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Patients with Postpartum Acute Kidney Injury: Clinico-Etiological Profile

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Abstract

Background: Acute kidney injury (AKI) is one of the most challenging and serious complications of pregnancy. It is associated with significant morbidity and mortality in young and often otherwise healthy women. The reported incidence of pregnancy related AKI in the developed countries is 1 to 2.8% while in developing countries it is between 9 to 25%. Acute kidney injury is more often due to pregnancy related diseases.

Materials and Methods: The present observational study was conducted in the Postgraduate Department of Medicine at SMHS Hospital an associated hospital of Government Medical College Srinagar over a period of two years. The study was done to study the clinico-etiological profile of patients with postpartum acute kidney injury and to evaluate the outcome at the end of 3 months of patients with postpartum AKI. After obtaining written informed consent and ethical clearance from Institutional Ethical Committee, patients who developed acute kidney injury in the postpartum period were analysed and statistical analysis was done using Medicalc version 17.

Results: The total number of deliveries conducted in these two years were 36550 which includes 21930 lower segment caesarean section and 14620 normal vaginal deliveries out of which 143 patients were admitted as postpartum acute kidney injury. Out of 143 patients maximum patients were in the age group of 20-24 years (57%) and 63.6% belonged to rural background. In our patients of AKI the value of mean Creatinine was 6.9±3.89mg/dl with maximum of 22.9mg/dl and minimum of 2.4mg/dl. 67.1% of patients had normal sized kidney. On USG abdomen out of 143 patients admitted (11.2%) patients underwent renal biopsy to look for cause of AKI, 50% of biopsied patients had cortical necrosis and 12.5% had vasculitis.

Conclusion: the study shows that the most common cause of postpartum AKI is sepsis including septic shock and urosepsis followed by postpartum haemorrhage, pregnancy induced hypertension and antepartum haemorrhage. 50 percent of biopsied patients had cortical necrosis and 25% had acute tabular necrosis. It is quite clear that by better health care facilities, proper referral to the tertiary care centre, avoidance of unnecessary drugs and early diagnosis lead to better outcome.

Keywords: Acute kidney injury (AKI), Acute tubular necrosis (ATN), Cortical necrosis (CN), Antepartum haemorrhage (APH), Postpartum haemorrhage (PPH).

Introduction

Acute kidney injury (AKI) is one of the most challenging and serious complications of pregnancy. It is associated with significant morbidity and mortality in young and often otherwise healthy women.[1,2] The reported incidence of pregnancy related AKI in the developed countries is 1 to2.8%, while in developing countries it is between 9 to 25%[3]. Since the 1960s, the incidence of pregnancy-related AKI in developed countries has declined from 1 in 3,000 to approximately 1 in 18,000.[1] This trend has been closely linked to improved obstetrical care and dramatic reductions in first-trimester AKI attributable to septic abortions.[4] However, the incidence of pregnancy-related AKI in developing countries like India is up to 25%, and has not changed significantly[5]. ARF is more often due to pregnancy-related diseases. The first peak of incidence, during the first trimester, is dominated by infection and illegal abortion in low-resource countries. The second peak occurs during the third trimester and is related to preeclampsia, hemolysis, elevated liver enzyme, and low platelet count (HELLP) syndrome, acute fatty liver disease of pregnancy, or *postpartum* hemorrhage. Classically, it is believed that AKI occurs in 1% of severe preeclampsia, 3% to 15% of HELLP syndromes[6,7] and 60% in acute fatty liver disease of pregnancy[8], with preeclampsia and HELLP syndrome covering 40% of all cases[9].

AKI in pregnancy bears a high risk of development of bilateral renal cortical necrosis and, consequently, of chronic renal failure. Obstetric complications constitute the most common cause of renal cortical necrosis (50-70%)[10,5,11]. Abruptio placenta, septic abortion, eclamptic toxemia, post-partum hemorrhage (PPH) and puerperal sepsis are the pregnancy-related situations responsible for causing renal cortical necrosis[11].

