A Dissertation on

OBSTETRIC RENAL FAILURE –A PROSPECTIVE STUDY

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CERTIFICATE

This is to certify that **Dr. B.BALAMURUGAN,** is a bonafide student of Department of Nephrology, Madras Medical College and Government General Hospital, Chennai – 600 003 and his study on "**OBSTETRIC RENAL FAILURE"** is a bonafide original work done by him, for his dissertation towards the partial fulfillment of the D.M NEPHROLOGY degree. He has done this study under my supervision and guidance and no part of this study has been submitted for the award of any other degree or diploma.

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REVIEW OF LITERATURE

Introduction

The unique vulnerability of the kidneys in pregnancy was first noted in 1843 when John Lever recognized that proteinuria occurring in association with maternal convulsions was an entity separate from chronic kidney disease. Lever's observation came 16 years after Richard Bright's recognition of proteinuria as a marker for kidney disease[1]. The distinction between primary kidney disease and preeclampsia remains one of the most common issues addressed by nephrologists seeing pregnant women.

Pregnancy in a woman with kidney disease or the development of primary kidney disease in a pregnant woman has long been recognized as dangerous. In 1936, Dieckman[2] recommended termination of pregnancy if nephrosis occurred before 30 weeks gestation. At that time, not only was there no effective treatment for kidney failure, but antibiotics, steroids, and effective antihypertensive treatment were not available. Women with known kidney disease were discouraged from becoming pregnant, and most of our knowledge about pregnancy in kidney disease has been acquired only because women wanted children ignored the advice of their physicians.

In 1970 Lindheimer and Katz[3] observed that successful pregnancy was the rule in women with kidney disease whose kidney function was well preserved. Some investigators associated poor outcomes with specific glomerular lesions[4], and only later did it become clear that preconception serum creatinine was more important than the specific kidney disease (except in case of lupus nephritis). Until the 1980, most series of pregnancy in women with kidney disease included only a few women with serum creatinine greater than 1.4 mg/dl. Over the following 10 years, more than 7 reports were published detailing 226 pregnancies in 196 women with moderate to severe preexisting kidney disease. Collective experience indicated that these women risked more rapid progression to kidney failure if they became pregnant[5-11]. At the same time, advances in the care of premature newborns resulted in the survival of more than 90% of infants from pregnancies in this group of women.

Fertility in women with chronic kidney disease is generally reduced, in part because of gonadal dysfunction and secondary amenorrhea[12-14]. There may be continuum of decreased fertility as the glomerular filtration rate declines, but further study in this area is needed[15]. When counseling women with chronic kidney disease about pregnancy, it must be clear that information on outcomes is limited. In part, this is because of lack of systemic data, inability to randomize patients, and difficulty to find appropriate controls[15, 16]. For most patients with a serum creatinine < 1.4 mg/dl, pregnancy leads to successful obstetrical outcome without affecting the course of the maternal kidney disease[17-20]. For patients with moderate renal impairment, the outlook is not optimistic; in patients with a serum creatinine > 2.0 gm/dl at the time of conception, one third progress to end stage renal disease (ESRD) over the postpartum year of follow up[21].

A history of dismal pregnancy outcomes in women with kidney disease has been observed since the 1960s[22, 23]. Although outcomes have improved with advancements in disease management, the presence of kidney disease during pregnancy still portends adverse outcomes for both mother and fetus[24, 25]. Beyond its association co morbid complications such as hypertension, chronic kidney disease is an independent risk factor for prematurity, low birth weight, neonatal death and preeclampsia[24-26]. Chronic kidney disease may increase the risk of such pregnancy complications by as much as 8-fold[24, 25]. Hence the coexistence of chronic kidney disease and pregnancy remains a daunting clinical scenario for both health care providers and patients.

Acute renal failure has become a rare complication of pregnancy due to the virtual disappearance of septic abortion and vigilant antenatal care in the western countries. In the 1960s pregnancy-related represented 20-40 percent of all cases of ARF, but the incidence have decreased dramatically there after. In the 1980s the estimated incidence if ARF requiring dialysis during pregnancy in the western countries was less than .01 percent. In contrast, obstetric ARF still remains a serious problem in developing countries; it constitutes 15-25 percent of all cases of ARF referred to dialysis centers in India.

However, in some parts of India, the incidence of obstetric ARF has shown a decline from 22 percent [of all ARF] in 1960s to 8 percent in 1990s. In Ethiopia, septic abortion is the underlying cause of ARF in 52 percent of all patients with ARF. In Argentina and Nigeria obstetric complications still account for 32 percent and 25 percent of cases of ARF, respectively. Obstetric ARF has a bimodal occurrence in developing countries. The first peak is seen between 8 and 16 weeks of gestation in association with septic abortions; whereas late peak is seen after 34 weeks associated with eclampsia, abruption-placentae, post-partum hemorrhage and puerperal sepsis. The first peak is nearly eliminated in industrialized countries due to the virtual disappearance of septic abortion.

Obstetric group

Based on the obstetric problems patients are divided into two groups: ARF in early pregnancy and ARF in late pregnancy. Acute renal failure is related to abortion in all cases in early pregnancy. However, ARF in late pregnancy develops, in association with multiple obstetric complications consisting of eclampsia, puerperal sepsis, uterine hemorrhage and postpartum hemolytic uremic syndrome.

Probable pathogenesis

Multiple factors are involved in the development of obstetric ARF. These include hypotension, sepsis, infection and blood loss.. Sepsis is more frequent in patients with induced abortion than in spontaneous abortion. In addition, intravascular hemolysis disseminated intravascular coagulation and adult HUS also is causes of ARF in pregnancy.

Chronic Kidney Disease and Pregnancy: Pathophysiology

It is well established that renal blood flow and glomerular filtration rate (GFR) increase substantially during pregnancy. GFR begins to rise within the first month after conception, increasing up to 50% by the 16^{th} to 18^{th} gestational week of pregnancy.

This state of hyper filtration persists throughout the rest of pregnancy with GFR returning to baseline values 3 months postpartum. Although the precise mechanisms for these changes in kidney hemodynamics remain unclear, a combination of increased extra cellular volume and cardiac output and decreased renal vascular resistance is involved.

Some have proposed that pregnancy induced hyper filtration may lead to acceleration of GFR decline in chronic kidney disease. However, hyper filtration in pregnancy appears to be caused primarily by increases in renal blood flow rather than increases in intra glomerular pressure so it is less likely to lead to pathological sequelae. In fact, animal models have not shown glomerulosclerosis after repeated gestations. Moreover, women with preexisting kidney disease, who are most likely to have a pregnancy associated GFR decline, often do not appear to acquire a hyper filtered state during pregnancy.

Closely related to this phenomenon of hyperfiltration is the hypervolemic state that exists during pregnancy. Substantial weight gain occurs during pregnancy with much of the weight gain resulting from sodium and fluid retention. The extra cellular fluid space often expands by 7L. The reasons for this volume expanded state are not entirely clear. “Under fill”, “overfill” and “normal fill” concepts invoking various hormonal changes have been hypothesized, and all may be at work in different stages of pregnancy. In addition to its influence on renal blood flow and GFR, this hypervolemic state also carries implications toward hypertension and its management during pregnancy.

A few other important factors influencing the effect of pregnancy on kidney function are

1) Increase in urinary protein and albumin excretion occurs commonly during pregnancy, often achieving microalbuminuria levels but generally staying below 500mg/d. The mechanism for this is not well understood, but alteration of GFR and tubular reabsorption are thought to contribute. Unfortunately the exact nature of these changes and their prognostic significance are not well studied. Prolonged recurrent periods of elevated proteinuria theoretically could lead to kidney damage and progressive kidney disease.

2) Pregnancy is associated with a relative hypercoagulable state. Several components of the coagulation cascade are increased, and fibrinolytic activity is decreased during pregnancy. These changes could mediate insidious thrombotic glomerular injury, especially with recurrent pregnancies.

3) The frequent development of hypertension in pregnancy complicated by preexisting kidney disease also may play a substantial role in deterioration of kidney function. However data are lacking regarding the precise burden imposed by gestational hypertension.

Prevalence and epidemiology of chronic kidney disease during pregnancy

Chronic kidney disease usually results from cumulative organ injury over an extended period of time. Therefore, it is not surprising that the presence of chronic kidney disease in pregnant women is rather uncommon because the pregnant population is usually young and relatively healthy. Two retrospective studies have characterized the prevalence of chronic kidney disease in pregnant population. Fink et al noted the incidence of kidney disease to be 0.03% among pregnant women in the state of Washington from 1987 – 1993. In two other reviews of kidney disease in pregnant women during 1990's from Colorado and Japan the prevalence of kidney disease was higher but still not excessive at 0.1% to 0.12%. it is important to realize that these findings are likely under estimates for several reasons.

It has become increasingly apparent that chronic kidney disease is under recognized by health care providers and hence under documented in patients' record. This under recognition is likely further embellished during pregnancy when one considers the physiological changes that normally occur with pregnancy. A customary increase in maternal renal plasma flow and GFR occur during pregnancy. Consequently a pregnant woman serum creatinine generally decreases by 0.4 mg/dl during pregnancy and the normal value is usually around 0.5 mg/dl.

