfeld et al. 1980; Stratta et al. 1996).*** In the 62 cases reviewed by ***Weiner (1987),*** only five developed HUS

before delivery. The disease occurred typically between a few hours and 8 to 10 weeks postpartum, but delay of several months has been reported occasionally. In most cases in these studies, pregnancy and delivery were uneventful and most patients were multiparous. It usually started with influenza-like symptoms. Moderate Proteinuria and microscopic Hematuria preceded the development of anuria, which is usual. Hypertension develops in half of the cases.

Intrinsic renal ARF was the most common type in our study (80%), 17.5% of patients had pre-renal ARF and 2.5% of patients had post renal-ARF in our study. In other studies also, intrinsic renal ARF was the most common type. Urinary tract obstruction is a rare cause of ARF in postpartum period. Although the frequency of renal stones is no greater in postpartum women, nephrolithiasis can cause ARF ***(Naqvi et al. 1996)***. Loin pain, gross hematuria and urinary infection, suggest the diagnosis. Ultrasonography allows quantification of the dilatation and often visualization of stones. When coupled with colour Doppler, it can demonstrate the interruption of urethral flux and the absence of ureteral ejaculation ***(MacNeily et al. 1991)***.

Renal tubular obstruction by uric acid has been reported in a few patients. Serum uric acid exceeded 700mol/l and was unexplained, but dehydration contributed (***Alexopoulos et al. 1992***). Ureteral obstruction by

broad ligament hematoma after caesarean section delivery has been reported to cause acute renal failure in rare instances.

In our study, majority of the patients (62.5 %) underwent hemodialysis and one patient underwent peritoneal dialysis due to hypotension and died later. Peritoneal dialysis was initially performed in 2 patients due to initial hypotension, one patient among which improved and received hemodialysis later. Similar to other studies, more than 50% of patients were given dialysis support (***Kumar et al: 2006***). Blood transfusion was given in 29 (72.5%) patients in our study for postpartum haemorrhage. Duration of hospital stay ranged from 1 to 30 days (mean 6.42 days).

In our study, among the 40 patients, 31 showed complete recovery (77.5%) and 3showed partial recovery (7.5%) (i.e. dialysis independence in two patient and one patient left against medical advice). In other studies, complete recovery was found in 60 % of patients and partial recovery was found in 9.7%.

10 % of the patients (4) died in our study among which, two patients developed ARDS and septic shock and two patients had DIC and hemorrhagic shock. The mortality due to postpartum related ARF has declined to < 10% in Europe and North America. While the reported

mortality rate of postpartum related ARF has decreased from 56% in 1987 to 24.39% in 2005 in India. The mortality rate was 10% in our study, which is in accordance to current trends in India but still significantly higher compared to the developed countries.

Although there has been a significant decline in postpartum related ARF at the international and national levels, it continues to be static in our study largely due to an insignificant decline in septic delivery and insignificant correction of postpartum haemorrhages. Hence, there is a need for education and improvement in ante- and postnatal care, especially in the rural areas, and the practice of septic delivery by untrained personnel has to be stopped.

CONCLUSION

7. CONCLUSION:

- In conclusion, our study showed that ARF in postpartum period was mostly due to renal and pre-renal causes. Maternal mortality was the highest with multiorgan failure, sepsis, DIC, and HUS. These factors can be prevented with good antenatal care and education of the rural masses.

- Pregnant women should have better obstetrical facilities. Unqualified persons handling of pregnancy and delivery should be avoided.

- Promptly recognise the postpartum haemorrhage, puerperal sepsis, to institute appropriate treatment

- At or after delivery, blood loss should be replaced quickly to the point of slight over transfusion, because any haemorrhage may be underestimated.

- Any patient with postpartum related ARF should be referred to higher centres as early as for prompt diagnosis and timely nephrological intervention.

- Some of the problems can be managed with by judicious conservative methods, but if such an approach is unsuccessful, dialysis will be necessary.

- Ideal care for women with acute renal failure in pregnancy or postpartum requires a multidisciplinary approach that may include maternal-foetal medicine, critical care medicine, nephrology, and neonatology specialities.