Both kidney length and volume change during pregnancy. Kidney volume increases by up to 30% as a result of increases in vascular and interstitial spaces, and kidney length increases by 1-1.5 cm with dilatation of the urinary collecting system.[12] Dilatation of the renal pelvises, calyceal systems, and ureters occurs as early as 6 weeks gestation and is typically more pronounced on the right side.[13] Hydronephrosis occurs in up to 80% of women.[13] Urinary stasis in this setting can also predispose women to ascending urinary tract infections such as pyelonephritis.[13]

Several hemodynamic changes also occur within the maternal circulation that contribute to hyperfiltration, the hallmark of healthy adaptation to pregnancy. These include vasodilation, decreased systemic vascular resistance, and decreased arterial pressure, which nadirs between 18 and 24 weeks gestation. Renal plasma flow falls rapidly after 36 weeks gestation, followed by normalization of glomerular filtration rate.[14]

In addition to the effect of hyperfiltration, altered tubular reabsorption is likely also responsible for increased levels of urinary protein[15], glucose,[16] and amino acids[17] during pregnancy. While the upper limit of normal for proteinuria in pregnancy cited in obstetrical guidelines is 300 mg/24-hour period, double the upper limit of normal for the non-gravid state, significant proteinuria in pregnancy is not considered adaptive.[18]

Several other physiologic changes occur during pregnancy that result in mild hyponatremia,[19] mild hypokalemia,[20] and chronic respiratory alkalosis.[21] These, and other anatomic and physiologic changes, typically return to pre-pregnancy state within a few weeks postpartum.

Hyperfiltration in pregnancy results in a physiologic decrease in serum creatinine concentration by an average of 35 micromol/L. The Cockcroft-Gault equation has been shown to underestimate glomerular filtration by 25% in 23% of cases studied.[22] The MDRD equation underestimated glomerular filtration by 25% in 61% of cases studied.[22] The CKD-EPI equation has not been assessed in pregnancy. Considering these limitations, assessment of kidney function in pregnancy is restricted to examining trends in serum creatinine concentration, the interpretation of which is also dependent on the presence of baseline measures of serum creatinine prior to pregnancy.

Acute kidney injury (AKI) is a sudden loss of kidney function, resulting in the retention of urea and other waste products, and dysregulation of fluid and electrolytes.[23] This can result from specific diseases of the kidney (e.g., interstitial nephritis, glomerulonephritis) or extrarenal pathology (e.g. dehydration, heart failure, sepsis, obstruction).[23] In severe cases, this manifests as metabolic acidosis, electrolyte abnormalities, fluid retention, hypertension and, in some cases, clinical symptoms of uremia such as confusion.[24] Only recently have several consensus definition for AKI been developed for use in general population to provide a more standardized quantitative definition of AKI. These include the RIFLE (Risk injury, Failure, Loss, End –Stage Renal Disease)[23] and Acute Kidney Network (AKIN)[25] criteria, and the Kidney Disease: Improving Global Outcomes (KDIGO) modification of the AKIN criteria.[26] These criteria take into account the timing and magnitude of changes in serum creatinine and urine output.

Given the limitations of using serum creatinine – based measures of kidney function in pregnancy, there are obvious limitations to using the serum creatinine – based diagnostic criteria for AKI in pregnancy, despite being widely adopted for use in the general population.[23,25,26] Although a wide range of criteria have been used to define pregnancy-related AKI in previous studies, including RIFLE criteria[27] and hospital diagnosis codes,[28,29] these should be expected to underestimate its true incidence.

Based on data from several diverse populations since the 1960s, the incidence proportion of AKI in pregnancy has declined dramatically from 1 in 3,000 to 1 in 15,000-20,000 worldwide.[1,30,31,32,33] Similarly, the proportion of all AKI cases accounted for by pregnancy related causes has dropped from 20-40% to 2-10%.[1,31,32] Short-term maternal morbidity after pregnancy-related AKI and dialysis dependence among survivors has also nearly disappeared, previously affecting as many as 30% and 11% of cases respectively. These trends have been closely linked to improved obstetrical care in developed countries and dramatic reductions in first-trimester AKI attributable to fewer septic abortions with the legalization of abortion.[1]

There are several conditions known to precipitate AKI that are either unique to pregnancy or worsened by the gravid state. Unfortunately, there are limited data on the incidence, risk factors, and outcomes of these conditions, including the associated risk of AKI.

Volume depletion from any cause can precipitate AKI in pregnancy through pre-renal ischemia or acute tubular necrosis. Prior to 20 weeks gestation, this may be due to hyperemesis gravidarum or obstetrical hemorrhage due to a spontaneous or induced abortion.[34] Later in pregnancy, cardiogenic shock as a result of an amniotic fluid embolism, obstetrical hemorrhage from placental abruption, or postpartum hemorrhage are more common.[34] Sepsis due to chorioamnionitis, endomyometritis, or

pyelonephritis can lead to AKI in pregnancy through similar mechanisms.[32,35]

Preeclampsia refers to the new onset of hypertension and either proteinuria or end-organ dysfunction after 20 weeks gestation.[18] The frequency of preeclampsia ranges from 2% to 7% in healthy nulliparous women and increases substantially in women with advanced maternal age, multifetal gestation, chronic hypertension, diabetes mellitus, and chronic kidney disease.[36,37] Preeclampsia is the leading cause of maternal morbidity and mortality worldwide and is the most common cause of AKI during pregnancy.