In fact the serum creatinine exceeding 0.8mg/dl is generally considered to be abnormally elevated. Thus chronic kidney disease is likely to be unrecognized during pregnancy. Second, it is known that pregnancy losses are more likely in the setting of chronic kidney disease and its associated co morbid conditions. Early pregnancy losses may not be always noted by the mother or captured by studies. These factors suggest that the occurrences of pregnancy in woman with chronic kidney disease as well as the presence of chronic kidney disease in pregnant woman are under estimated.

A broader estimate of the prevalence of chronic kidney disease can be viewed from the frequency of kidney disease in women of child bearing age. Based on data gathered by Nissenson et al from a large health maintenance organization in the south western United States during 1994 to 1997, it appears that up to 4% of child bearing – aged women (up to 49 years old) have chronic kidney disease (defined by a serum creatinine greater than or equal to 1.2 mg/dl). Because a number of women may have substantial decreases in GFR at creatinine lower than 1.2mg/dl or have kidney disease manifested in ways other than substantial GFR decline, it is likely that chronic kidney disease is present in an even larger percentage of child bearing – aged women.

A number of types of chronic kidney disease will be found in a setting of pregnancy, including diabetic nephropathy, chronic glomerulonephritis, reflux nephropathy, tubulointerstitial disease, systemic lupus erythematosis, and polycystic kidney disease. Diabetic nephropathy is generally the most common form of chronic kidney disease during pregnancy.

Because most studies of kidney disease are retrospective analysis relying on administrative data or case series, a precise characterization of the frequency of different kidney disease in the setting is challenging and compromised by a lack of detail and specificity. Nonetheless, the largest and broadest description of etiology of chronic kidney disease in pregnancy is provided in the study by Fink et al.

In their analysis, the following causes of chronic kidney disease were observed: diabetic nephropathy (7%), chronic glomerulonephritis (8%), unspecified nephritis or nephropathy (11%), chronic renal failure (7%), nephritic syndrome (13%), renal agenesis (14%), and cystic diseases of the kidney excluding acquired cystic disease (18%).based on these data and others, it is important to recognize that significant heterogeneity exists among the causes of chronic kidney disease in pregnant women.

Pregnancy outcomes and chronic kidney disease

A number of small studies have evaluated pregnancy outcomes in the setting of maternal kidney disease. In reviewing this subject, it is important to distinguish between primary kidney disease and systemic diseases possess additional specific features beyond nephropathy that contribute to maternal and fetal outcomes.

It is critical to recognize the limitation of the published analyses. First, most of these studies consist of small sample sizes at single centers, compromising the generalizability of the conclusions. Second, the vast majority of these studies are retrospective in design, subjecting their findings to bias and confounding. Third, few these studies involved the comparison groups of healthy women without kidney disease are invoked multivariable analysis statistical methods. Thus, attributing the nature and extent of suboptimal maternal and fetal outcomes to kidney disease on the basis of published literature is difficult. Moreover, a variety of definitions for kidney disease were used by the authors, limiting comparisons between the published studies. In addition, the definition of important maternal and fetal outcome measures often differed between studies. In particular, the nature, reversibility and acceleration of kidney function (GFR) decline and the persistence of changes in hypertension and proteinuria have had varying meanings and occasionally were not clarified by the authors. Nonetheless, the existing literature is helpful in understanding the relationship between chronic kidney disease and pregnancy and the underscores the importance of maternal kidney function at consumption on pregnancy outcomes.

Primary kidney disease and pregnancy outcomes

Mild kidney disease (maternal serum creatinine < 1.3 mg/dL)

Maternal outcomes: Pregnancy in the setting of mild maternal kidney disease does not precipitate an accelerated worsening of maternal kidney function. However, the development or worsening of hypertension, proteinuria, and preeclampsia occurs in as many as one third of pregnant women with mild kidney disease. The long-term implications of these events have not been well evaluated in this population.

Early studies raised the concern that maternal kidney function could undergo irreversible deterioration during a normal gestation. Four studies of roughly 600 pregnancies in women with the serum creatinine < 1.3 mg/dl did not observe any accelerated decline in maternal GFR during pregnancy. A review by Imbasciati and Ponticelli summarized the result of 6 large studies of pregnancy and kidney diseases during the last 25 years. Collectively, 906 pregnancies in 558 women with histologically defined primary kidney disease were included. Over 95% of the subjects had a serum creatinine <1.5 mg/dl. Over all, by the end of gestation, kidney function showed a reversible decline in 8% of women but a progressive decline in only 3%.

More recent studies from the 1990s also deserve mention in an observational trail, Abe compared 32 pregnant women with biopsy proven ImmunoglobulinA (IgA) nephropathy with 30 women with IgA nephropathy who did not conceive over a 5-year period, the initial and final GFR measurements did not vary significantly between the 2 groups. Jungers et al studied the incidence of n stage renal disease over 30 years of follow up in 320 women with various glomerular diseases and mildly abnormal kidney function. Kidney survival did not differ significantly between the group of women who became pregnant and who did not. More over, in multi variable analysis, pregnancy was found to be a risk factor for end stage real disease.

Fetal outcomes

Prematurity, low birth weight and fetal death are slightly higher in the setting of mild maternal primary chronic kidney disease. However, it does appear that these negative outcomes have dissipated over time with advances in medical care. Unfortunately, the independent roles of proteinuria and hypertension are not well distinguished. The aforementioned review by Imbasciati and Ponticelli addresses the typical fetal outcomes associated with mild maternal kidney disease. In these studies the overall rate for fetal loss was 21%; preterm delivery was also high at 19%. Importantly this article incorporated data from 1980s. A more recent analysis by Bar et al suggests that fetal outcomes in the presence of mild chronic kidney disease have improved over time; in this study successful fetal outcome was reported in 98% of patients. About 65% of pregnancies resulted in no obstetrical or fetal complication such as preeclampsia, intrauterine growth retardation, or preterm delivery. Similar findings were found in Chapman et al who found no difference in fetal outcomes between women with and without autosomal dominant polycystic kidney disease. In contrast in a prospective matched control study by Holley et al, pregnancy losses were found to be higher in women with chronic kidney disease (32%) compared with controls (7%), regardless of the presence of hypertension. One explanation for these findings may be the more sensitive detection of early pregnancy losses with this study design and the inclusion of high proportion of women with non primary kidney disease such as diabetic nephropathy and lupus nephropathy.

Clearly, although improvements have occurred, some degree of fetal compromise in the setting of mild maternal kidney disease continues to exist as represented by higher rates of preterm delivery, low birth weight, and fetal loss compared with normal controls.

Moderate kidney disease (Maternal Serum Creatinine 1.3 to 1.9 mg/dl)

Maternal outcomes

A decline in maternal kidney function during pregnancy is more common in the setting of moderate maternal kidney disease. On average, one fifth to one third of such women may have an accelerated decline in GFR as shown in regression analysis by Jungers et al. Furthermore hypertension and proteinuria were more common, more often worsen, and strongly associated with decline in kidney function. A study by Jones and Hayslett sheds some insight into the impact of pregnancy on maternal kidney function in moderate kidney disease at conception. Overall the mean serum creatinine increased from 1.9 mg/dl to 2.5 mg/dl over the course of pregnancy or up to 6 weeks postpartum ($P<.001$). A pregnancy related loss of kidney function occurred in 50% women. Furthermore, this decline in kidney function persisted for 6 months postpartum in 31% of the study population and 10% of the total group reached end-stage renal disease. Thus women with serum creatinine levels of 1.3 to 1.9 mg/dl at conception are at higher risk of pregnancy associated decline in kidney function.

Fetal outcomes

Significant fetal risks occur in the setting of moderate maternal kidney disease; prematurity, low birth weight, and fetal loss are more likely than with mild maternal chronic kidney disease, and these complications often exceed 50%. In the review by Jones and Hayslett both preterm delivery (55% compared with the average rates of 10%) and fetal mortality (6% increases) were higher. Mean fetal birth weight was reduced, and 37% of infants were below the tenth percentile for birth weight. These observations have also been noted in other contemporary studies. Imbasciati ant Ponticelli found preterm delivery occurring in 50% of pregnancies to mothers with moderate kidney disease; 34% of infants were for small for gestational age. Although fetal compromise is often present, most infants born in these setting do survive.

Severe kidney Disease (Maternal Serum Creatinine >1.9 mg/dl)

Maternal Outcomes:

A paucity of observations exists to characterize the effect of pregnancy on maternal outcomes in women with more severe kidney disease at conception. Nonetheless, it does appear that significant declines in maternal kidney function more consistently occur in this setting than in milder forms of disease and that hypertension and proteinuria are more frequent. Imbasciati et al noted accelerated decline in kidney function occurred in over 25 % of women with severe kidney disease at conception.

In the analysis of Jones and Hayslett, similar reductions in GFR were seen in the moderate and severe kidney disease sub groups. However, it was clearly noted that the risk of GFR decline was highest when maternal serum creatinine >2mg/dl.