- Postpartum related acute renal failure is a critical condition, associated with serious prognosis for both women and kidneys. So far, the most effective measures still remain the careful prevention and aggressive management of the obstetric complications.

- Early reorganization of this disorder, improvement of health infra structure, antenatal health care and intensive supportive therapy, can reduce postpartum related ARF and maternal mortality.

APPENDIX

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Clinical profile, Management and Outcome of the Patient with Postpartum related

Acute Renal Failure (PRARF)

Name of the patient: Age: IP No:

Height: Address: D.O.A

Weight: D.O.D

Obstetric code: P /L /A /IUD /Stillborn /Dearborn

Marital history: Married /unmarried; Consanguineous/ Nonconsang

Presenting illness:

Past history:

Pre disposing factors: Nephrotoxic drugs ☐ Sepsis ☐ Hypotension ☐ Rhabdomyolysis ☐ ☐ others

Antenatal history: Primi/ multigravid; Booked/unbooked; Immunized/Unimmunized

Drug history

PIH/ APH/ Hyperemesis/ Others

Intrapartum: Term/ preterm/ Post term; Vaginal delivery/ LSCS, Blood transfusion + / -

General examination:

PR: BP: RR: Fundus:

CVS: RS:

Abd: CNS:

Urine Volume status at onset of azotemia: Anuria(50ml) ☐ Oliguria ☐ Non Oliguria ☐

Clinical Features : Haematuria/ HTN / Jaundice / Edema / Vomiting / Diarrhoea / Others

Complications: Fluid overload ☐ Hyponatremia ☐ Hyperkalemia ☐ Met.acidosis ☐

Anemia ☐ infection ☐ Arrythmias ☐ GI bleed ☐ pericarditis ☐

Investigations: Urine-Alb:…….Sugar:……Deposits:…….Spot protein creatine ratio……

Blood-Hb:..…...TC:……DC:…….. .....PCV:…….. platelet Count…….....

Blood group : Peripheral smear: …… LFT

Day	1	2	3	4	5	6	7	8	9	10
Sugar										
Urea										
Creatine										
Sodium										
Potassium										
Urine vol										

Blood culture / Urine culture Serum uric acid.……S.calcium…… S.phos……..; Serum Ck……;
Urine Osmolality: Plasma Osmolality:
Fractional excretion of sodium……

Renal failure index- (urineNa)/(U/P.creat) ……..

USG Abdomon: Kidney size-(R)…… (L)……; Impression:

Treatment: Use of frusemide ☐ Mannitol ☐ Dopamine ☐ Saline challenge ☐

Response to above measures: YES ☐ NO ☐

Antibiotics:

Renal Replacement Therapy: (YES/No) PD ☐ HD ☐

Indication of dialysis: Duration of azotemia: Duration/cycles of dialysis:

Outcome: Complete improvement ☐ Partial ☐ Deterioration ☐ Death:Renal ☐ Non-Renal ☐

S U M M A R Y

TYPE OF RENAL FAILURE :

ETIOLOGY :

NATURE OF RENAL INJURY :

DURATION OF HOSPITAL STAY :

COMPLICATION :

RESPONSE TO FRUSEMIDE :

DIALYSIS(FREQUENCY) :

OUT COME :

ABBREVIATIONS AND ACRONYMS

APH- Ante Partal Haemorrhage

ARDS-Adult Respiratory Distress Syndrome

ARF –Acute Renal Failure

BUN-Blood Urea Nitrogen

CT-Computerised Tomography

CVT-Cerebral vein thrombosis

DIC-Disseminated Intravascular Coagulation

FF-Filtration fraction

GFR-Glomerular Filtration Rate

HELLP- Haemolysis, Elevated Liver enzyme,Low Platelet

HUS-Haemolytic Uremic Syndrome

PIH-Pregnancy Induced Hypertension

RHD-Rheumatic Heart Disease

RPF-Renal Plasma flow

USG-Ultrasonography