Although much less common, thrombotic microangiopathy is another important cause of AKI in pregnancy; which, like preeclampsia, typically presents after 20 weeks gestation. Thrombotic microangiopathy is a pathological process characterized by microangiopathic hemolytic anemia, thrombocytopenia, and variable signs of organ injury from endothelial damage and thrombosis.[38] Thrombotic microangiopathy is a defining feature of thrombotic thrombocytopenic purpura (TTP), a condition caused by a congenital or acquired deficiency in ADAMTS13.[39] However, thrombotic microangiopathies may be caused by other mechanisms, including enteric infection with Shiga toxin (known as hemolytic uremic syndrome [HUS]), drug-mediated toxicity or immune reaction, and complement dysregulation (known as atypical HUS) or may be the manifestation of several other underlying disorders in up to 60% of cases.[39] AKI is most common among patients with atypical HUS with severe AKI requiring dialysis reported in over 80% of cases.[40]

Management of AKI is supportive and is focused on identification and treatment of the underlying cause (which, depending on the cause in pregnancy, may necessitate early delivery of the fetus).[18,34] However, if conservative therapy fails to control complications such as acidosis, hyperkalemia, volume overload, or symptomatic uremia, dialysis may be indicated.[26] In a population-based study of hospital deliveries in Canada between 2003 and 2011, the proportion of pregnancies affected by AKI that required dialysis was 8.8% and maternal death occurred in 2.8% of cases.[41]

Aims and Objectives

1. To study the clinico-etiological profile of patients with postpartum acute kidney injury.
2. To evaluate the outcome at the end of 3 months of patient with postpartum AKI.

Materials and Methods

A prospective observational study of patients with postpartum AKI was conducted over a period of one and a half year in the Department of Medicine in collaboration with the Department of Obstetrics and Gynaecology, Govt. Medical College, Srinagar.

After obtaining written informed consent and ethical clearance from Institutional Ethics Committee, patients who developed acute kidney injury in the postpartum period were analyzed.

Exclusion Criteria

All patients with pre-existing renal insufficiency, hypertension, chronic liver disease, cardiac disease, diabetes, history of renal stone disease, small size of kidneys were excluded.

For each case, a detailed history, thorough physical examination like temperature, pulse rate, blood pressure, fluid intake and urine output were recorded. Relevant laboratory investigations such as complete hemogram, blood urea, serum creatinine, electrolytes, coagulation profile, liver function test, 24-hour urinary protein, and ultrasound abdomen were carried out. Blood culture and

vaginal swab were taken for culture and sensitivity in patients with septicemia.

Acute kidney injury (AKI) was diagnosed by history of sudden onset oliguria (urine output< 400ml/24hrs) or anuria, with an increase in serum creatinine of more than 0.3mg/dl/day from baseline based on AKIN criteria. Post-partum phase was defined as the period that begins immediately after delivery and extends upto 6 weeks.

Obstetric records including parity data, pregnancy-related disorders such as pre-eclampsia (defined by a set of three signs including hypertension, edema and proteinuria after 20 weeks of gestation), eclampsia (defined as the onset of grand mal seizure in a patient with preeclampsia), HELLP syndrome (defined as laboratory evidence of hemolysis, elevation of enzymes and low platelet count in pre-eclamptic women), puerperal sepsis, other infections, and delivery information (route of delivery and associated complications) were analyzed. The diagnosis of severe sepsis was defined by using the definitions of the American college of chest physicians and the society of critical care medicine. DIC was defined as coagulation of blood in small blood vessels with bleeding that resulted from the consumption of coagulation proteins and platelets. Postpartum hemorrhage was diagnosed when blood loss in the first 24 h after delivery was greater than 500ml following vaginal delivery and 1000ml following cesarean section. Thrombotic microangiopathy defined by microangiopathic hemolytic anemia, low platelet count, renal failure and elevated LDH.

Hemodialysis or peritoneal dialysis was done when indicated. Renal biopsy was done in non-recovering AKI lasting > 3 weeks. Maternal outcome was recorded as full recovery, partial recovery, end stage renal failure or death.