Fetal outcomes

Because of infertility and a high rate of early pregnancy failure in women with a serum creatinine > 1.9 mg/ dl, little information exists regarding fetal outcomes in this setting. When pregnancy does occur, prematurity, low birth weight, and neonatal death are more the rule than the exception. In a comparison of moderate with severe kidney disease groups, adverse fetal outcomes were clearly linked to the degree of kidney functional impairment. With moderate kidney disease, the proportional of preterm delivery was 55% and intrauterine growth retardation was 31%. In the setting of severe kidney disease, preterm delivery occurred 73% of the time and intrauterine growth retardation 53% of the time. Cunningham et al noted that sever kidney failure correlated with adverse fetal outcomes means fetal birth weight was 1,520 grams in the setting of severe maternal kidney disease and 2,500 grams in moderate kidney disease, whereas preterm delivery rose from 30% in moderate maternal kidney disease to 86% in severe maternal kidney disease.

Other Factors Related To Pregnancy Outcomes in Primary Maternal Kidney Disease

Etiology of Maternal Primary Kidney Disease

Whether the etiology of maternal primary kidney disease specifically influences maternal and fetal outcomes has been investigated by some. The general consensus is that fetal outcomes are not differentially impacted by different classes of glomerulonephritis, assuming other important factors are similar. The role of kidney disease etiology on changes in maternal kidney function is less clear. Conflicting findings exist in regard to a pregnancy-induced accelerated decline in maternal kidney function with membranoproliferative glomerulonephritis and focal segmental glomerulosclerosis. However, in two of the larger series of patients, Katz, et al and Jungers et al noted the changes in maternal GFR did not appear to differ between types of glomerulonephritis. Hence, although this topic has not been thoroughly studied, no conclusive evidence exists to suggest that maternal or fetal prognosis differs significantly by the underlying cause of kidney disease.

Proteinuria

Transient increases in proteinuria are common during normal pregnancy and even more pronounced during pregnancy complicated by chronic kidney disease. Several studies found increased protein excretion in up to two thirds of women with chronic kidney disease, reaching nephritic range in up to one third of such individuals. Proteinuria not only commonly reflects the degree of kidney damage but also holds prognostic value for progression of kidney disease.

Several investigators have noted that subgroups of pregnant women with chronic kidney disease and proteinuria exceeding 1 gm/day have a greater tendency for accelerated GFR decline and nearly a 2- fold higher incidence of end- stage renal disease.

The most thorough analysis of the role of proteinuria on maternal kidney disease performed by Hemmelder et al. in a retrospective analysis using linear regression analysis, the degree of proteinuria at conception in women with glomerular disease was found to be a significant independent risk factor for accelerated GFR decline in pregnancy. Equally important is the impact of proteinuria on fetal outcomes, an increase in adverse fetal outcome has generally been observed with increasing degrees of proteinuria, especially when proteinuria is within nephrotic range. Barcelo et al observed an inverse relationship between the degrees of proteinuria and fetal birth weight. Comprehensive long term studies evaluating the occurrence, persistence, and significance of proteinuria and microalbuminuria in this setting are needed.

Hypertension

In pregnancies of women with kidney disease, hypertension is more likely to occur and preexisting hypertension often worsens. In the setting of mild maternal kidney disease, new onset hypertension or an exacerbation of existing hypertension occurred in more than 25% of pregnancies. Hypertension is an even more frequent occurrence in moderate and severe kidney disease, present in more than 50% of such pregnancies. The development of hypertension portends a greater likelihood of GFR deterioration. In the review by Imbasciati and Ponticelli, a 3-fold higher prevalence of hypertension was noted in the group with GFR decline during pregnancy. Others have found hypertension to be an independent predictor of maternal GFR decline 9-fold. Hypertension in pregnancies complicated by maternal kidney disease further aggravates fetal outcomes. Many studies have noted that hypertension leads to a 2 to 10- fold increase in adverse fetal events such as prematurity, low birth weight and death. Furthermore, the coexistence oh hypertension and chronic kidney disease appear to have a synergistic effect on poor fetal outcomes. A few studies have even found hypertension to be a more lethal independent risk factor for fetal compromise than mild chronic kidney disease.

Chronic kidney disease clearly plays an important role during pregnancy. Because of advances in medical care, maternal and fetal outcomes in the setting of chronic kidney disease have greatly improved since the initial case series observations in Australia during the 1960s when fetal mortality approached 100%. The 2 most important factors affecting prognosis in this setting are the degree of kidney functional impairment and the presence of hypertension at conception. In the absence of poorly controlled hypertension, moderate to severe primary chronic kidney disease, advanced diabetic nephropathy or active lupus nephropathy, the rates of uncomplicated fetal outcomes are good.

Furthermore, pregnancy does not appear to accelerate the loss of kidney function with mild disease, in part because of the lesser risk of hypertensive complications. As the severity of kidney function and hypertension at conception increase, the likelihood of unfavorable outcomes for both the mother and the fetus grow substantially. Truly, a compelling dilemma confronts women with moderate and severe kidney disease. Although medical care has led to improvements in fetal viability in this setting, a high risk of rapid maternal kidney disease progression continues to exist. Unfortunately, as their fertility declines with progression of their kidney disease over time, future opportunities for pregnancy will become limited. Therefore, it is critical that women of child bearing age with chronic kidney disease be carefully counseled about the risk of pregnancy based on the available epidemiologic data.

Neonatal Mortality

According to the most recent national vital statistics report, the two leading causes of infant death in the United States are congenital malformations and complications of prematurity, accounting for 20% and 175 of infant deaths, respectively. Overall infant mortality rate in the United States in the year 2003 was 0.68%, but the rates were much higher for preterm infants, especially in those born before 32 weeks of gestational age.

Whereas mortality in full term infants (37 weeks or more) was 0.25%, it was 0.85% in those born between 32 and 36 weeks of gestational age and increased considerably (18.8%) in those born at less than 32 weeks of gestational age. Small for gestational age (SGA) infants, defined as the birth weight less than the tenth percentile for gestational age, also have a higher mortality than an appropriately sized infant of the same gestational age.

Compared with general population babies born to mothers with CKD and ESRD are more likely to be born premature or SGA and are at an increased risk for death during the neonatal period. Information about the neonatal outcome of pregnancies in renal transplant recipients comes from National Transplantation Pregnancy Registry (NTPR), a United States – based registry, the European Dialysis and Transplant Association (EDTA) Registry, the United Kingdom Transplant Pregnancy Registry, and a few small case series.

Neonatal outcome in Mothers with Chronic Kidney Disease

Information on the pregnancy outcomes of mothers with CKD is less well organized than the registry data available for ESRD. The CKD information comes mainly from retrospective case series and collectively contains about 1,300 pregnancies from mothers with varied etiologies and stages of CKD. The reported neonatal mortality in these series ranges from 3% to 12%, the rate of preterm births is 16% to 59%, and the incidence of SGA infants is 7% to 37%. Similar to the ESRD data, the rate of preterm births and SGA births in women with CKD appears to increase with worsening maternal kidney function, severe proteinuria, and hypertension.

Neonatal Morbidity

No detailed information is available on short term neonatal morbidity in infants born to mothers with CKD or ESRD, aside from the reported generic “newborn complication” rate of 41% to 54% in babies born to women with functioning kidney transplant. Despite the lack of specific details on neonatal morbidity associated with pregnancies complicated by ESRD and CKD, one can infer the potential outcomes by recognizing the morbidity associated with prematurity, the most common outcome of pregnancy associated with ESRD and CKD.

Despite the achievement of substantial improvement in neonatal care over the past decade, premature infants especially very low birth weight infants (< 1,500 g) continue to be at a very high risk for a variety of clinical complications, such as pneumothorax, respiratory distress syndrome, sepsis, necrotizing enterocolitis, chronic lung disease, deafness and intraventricular hemorrhage. Fortunately the risk of extreme prematurity appears to be small in infants born to mothers with CKD and ESRD, significant morbidity can still occur in even the most mature (>2,500 g) of the premature infant population.

The ability to provide an accurate assessment of neonatal outcome of infants born to women with CKD is made difficult by the lack of good data. Currently, we are aware that pregnancy in women with CKD and ESRD is associated with high rate of preterm birth and SGA infants with resultant increased risk of neonatal mortality. Whether the risks are caused by the specific underlying maternal kidney disease, factors unique to kidney transplantation or dialysis, or one of the many co morbid conditions or required medications is not clear and requires further investigation. Although an increased risk of congenital anomalies does not appear to occur in this population, the information should be viewed with caution until more specific studies can be done to assess the short term impact of newer immunosuppressive regimens and the completion of long term studies to assessing the potential for developmental anomalies and delayed toxicity.

Features of obstetric ARF in two periods

Over a period of 16 years (1982-1997) we collected 131 cases of ARF in pregnant women. Septic abortion was the sole cause of ARF in early pregnancy. In late pregnancy, main causes of ARF were eclampsia and puerperal sepsis. However, the overall main cause of obstetric ARF is preeclampsia. Acute tubular necrosis (ATN) was the predominant histopathologic lesion in pregnancy-related ARF. The overall prevalence of renal cortical necrosis was 15 to 25 percent. The incidence of septic abortion related ARF has declined from 9.3% in 1982-89, to 7% in 1990-97, of total number cases in these periods. Maternal mortality has been also reduced. However, overall prevalence of obstetric ARF has not changed much over a period of 15 years.

Classification and etiology of obstetric ARF

Pregnancy-related ARF in the Indian subcontinent may be classified as follows:

1. ARF in early pregnancy
2. ARF in late pregnancy
3. renal cortical necrosis in pregnancy
4. ARF peculiar to pregnancy
5. miscellaneous cause of ARF during pregnancy

ARF in early pregnancy

ARF in early pregnancy is almost always due to septic abortion. Abortion is only rarely spontaneous; whereas in the vast majority of cases (85%) abortion is conducted by self-trained and unauthorized personnel under unhygienic conditions. Pregnancy is terminated by the insertion of abotifacient chemical (soap, phenol, and quinine), stick, oral drugs or a combination of methods. The legalization of abortion in France followed by a decrease in the percentage of cases of ARF attributable to obstetric causes from 40%in 1978 to 4.5% in 1996. Complications of illegal abortion were a major cause of ARF in Rumania from 1966 to 1989. From 1990 to 1992, after the legalization of abortion, obstetric ARF accounted for only 1.52% of the total cases. In South Africa, with improvement obstetric care, the proportion of obstetric ARF secondary to septic abortion decreased from 65 percent in 1978 to 19 percent in 1991.The factors contributing to renal damage associated with septic include septicemia, nephrotoxic abortifacients, uterine hemorrhage, hypotension intravascular hemolysis, and volume depletion from vomiting, and disseminated intravascular coagulation (DIC).

ARF can occur in the setting of sepsis from any organism following abortion but is most common and most dramatic with infection by clostridium welchii, because of its production of toxins which cause hemolysis and renal failure. The infection may follow a fulminant course characterized by severe abdominal pain and vascular collapse. Clostridium welchii is difficult to culture but characteristic gas in the uterine wall on X-ray is highly suggestive.

The care is supportive, aimed at fluid administration and control of infection. The need for hysterectomy has been debated but can be avoided with early aggressive conservative treatment. Further more, the prevention of unwanted pregnancy and prohibition of illegal abortion are keys to eliminate ARF associated with septic abortion in early pregnancy.

ARF in late pregnancy

Obstetric complications such as eclampsia, abruptio placentae, intrauterine fetal death, ante/postpartum hemorrhage and puerperal sepsis may cause ARF in late pregnancy. Renal hypo perfusion is a major factor responsible for ARF in these patients. The DIC and adult HUS may occur in association with the puerperal sepsis. Decreased glomerular filtration rate and decreased sodium excretion are characteristics of preeclampsia. However, frank ARF is unusual, unless complications such as abruptio-placentae is super imposed on preeclampsia or preeclampsia progresses to develop hemolysis with elevated lever enzymes and low platelet count (HELLP syndrome). In the largest series of patients with HELLP, only 7.7% developed. Women who do not have underlying renal disease or essential hypertension usually recover if they have ARF secondary to preeclampsia when ARF occurs in women with chronic renal disease and renal insufficiency, as many as 80 percent eventually become dialysis dependent.

Renal cortical necrosis (RCN)

Renal cortical necrosis is an uncommon entity that accounts for only 2 percent of all causes. The majority of patients with obstetric ARF follow a course typical of acute tubular necrosis with recovery. However, obstetric complications are the most common causes of renal cortical necrosis. Obstetric causes account for RCN in 56 percent and 65.2 percent of patients reported in two Indian studies. The incidence of renal cortical necrosis ranges between 10 and 30 percent of all cases of obstetric ARF. The overall incidence of cortical necrosis in obstetric ARF was 25 percent in a series. RCN should be suspected in the setting of obstetric ARF with a prolonged period of oligoanuria. The oligoanuria phase may extend for weeks to months and patients with diffuse cortical necrosis may never enter a diuretic phase. The patients with patchy cortical necrosis may have partial recovery but may show later progression to end-stage renal disease.

The gold standard for establishing the diagnosis is the renal biopsy study. Detection of eggshell or cortical tram-track calcification on plain x-ray of abdomen is helpful but is seen only in minority of patients. In recent years, CT scan has emerged as a good non-invasive modality for early diagnosis of renal cortical necrosis.

The characteristic finding is the presence of hypo-attenuating sub capsular rim of renal cortex following contrast injection. In addition, a non-contrast CT scan is more sensitive in picking up the cortical calcification. The diagnosis of RCN is made primarily for prognostic purposes.

Acute renal failure peculiar to pregnancy

There are two uncommon factors peculiar to pregnancy:

a. ARF in association wit acute fatty lever of pregnancy.
b. Post partum hemolytic uremic syndrome.

Acute fatty-lever of pregnancy

It is an obstetric emergency which, if untreated, may progress to a fulminant hepatic failure, a life threatening condition for both the mother and fetus. The disease is fortunately rare most often, a patient presents in the third trimester with complaints of headache, fatigue, malaise, nausea and abdominal pain. Late signs include jaundice, bleeding seizures, hepatic encephalotathy. Hyperbilirubinemia (predominantly conjugated); and slightly elevated serum transaminase and alkaline phosphatase levels are common .Liver biopsy micro vascular fatty infiltration of hepatocytes in a centrilobular distribution. The maternal mortality rate for the disease has improved from 85 percent in the 1970's to less than 20 percent in recent series, with the lowest mortality rate reported being 6.6 percent. The etiology of syndrome is unknown but recent investigations have found the familial genetic defect in fatty acid metabolism.

ARF of some degree occurs in up to 90 percent of patients with acute fatty liver of pregnancy and is due to either to a pre renal mechanism or to acute tubular necrosis. Renal failure is rarely severe enough to require dialysis and death is commonly due to hepatic failure, bleeding or acute pancreatitis rather than renal failure. Treatment includes delivery of the baby and supportive care. Acute renal failure usually resolves post-partum, as does the liver failure. In survivors, no recurrence of acute fatty-liver of pregnancy has been observed in the subsequent pregnancies.

Postpartum hemolytic uremic syndrome(HUS)

The syndrome of HUS is characterized by a micro-angiopathic hemolytic anemia, thrombocytopenia and renal failure. Post-partum HUS referred to as idiopathic post partum renal failure in early reports which usually occurs between as early as day one and as late as three months post-partum. It may occur prior to delivery as well (antepartum HUS). The pathogenesis of the syndrome is multi factorial but vascular endothelial damage appears to be the primary lesion. Familiar occurrence is well known and pregnancy associated HUS has been reported in sisters. Renal failure in postpartum HUS is often severe requiring temporary dialysis. However, some patients develop renal damage and may progress to end-stage. Renal diseases requiring chronic dialysis are transplant.

It should be noted that renal failure, thrombocytopenia and microangiopathic hemolytic anemia may be associated with the other gestational complications such as preeclampsia, HELLP syndrome, thrombotic thrombocytopenic purpura and antepartum HUS. Sometimes there is difficulty in distinguishing HUS from severe preeclampsia accompanied by HELLP syndrome. Some distinguishing features include isolated elevation of LDH in HUS as opposed to elevation of in preeclampsia/HELLP syndrome. Elevation of prothrombin time and partial thromboplastin time are unusual in HUS and suggest preeclampsia. Preeclampsia generally improves following delivery, although there may be transient worsening for 48 hours HUS is not improved by termination of pregnancy. This distinction is important, as preeclampsia resolves supportive care following delivery, whereas HUS is generally irreversible without plasma exchange.

Plasma exchange is the mainstay of treatment of HUS and the related syndrome such as thrombotic thrombocytopenic perpura. Prior to the advent of plasma pheresis the maternal mortality rate was 90 percent. With this treatment, maternal survival has increased to 70-80 percent, with the most clear-cut efficacy in thrombotic thrombocytopenic purpura. Plasma exchange during pregnancy has not shown adverse effects on the fetus.

While advances in treatment have dramatically increased patients survival in obstetric HUS, it still poses a significant threat to both mother and fetus, particularly if not diagnosed and treated promptly. Patients with pregnancy associated HUS should be aware of the possibility of recurrence in future pregnancies or with estrogen containing oral contraceptives. However, at present there is no method to identify those risks.

Miscellaneous causes of ARF

Obstructive ARF has been reported in pregnancies complicated by hydramnious or incarcerated gravid uterus, and even in normal pregnancy. ARF may develop after amniotic fluid embolism, which occurs primarily in multiparas after difficult and prolonged labor. Gravidas with underlying renal disease are more apt to ATN especially in the presents of significant hypertension or superimposed preeclampsia. Finally, ARF may be caused by factors unrelated to pregnancy, such as incompatible blood transfusion, acute glomerulonephritis, and endocarditis or drug nephrotoxicity.

Acute pyelonephritis

It is a relatively rare cause of ARF. Renal failure may be precipitated or aggravated by the use of nonsteroidal anti-inflammatory drugs or potential nephrotoxic antibiotics for example, Amnioglycosides. Early and adequate antibiotic therapy, supportive management and closed supervision should prevent ARF in gravidas with acute pyelonephritis.