- Complete recovery in which creatinine dropped to ≤1.4 mg/dl at the time of discharge/or on follow-up of 12 weeks.
- Partial recovery in which creatinine did not return to 1.4 mg/dl or less after 12 weeks of follow-up or those who lost follow-up.
- Dialysis-dependent: Patients who persist to be anuric and needed dialysis continuously at the time of discharge and on follow-up for more than 3 months and patients with biopsy showing diffuse cortical necrosis.
- Death End stage renal disease was diagnosed in patients with impaired renal functions for more than 3 months.

Statistical Analysis

Continuous variables were summarized as mean and standard deviation. Categorical variables were summarized as frequency and percentages. Data analysis was done using MedCalc version 17.

Results

The present prospective study was conducted on postpartum females with acute kidney injury over a period of one and a half year. The total number of deliveries conducted in this period were 36550 which includes 21930 lower segment caesarean section and 14620 normal vaginal deliveries, out of which 143 patients were admitted as postpartum Acute kidney injury.

Table 1: Age Distribution

Age Groups (in years)	Frequency *(n)*	Percentage (%)
15-19	1	0.7
20-24	57	39.86
25-29	54	37.76

30-34	31	21.67
TOTAL	143	100.0

Out of 143 patients admitted as postpartum acute kidney injury maximum patients were in the age group of 20-24 years (57%). The mean age in our study group was 26.04 ± 3.605 years with minimum and maximum age 34 years and of 19 years respectively.

In our studied population, 63.6% patients belong to rural background while 36.4% had urban background. According to modified Kuppuswamy scale, most of the patients belong to lower middle class (46.2%) followed by upper middle class (28.7%). Out of 143 patients, 57.3% had undergone caesarian section as the mode of delivery while 42.7% had normal vaginal delivery. Out of our study population, 11.1% of the patients had history of NSAID intake in the form of injectables for relief of postoperative pain and 9.1% has history of intake of labetolol for pregnancy induced hypertension.

In our study population, 42.7% of the patients had no significant history on admission while postpartum haemorrhage was seen in 29.4% and pregnancy induced hypertension was seen in 15.4% of the patients. Eclampsia was seen in 2.1% of the patients.

Table 2: Post-delivery Complications

Post Delivery Complications	**Frequency *(n)***	**Percentage (%)**
Convulsion	6	4.2
Hysterectomy	1	0.7
Intubated	4	2.8
IUD	5	3.5
LVF/ ACC HTN	1	0.7
No complication	82	57.3
Sepsis	25	17.5
Septic shock	14	9.8
Stitch hematoma	1	0.7
Urosepsis	4	2.8
Total	143	100.0

Majority of the studied patients (57.3%) had no post-delivery complications while sepsis was observed in 17.5% of the patients with septic shock in approximately 9.8% of the patients and intrauterine death occurring in 3.5%. Out of total no of patients admitted in the hospital majority of the patients i.e. 83.2% of the patients were found to have anaemia predominantly iron deficiency anaemia.

Table 3: Kidney Function Tests

Creatinine (mg/dl)	**Frequency (n)**	**Percentage (%)**
2-4	22	15.4
4-6	58	40.5
6-8	29	20.9
8-10	12	8.4
10-12	7	4.9
12-14	7	4.9
14-16	0	0
16-18	4	2.8
18-20	2	1.4
20-22	0	0
22-24	2	1.4
TOTAL	143	100.0

In our patients of acute kidney injury the value of mean creatinine was 6.9±3.89 mg/dl with maximum of 22.9mg/dl and minimum of 2.4mg/dl with most of the patients having values of 4-6 while mean urea was found out to be 118.34 mg/dl ± 57.3 mg/dl with maximum of 340mg/dl and minimum value of 48mgldl.

Table 4: USG KIDNEY		
USG Findings	**Frequency *(n)***	**Percentage (%)**
Ascites	1	.7
Empyema	1	.7
Gb stone	1	.7
Normal	96	67.1
Pleural effusion	12	8.4
Renal parenchymal disease	8	5.6
RPOC	24	16.8
Total	143	100.0

During hospital admission ultrasound of abdomen and pelvis conducted in every patient revealed normal size of the kidneys in 67.1%of the patients while 16.8% of the patients had retained products of conception and 5.6% of the patients had renal parenchymal disease.