Volume contraction

###Volume contraction is an important cause of ARF in pregnancy. In late pregnancy, the causal factor is often blood loss resulting from concealed or over uterine hemorrhages, whereas hyperemesis gravidarum and vomiting late in gestation or much less common causes. The deleterious effects of blood loss are probably maximal in preeclamptic women whose intravascular volume is already relatively contracted, who possibly have relative prostaglandin deficiency with increased reactivity to vasoconstrictive hormones, or in patients with abruptio placentae who show severe coagulation disturbances. Early and appropriate restoration of blood volume should prevent renal failure. Volume depletion from vomiting and sepsis contribute to renal hypo perfusion in patients with septic abortion.

Summary

Acute renal failure (ARF) in pregnancy constitutes about 15-22 percent of all cases of ARF referred for dialysis in India. In contrast, ARF is a rare complication of pregnancy in developed countries. The majority of gravidas (60-70%) develop ARF following septic abortion in India. The remaining gravidas (30-40%) develop ARF due to the complications of late pregnancy, i.e. Eclampsia, intrauterine fetal death, uterine hemorrhage. Pathogenetic factors that predispose to ARF include enhanced vascular reactivity to catecholamine and Angiotensin, renal ischemia due to hypotensive effect of hemorrhage and severe sepsis, intravascular hemolysis, disseminated intravascular coagulation (DIC) and volume contraction. The vast majority cases of obstetric ARF show histologically changes of acute tubular necrosis (ATN). However, renal cortical necrosis (RCN) is observed in 20-25 percent of cases.

The frequency of RCN in association with septic abortion is higher in developing countries as compared to that in the developed ones. The disease carries a high maternal mortality. Sepsis and pulmonary complications are the major causes of death

Materials and methods

All patients who were referred to Department of Nephrology with renal failure complicating pregnancy between Jan 2006 and Jan 2008 were included in this study. Total patients were 52 in number. All of these patients were referred from different parts of Tamilnadu. Most of these patients were referred from Department of Obstetrics attached to our college and from various District headquarters hospital. Their age ranged between 20 and 35 years, the mean being 25.5 years.

Statistical analysis

Variables which were analyzed as qualititative data included Age group, Place of delivery, antenatal check up, pre existing Hypertension, dialysis requirement, duration of dialysis session, birth weight and cause of renal failure. Qualititative data are given in frequencies with their percentages. Quantitative data are given in mean and standard deviation. Quantitative data were analyzed using One way analysis of Variance F – test. Correlation between age and other parameters were analyzed using Pearson's correlation coefficient. Incidence of obstetric ARF was given in proportion with Fisher 95% confidence interval. P value less than 0.05 were taken as significant

Observations

Most of our patients were between 21-25 years contributing more than half of our cases. Patients with Postoperative cause were older, the mean age being 31.6 years. 65.6% of them delivered in hospital and remaining 34.6% delivered in home. 69.2% had antenatal check ups whereas 30.8% of the patients who were referred with renal failure did not have any antenatal check up. 23.1% had preexisting hypertension.75% of total patients required dialysis.

Among the causes of renal failure, Sepsis contributed most of cases accounting for 28.8% of cases. PIH stood as second most common cause contributing19.2% of total cases. Other significant causes include Preexisting renal diseases, HUS, HELLP, Postoperative causes, Ante partum eclampsia, DIC

Pregnant patients with renal failure were analyzed based on their place of delivery. 73.3% of sepsis related cause was delivered at home. 55.6% of patients with preexisting renal diseases delivered at home. Both of patients who had DIC in our study had home delivery.

13 of 52 patients did not require dialysis which included 50% of PIH, 26.7% of Sepsis,50% of Ante partum Eclampsia cases

Duration of dialysis session was correlated with independent cause of renal failure. Three fourth of HELLP and two thirds of Post operative cases required less than 5 sessions whereas two thirds of preexisting renal diseases and 50% of PIH required more than 15 sessions.

Correlating birth weight with independent cause of renal failure in our patients we found that pre existing renal disease patients delivered babies with mean birth weight of 2.21 kg

Two thirds of preexisting renal diseases and 60% of PIH cases did not have any Antenatal check up.

70% of PIH cases and 55.6% of patients with preexisting renal diseases had family history of hypertension.

Discussion

Over a period of 16 years (1982-1997), septic abortion was the sole cause of ARF in early pregnancy. In late pregnancy, main causes of ARF were Eclampsia and puerperal sepsis. The overall prevalence of renal cortical necrosis was 15 to 25 percent. The incidence of septic abortion related ARF has declined from 9.3% in 1982-89, to 7% in 1990-97, of total number cases in these periods. Maternal mortality has also been reduced.

Acute renal failure (ARF) in pregnancy constitutes about 15-22 percent of all cases of ARF referred for dialysis in India. The majority of gravidas (60-70%) develop ARF following septic abortion in India. The remaining gravidas (30-40%) develop ARF due to the complications of late pregnancy, i.e. Eclampsia, intrauterine fetal death, uterine hemorrhage

Our study which was primarily done as single point study revealed sepsis as leading cause. Further it was found that 30.8% of cases did not have any antenatal check up.17% of cases had preexisting renal disease which was detected for first time during their workup of renal failure. Rising incidence of renal disease probably getting reflected by undetected renal disease accounting for nearly 17 % of cases

Among cases of ARF obstetric causes contributed to 8.8% of total number which is more or less in comparison to other countries.

Cause of renal failure	Percent contribution
Sepsis	28
PIH	19
Preexisting renal disease	17
HUS	12
HELLP	8
Postoperative cause	6
Ante partum Eclampsia	4
DIC	4
Hyper emesis Gravidarum	2

Conclusion

Pregnancy-related ARF has virtually disappeared from the economically advanced countries. This progress is chiefly attributed to legalized abortion, consequently, to a decline in septic abortion, and improvement in antenatal care. The incidence of severe ARF in pregnancy is high in the Indian and septic abortion remains the major cause of ARF. Despite legalization of abortion, good number pregnant women seek abortion from untrained and unlicensed abortionist under unhygienic conditions, probably due to social stigma attached to abortion. In contrast to the low rates of cortical necrosis (1.5%). In early pregnancy in western countries its incidence remains high (25%) in India.

The frequency distribution of ARF in pregnancy is still bimodal in Indian subcontinent, where as early peak of ARF in pregnancy has disappeared in the developed countries. Thus, ARF in pregnancy is larger in largely preventable because it is usually the result of obstetric complication and not intrinsic renal disease. With this in mind, pregnancy related acute renal failure could be viewed as a public health problem rather than a nephrological problem.

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STATISTICAL ANALYSIS

Qualitative data were given in frequencies with their percentages.
Quantitative data were given in mean and standard deviation.
Quantitative data were analyzed using one way Analysis of Variance F-test.
Correlation between age and other parameters were analyzed using pearson correlation coefficient.
Incidence of obstetric ARF were given in proportion with Fisher 95% confidence interval
P value less than 0.05 was taken as significant.

		n	%
Age group	<= 20	2	3.8%
	21 - 25	28	53.8%
	26 - 30	16	30.8%
	31 - 35	6	11.5%
Delivered at	Hospital	34	65.4%
	Home	18	34.6%
AN_check_up	No	16	30.8%
	Yes	36	69.2%
Pre_existing_HT	No	40	76.9%
	Yes	12	23.1%
dialysis	No	13	25.0%
	Yes	39	75.0%
Dialysis_session	<=5	11	21.2%
	6 - 10	12	23.1%
	11 - 15	10	19.2%
	> 15	19	36.5%
Birth_ wt	<2.5 kg	9	17.3%
	2.5 - 3.0 kg	34	65.4%
	> 3.0 kg	9	17.3%
Diagnosis	PIH	10	19.2%
	Sepsis	15	28.8%
	HELLP	4	7.7%
	Post op	3	5.8%
	Pre existing renal diseases	9	17.3%
	HUS	6	11.5%
	Ante partum eclampsia	2	3.8%
	DIC	2	3.8%
	Hyperemesis gravidarum	1	1.9%

		Age group							
		<= 20		21 - 25		26 - 30		31 - 35	
		n	%	n	%	n	%	n	%
Diagnosis	PIH			5	50.0%	3	30.0%	2	20.0%
	Sepsis			7	46.7%	6	40.0%	2	13.3%
	HELLP			2	50.0%	2	50.0%		
	Post op					1	33.3%	2	66.7%
	Pre existing renal diseases	1	11.1%	7	77.8%	1	11.1%		
	HUS			4	66.7%	2	33.3%		
	Ante partum eclampsia			2	100.0%				
	DIC			1	50.0%	1	50.0%		
	Hyperemesis gravidarum	1	100.0%						

		Delivered at			
		Hospital		Home	
		n	%	n	%
Diagnosis	PIH	10	100.0%		
	Sepsis	4	26.7%	11	73.3%
	HELLP	4	100.0%		
	Post op	3	100.0%		
	Pre existing renal diseases	4	44.4%	5	55.6%
	HUS	6	100.0%		
	Ante partum eclampsia	2	100.0%		
	DIC			2	100.0%
	Hyperemesis gravidarum	1	100.0%		

		AN_check_up			
		No		Yes	
		n	%	n	%
Diagnosis	PIH	6	60.0%	4	40.0%
	Sepsis			15	100.0%
	HELLP	2	50.0%	2	50.0%
	Post op			3	100.0%
	Pre existing renal diseases	6	66.7%	3	33.3%
	HUS	2	33.3%	4	66.7%
	Ante partum eclampsia			2	100.0%
	DIC			2	100.0%
	Hyperemesis gravidarum			1	100.0%

		Family_History_of_HT			
		No		Yes	
		n	%	n	%
Diagnosis	PIH	3	30.0%	7	70.0%
	Sepsis	15	100.0%		
	HELLP	3	75.0%	1	25.0%
	Post op	2	66.7%	1	33.3%
	Pre existing renal diseases	4	44.4%	5	55.6%
	HUS	6	100.0%		
	Ante partum eclampsia	2	100.0%		
	DIC	2	100.0%		
	Hyperemesis gravidarum	1	100.0%		

		Pre_existing_HT			
		No		Yes	
		n	%	n	%
Diagnosis	PIH	10	100.0%		
	Sepsis	12	80.0%	3	20.0%
	HELLP	4	100.0%		
	Post op	3	100.0%		
	Pre existing renal diseases			9	100.0%
	HUS	6	100.0%		
	Ante partum eclampsia	2	100.0%		
	DIC	2	100.0%		
	Hyperemesis gravidarum	1	100.0%		

		dialysis			
		No		Yes	
		n	%	n	%
Diagnosis	PIH	5	50.0%	5	50.0%
	Sepsis	4	26.7%	11	73.3%
	HELLP			4	100.0%
	Post op			3	100.0%
	Pre existing renal diseases	2	22.2%	7	77.8%
	HUS			6	100.0%
	Ante partum eclampsia	1	50.0%	1	50.0%
	DIC			2	100.0%
	Hyperemesis gravidarum	1	100.0%		

		Dialysis_session							
		<=5		6 - 10		11 - 15		> 15	
		n	%	n	%	n	%	n	%
Diagnosis	PIH	4	40.0%	1	10.0%			5	50.0%
	Sepsis	2	13.3%	5	33.3%	4	26.7%	4	26.7%
	HELLP	3	75.0%	1	25.0%				
	Post op	2	66.7%	1	33.3%				
	Pre existing renal diseases					2	22.2%	7	77.8%
	HUS			3	50.0%	3	50.0%		
	Ante partum eclampsia			1	50.0%			1	50.0%
	DIC					1	50.0%	1	50.0%
	Hyperemesis gravidarum							1	100.0%

		Birth_ wt					
		<2.5 kg		2.5 - 3.0 kg		> 3.0 kg	
		n	%	n	%	n	%
Diagnosis	PIH			10	100.0%		
	Sepsis			8	53.3%	7	46.7%
	HELLP			4	100.0%		
	Post op			1	33.3%	2	66.7%
	Pre existing renal diseases	8	88.9%	1	11.1%		
	HUS			6	100.0%		
	Ante partum eclampsia			2	100.0%		
	DIC			2	100.0%		
	Hyperemesis gravidarum	1	100.0%				

Statistics

	age	Dialysis_session	Birth_ wt
Mean	25.52	9.46	2.723
Std. Deviation	3.622	4.914	.3551
Minimum	18	3	2.0
Maximum	33	21	3.4

Correlations

Age and other parameters	Dialysis_session	Birth_ wt
Pearson Correlation	-.285	.440(**)
Sig. (2-tailed)	.079	.001
N	39	52

** Correlation is significant at the 0.01 level (2-tailed).

Descriptives

age

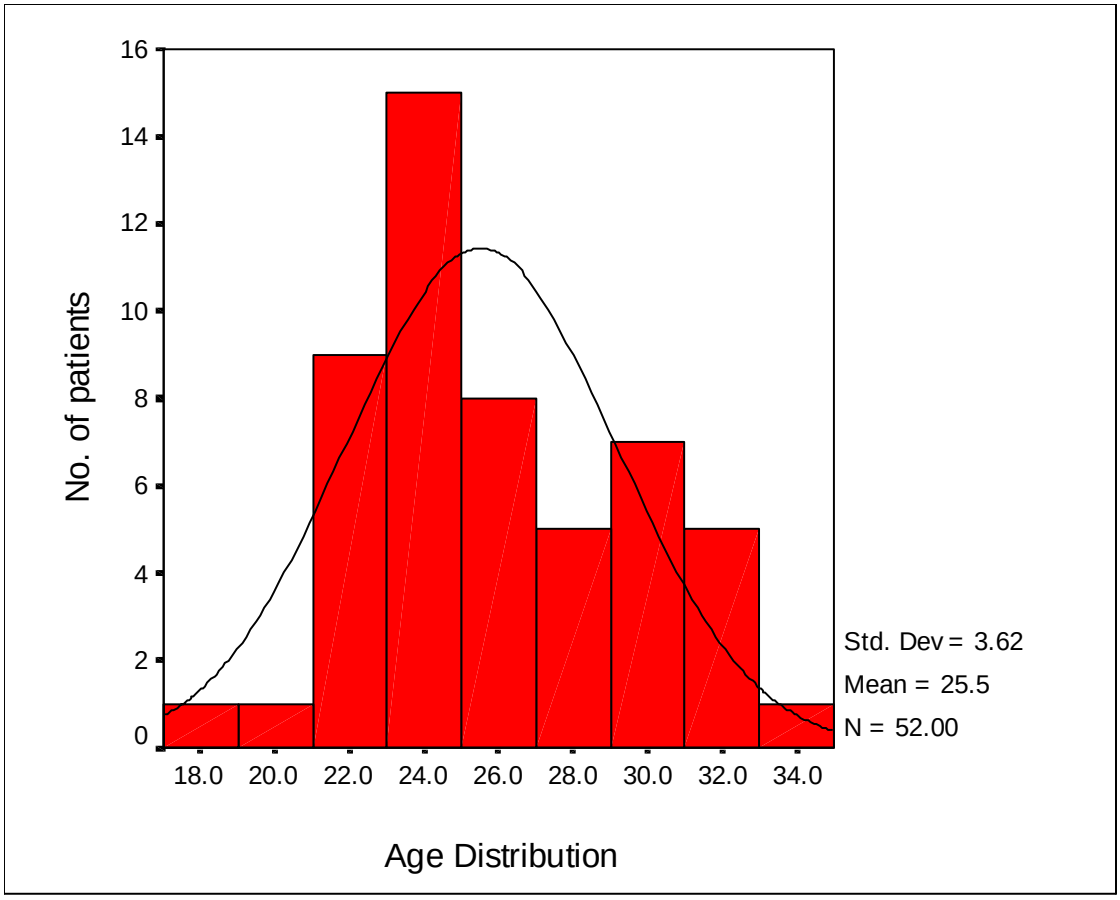
	N	Mean	Std. Deviation	Oneway ANOVA
PIH	10	26.00	4.320	
Sepsis	15	26.60	3.247	
HELLP	4	25.50	2.887	
Post op	3	31.67	1.528	
Pre existing renal diseases	9	23.44	1.944	F=3.37 P=0.004 significant
HUS	6	24.17	2.563	
Ante partum eclampsia	2	22.50	.707	
DIC	2	26.00	1.414	
Hyperemesis gravidarum	1	18.00	.	
Total	52	25.52	3.622	

Dialysis_session

	N	Mean	Std. Deviation	Oneway ANOVA
PIH	5	4.80	.837	
Sepsis	11	8.45	3.616	
HELLP	4	5.25	1.258	
Post op	3	4.33	1.528	
Pre existing renal diseases	7	16.71	2.059	F=16.39 P=0.001 significant
HUS	6	10.83	2.229	
Ante partum eclampsia	1	6.00	.	
DIC	2	15.00	1.414	
Total	39	9.46	4.914	