Table 5: Renal biopsy		
	Frequency *(n)*	**Percentage (%)**
NO	127	88.8
YES	16	11.2
Total	143	100.0

Out of 143 patients admitted 11.2% patients underwent renal biopsy to look for the cause of acute kidney injury and in those patients who did not respond to treatment and showed rapid rise in creatinine beyond 3 weeks of admission.

Table 6: Biopsy findings of biopsied patients		
	Frequency *(n)*	**Percentage (%)**
ATN	4	25.0
Cortical Necrosis	8	50.0
Vasculitis	2	12.5
TMA	2	12.5
Total	16	100.0

Out of 16 patients who underwent renal biopsy 8 (50%) of the patients were seen to have cortical necrosis, 4 (25%) had acute tubular necrosis followed by thrombotic microangiopathy and vasculitis in 2 (12.5%).

Table 7: Recovery			
		Frequency *(n)*	**Percentage (%)**
Dialysis dependent		30	21.0
Death		9	6.3
Recovery	Complete	73	51.0
	Partial	31	21.7
Total		143	100.0

Out of 143 patients admitted as postpartum acute kidney injury, majority of the patients i.e. 73 (51.0%) recovered completely while as 31 (21.7%) recovered partially. 30 (21%) patients became dialysis dependent and 9 (6.3%) died.

Discussion

Obstetrical AKI is now a rare entity in the developed countries. The incidence of AKI in pregnancy has drastically decreased in the past 50 years from 20 to 40% in 1960 to <10% in recent series due to meticulous antenatal management. No case of AKI was observed in 12,000 and 20,000 births, respectively, in two studies reported from western countries[1,42]. In developing countries, AKI in pregnancy is on decreasing trend including India but is still common in some parts of the developing countries. Recent epidemiological studies have

confirmed the decreasing incidence of PRAKI in India, with a decrease from 14.5% in 1987 to 4.3% in 2005[43,44].

In our study a total of 36550 deliveries took place in one a half year including 21930 lower segment caesarean section and 14620 normal vaginal deliveries. 143 patients were admitted as postpartum acute kidney injury reporting an incidence of 4 per 100,000 of pregnant population. Huang C[45] reported an incidence of 0.81% in their study of AKI during pregnancy and puerperium. Gullipalli *et al*.[46] reported an incidence of 48 (10.5%) from Andhra Pradesh in 2014. Pahwa *et al*.[47] reported an incidence of 27 (3.59%) from Madhya Pradesh in 2012.

Out of total 143 patients admitted as postpartum acute kidney injury in our study most of the patients were seen in age group of 20 years to 24 years with mean age of 26 years with minimum age 19 years and maximum of 34 years. KR Goplani et al[48] in a study of pregnancy related AKI done in Department of Nephrology, Institute of Kidney Disease and Research Centre and Dr HL Trivedi Institute of Transplantation Sciences, Civil Hospital, Asarwa, Ahmedabad, Gujarat, India stated that the mean age of patients with pregnancy-related ARF was 25.6 years. The youngest patient was 20 years old and the eldest was 35 years old. The possible reasons for a very high incidence of ARF in postpartum period were poor socioeconomic status, ignorance, and non-availability of equipped hospitals for management of complicated obstetrical complication and a long time needed in travelling to reach the hospitals. Most of our patients were referred from periphery district hospitals after delivery in view of lack of adequate resuscitation and health care facilities.

In our study majority of the patients had sepsis as the cause of AKI that was seen in 25 patients i.e. 17.5% with septic shock occurring in approximately in 14 patients and urosepsis in 4patients i.e. 2.8%. Similar to the studies which were carried in rest of India like Gullipalli *et al*.[49] reported puerperal sepsis in 16 (33.33%) and uterine hemorrhage including post- and ante-partum hemorrhage as the causes for AKI in 18.88% of patients as causes contributing to PP-AKI in their study. Pahwa *et al*.[50] reported sepsis in 70.3% and PPH in 22.2% patients of PP-AKI in their study. Naqvi *et al*.[51] reported sepsis as the cause of PR-AKI in 24% and PPH in 28.03% of cases.

When looking for significant risk factors and history of any comorbidities, it was found that postpartum hemorrhage was seen in 42 patients i.e. 29.4% while 22 patients gave history of pregnancy induced hypertension complicated by eclampsia in 3 patients, accounting for two most common causes of kidney injury in postpartum period followed by antepartum haemorrhage in 15 patients i.e. 10.5%. Gurrieri et al.[52] identified 55 cases of ARF over a 5-year period, seven of which were related to HELLP syndrome. Sibai et al.[6] documented an incidence of ARF of 7.4% among HELLP syndrome patients

Prasad Gullipalli et al[49] carried out a study of spectrum of postpartum acute kidney injury, concluded that Puerperal sepsis was the most common cause of Acute kidney injury observed in 16 (33.33%) patients. Preeclampsia/eclampsia/HELLP syndrome contributed in 12 (25%), postpartum hemorrhage in 6 (12.5%).