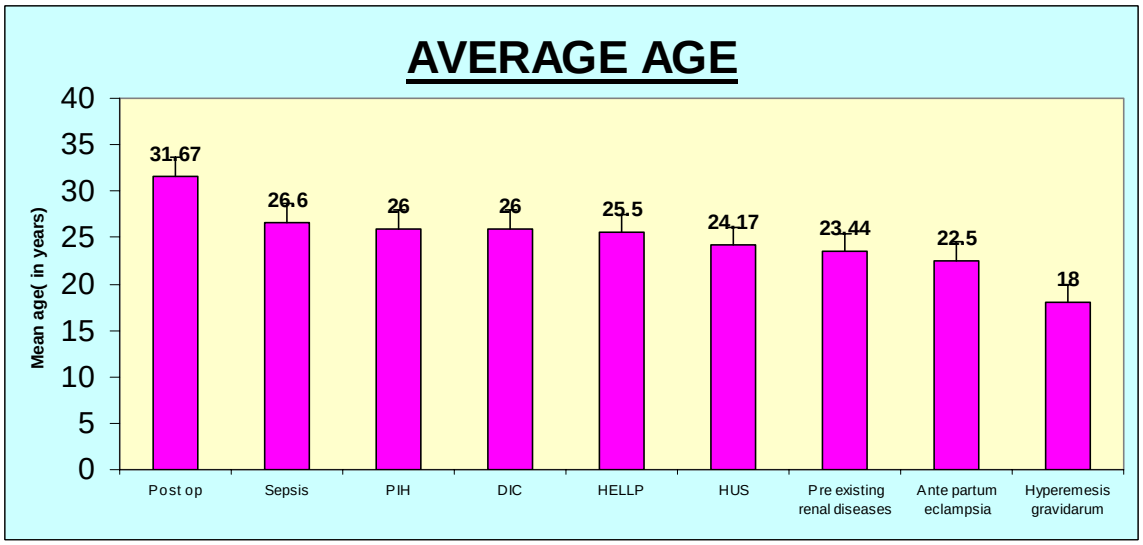
Birth_ wt

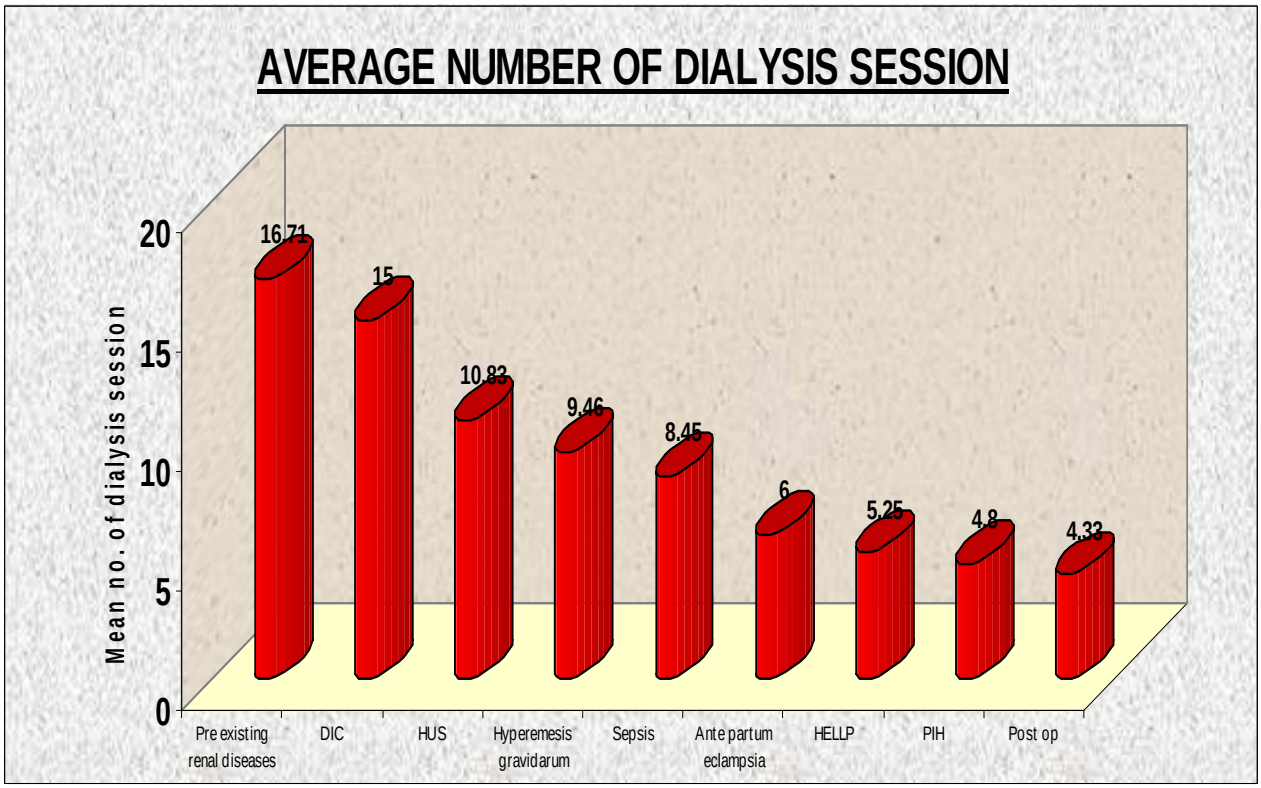
	N	Mean	Std. Deviation	Oneway ANOVA
PIH	10	2.730	.1703	
Sepsis	15	3.000	.2726	
HELLP	4	2.600	.0816	
Post op	3	3.200	.2000	
Pre existing renal diseases	9	2.211	.1833	F=14.4 P=0.001 significant
HUS	6	2.683	.1169	
Ante partum eclampsia	2	2.850	.0707	
DIC	2	2.750	.0707	
Hyperemesis gravidarum	1	2.100	.	
Total	52	2.723	.3551	

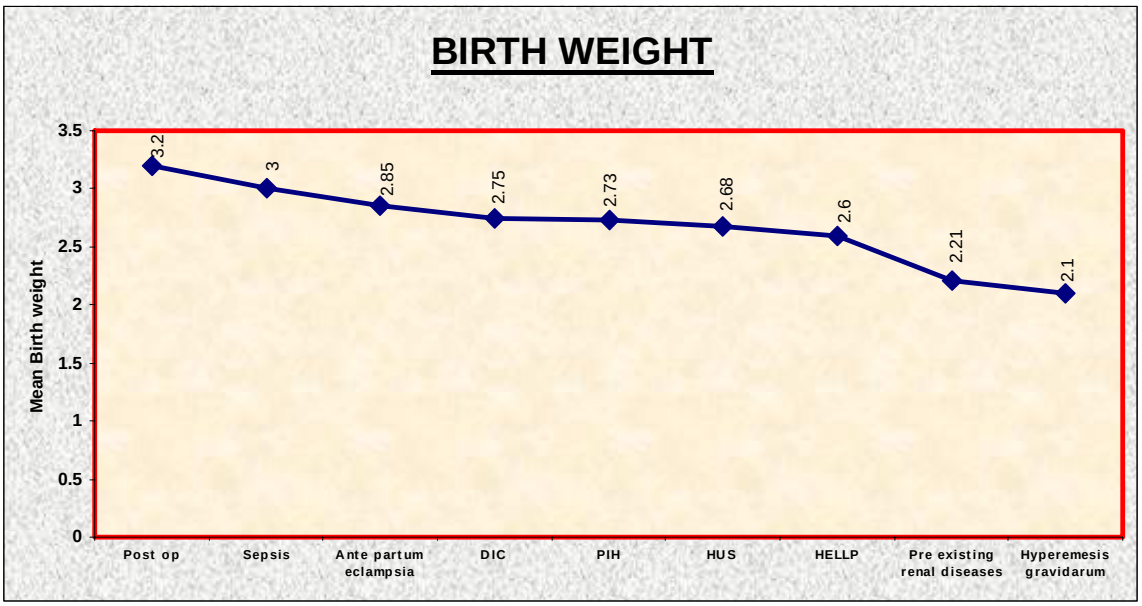

16
14
12
10
8
6
4
2
0
No. of patients
18.0
20.0
22.0
24.0
26.0
28.0
30.0
32.0
34.0
Age Distribution
Std. Dev = 3.62
Mean = 25.5
N = 52.00

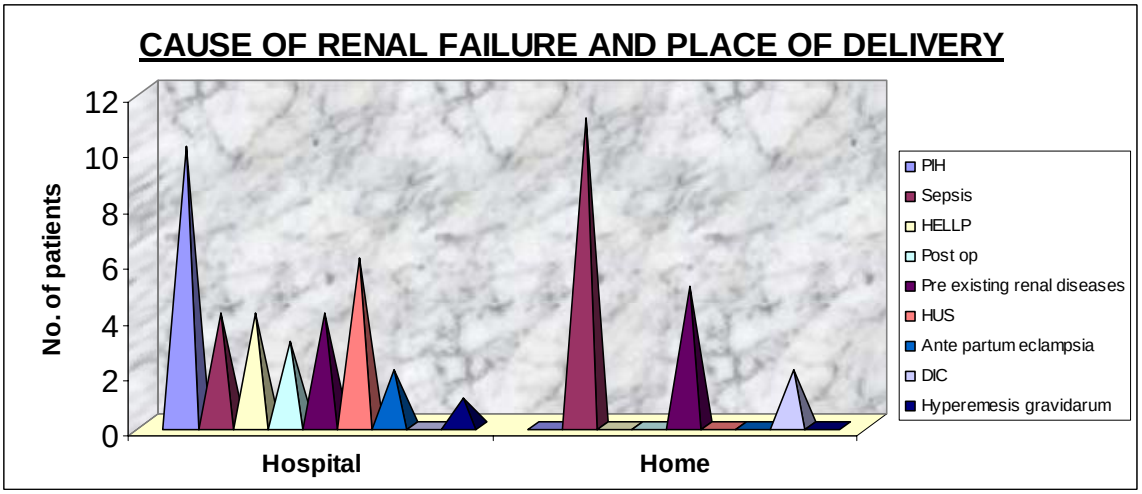
Dialysis_session

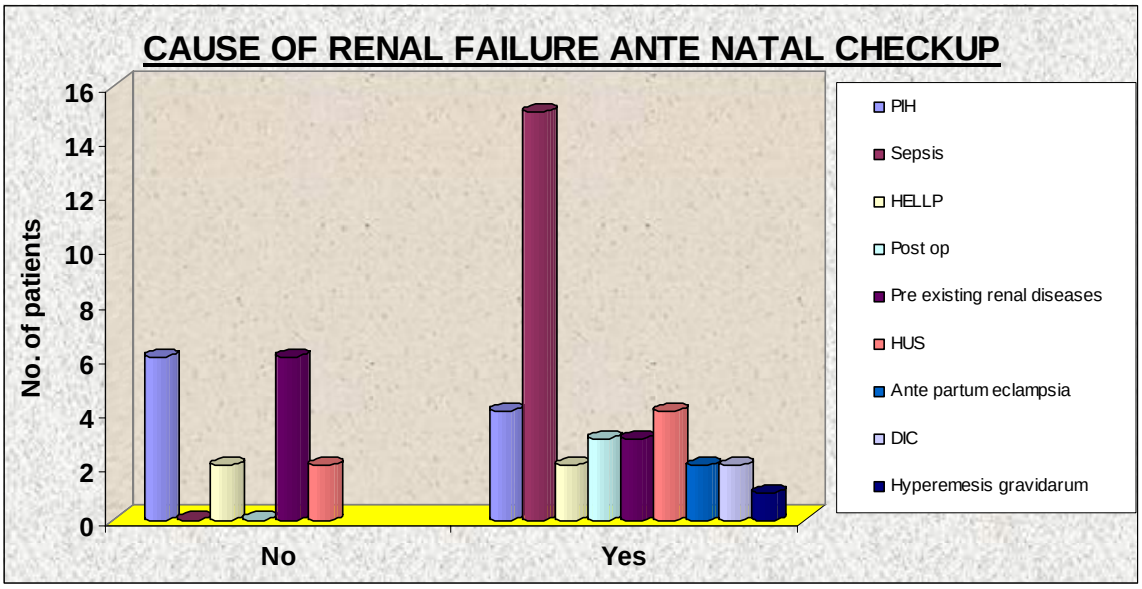
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	3	1	1.9	2.6	2.6
	4	6	11.5	15.4	17.9
	5	4	7.7	10.3	28.2
	6	6	11.5	15.4	43.6
	7	2	3.8	5.1	48.7
	8	1	1.9	2.6	51.3
	9	1	1.9	2.6	53.8
	10	2	3.8	5.1	59.0
	12	5	9.6	12.8	71.8
	14	3	5.8	7.7	79.5
	15	2	3.8	5.1	84.6
	16	3	5.8	7.7	92.3
	17	2	3.8	5.1	97.4
	21	1	1.9	2.6	100.0
	Total	39	75.0	100.0	
Missing	System	13	25.0		
Total		52	100.0		

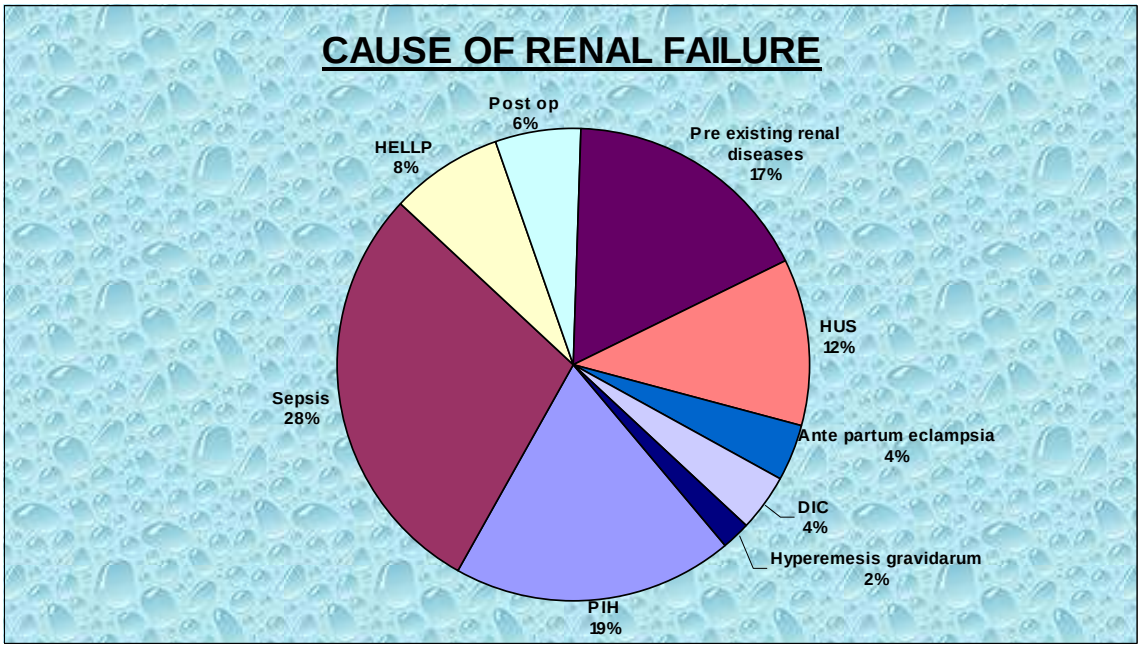

AVERAGE AGE
Mean age(in years)
40
35
30
25
20
15
10
5
0
31.67
26.6
26
26
25.5
24.17
23.44
22.5
18
Post op
Sepsis
PIH
DIC
HELLP
HUS
Pre existing renal diseases
Ante partum eclampsia
Hyperemesis gravidarum

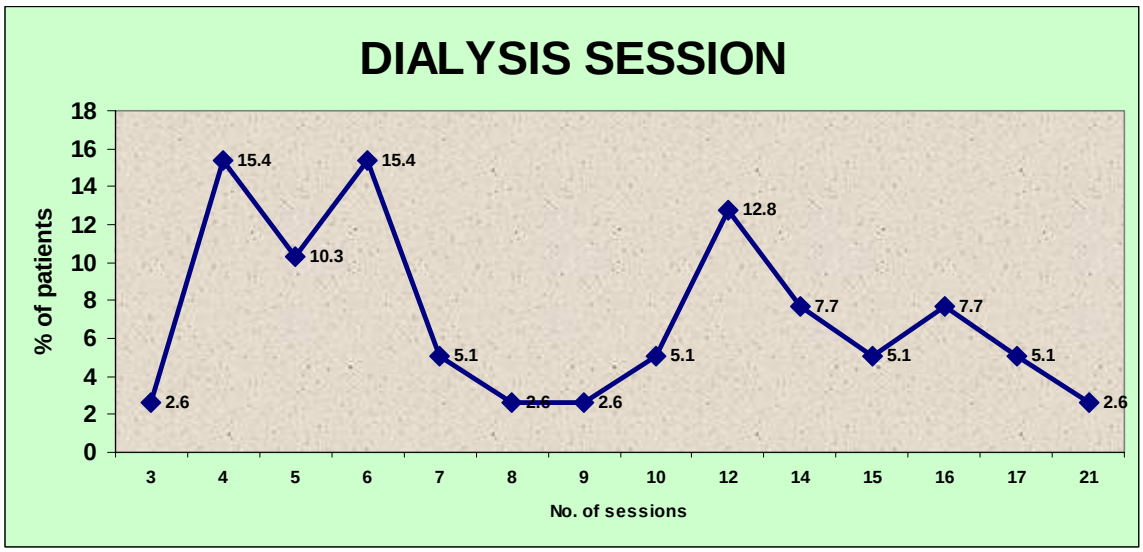

AVERAGE NUMBER OF DIALYSIS SESSION
Mean no. of dialysis session
20
15
10
5
0
16.71
15
10.83
9.46
8.45
6
5.25
4.8
4.33
Pre existing renal diseases
DIC
HUS
Hyperemesis gravidarum
Sepsis
Ante partum eclampsia
HELLP
PIH
Post op


BIRTH WEIGHT
Mean Birth weight
3.5
3
2.5
2
1.5
1
0.5
0
3.2
3
2.85
2.75
2.73
2.68
2.6
2.21
2.1
Post op
Sepsis
Ante partum eclampsia
DIC
PIH
HUS
HELLP
Pre existing renal diseases
Hyperemesis gravidarum


CAUSE OF RENAL FAILURE AND PLACE OF DELIVERY
No. of patients
12
10
8
6
4
2
0
Hospital
Home
PIH
Sepsis
HELLP
Post op
Pre existing renal diseases
HUS
Ante partum eclampsia
DIC
Hyperemesis gravidarum


CAUSE OF RENAL FAILURE ANTE NATAL CHECKUP
No. of patients
16
14
12
10
8
6
4
2
0
No
Yes
PIH
Sepsis
HELLP
Post op
Pre existing renal diseases
HUS
Ante partum eclampsia
DIC
Hyperemesis gravidarum


CAUSE OF RENAL FAILURE
Post op 6%
HELLP 8%
Pre existing renal diseases 17%
HUS 12%
Ante partum eclampsia 4%
DIC 4%
Hyperemesis gravidarum 2%
PIH 19%
Sepsis 28%


DIALYSIS SESSION
% of patients
18
16
14
12
10
8
6
4
2
0
15.4
15.4
10.3
12.8
7.7
7.7
5.1
5.1
5.1
5.1
2.6
2.6
2.6
2.6
3
4
5
6
7
8
9
10
12
14
15
16
17
21
No. of sessions