In a recent study carried in Canada, obstetric acute renal failure increased significantly, from 1.6 per 10 000 deliveries in 2003 to 2.3 per 10 000 deliveries in 2007[2], whereas the rate in the United States increased from 2.3 in 1998 to 4.5 per 10 000 deliveries in 2008[53]. These increases are of concern because obstetric acute renal failure is associated with high rates of maternal morbidity and a case fatality rate of 2.9%. Major risk factors for obstetric acute renal failure include chronic hypertensive

disease, pre-eclampsia, postpartum haemorrhage, antepartum haemorrhage, sepsis, and other infections[54,55,56,57].

We also observed that patients who took NSAIDS in the form of injectables in the periphery district hospitals for the relief of postoperative pain showed a significant rise in creatinine causing acute kidney injury as 11.1% of our patients had history of NSAID intake. Adverse renal effects from these drugs are caused by two distinct pathological entities. The first mechanism of acute kidney injury (AKI) from NSAIDs is due to reduced renal plasma flow caused by a decrease in prostaglandins, which regulate vasodilation at the glomerular level. NSAIDs disrupt the compensatory vasodilation response of renal prostaglandins to vasoconstrictor hormones released by the body[93]. Inhibition of renal prostaglandins results in acute deterioration of renal function after ingestion of NSAIDs. The second mechanism of AKI is acute interstitial nephritis (AIN), which is characterized by the presence of an inflammatory cell infiltrate in the interstitium of the kidney. AIN is caused by an immunological reaction after NSAID exposure of about a week[58,59,60]. AIN is now recognized as a major cause of drug induced AKI and accounts for about 15% of all patients with unexplained AKI. As an entity, AIN due to NSAIDs is under recognized as well.

Out of 16 patients who underwent renal biopsy 8 (50%) of our patients were seen to have cortical necrosis followed by acute tubular necrosis in 4 patients (25%), thrombotic microangiopathy and vasculitis in 2 patients (12.5%).

In our study, out Of 143 Patients admitted as postpartum acute kidney injury majority of the patients i.e. 73 (51%) completely recovered while as 31 (21.7%) recovered partially. 30 (21%) of the patients became dialysis dependent and 9 (6.3%) patients died. In a single-center experience study on pregnancy related acute kidney injury by KR Goplani et al[48] the maternal mortality was 18.57%, while in a previous study conducted in India, it was approximately 30%. Chugh KS *et al*[61], Kumar *et al*[62] recently reported a maternal mortality rate of 24%. This appears to be the result of aseptic delivery practices and early management of antepartum and postpartum hemorrhages.

Overall in our prospective study carried in a period of 18 months at a tertiary care centre we observed that unlike developed countries where the most common cause was pregnancy induced hypertension complicated by eclampsia and HELLP syndrome followed by postpartum haemmohage and antepartum haemorrhage, in developing countries like India or other low income areas in the country as seen in our study carried in Kashmir the most common cause of postpartum AKI was sepsis including puerperal sepsis, septic shock, urosepsis followed by postpartum haemmorhage and PIH indicating that in our part of the world the damage is reversible and preventable by improving the basic health care facilities, sanitation, early shifting the patients to higher centre from periphery and experienced staff personal.

Conclusion

In our prospective study of 18 months we found that the incidence of postpartum AKI has decreased due to early referral to a tertiary care centre, therefore is preventable and treatable. Out of 143 patients, 73 (51%) of the patients recovered completely while as 31 (21.7%) patients partially recovered. 30 (21%) patients became dialysis dependent and 9 (6.3%) patients died. The most common cause of postpartum AKI is sepsis including septic shock and urosepsis followed by postpartum haemmorhage, pregnancy induced hypertension and antepartum haemmorhage. Sixteen patients underwent renal biopsy

out of which 8 patients have cortical necrosis followed by acute tubular necrosis in 4 patients, TMA and vasculitis in 2 patients each. It is quite clear that by better health care facilities, proper referral to the tertiary care centre, avoidance of unnecessary drugs and early diagnosis leads to better outcome.

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