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"CLINICAL AND ETIOLOGICAL STUDY OF PREGNANCYRELATEDACUTEKIDNEYINJURY"

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ABSTRACT

BACKGROUNDANDOBJECTIVES

Acute Kidney Injury as a complication in pregnancy contributes to a high rate of morbidity and mortality in our country. The aim of this study is to analyze the variouscausesofacutekidneyinjuryinpregnancy,thefactorsaffectingitscourseand to determine the outcome of pregnancy in the affected cases. This is necessary to help in the early diagnosis and prevention of complications to the mother and the child.

OBJECTIVES

The objectives are to study the etiological profile of acute kidney injury in pregnancy and to study the clinical course of acute kidney injury in pregnancy.

METHODS

This is a prospective cohort study, conducted over a period of one and half years with a total of 53 pregnant patients with acute kidney injury admitted to a tertiary care hospital and those without previous history of hypertension, diabetes mellitus or renal disease. The subjects were observed and serum urea, creatinine and hemoglobin levels assessed and progress noted. The final prognosis was then charted and analyzed.

RESULTS

Ourstudyshowedthat43.39%patientsbelongedtotheagegroupsof 26-30 years and 47.1% were primi patients. 85% recovered and only 15 % mortality was seen in the study group.60.38% had blood urea levels between 40-100 mg/dl and 47.12%hadbloodcreatininelevelsbetween1-2mg/dl.49%patientshadhemoglobin levels between 7-9.9mg/dl. Most common cause for acute kidney injury in our studyis due to hypertensive disorders of pregnancy.

INTERPRETATIONANDCONCLUSION

This concludes that pre-eclampsia is the most frequent etiology followed by post-partum hemorrhage and abruptio placenta. This concludes that prevention is the best and least expensive solution. Prenatal care and greater access to emergency obstetric care services could decrease pregnancy related acute kidney injury and its consequences.

KEYWORDS: Acutekidneyinjury;Pre-eclampsia;Pre-natalcare

INTRODUCTION

AKI is a sudden loss of kidney function, resulting in the retention of urea and other waste products, and dysregulation of fluid and electrolytes.[1] This can resultfrom specific diseases of the kidney (e.g., interstitial nephritis, glomerulonephritis) or extra renal pathology (e.g. dehydration, heart failure, sepsis, obstruction).[1] Recoveryis the rule when it is diagnosed and treated early.

RenalEtiology of AcuteKidney Injury in Pregnancy

Clinical investigation for the cause of AKI in pregnancy is the same as in the nonpregnant population, which includes consideration of pre-renal, renal, and post-renal etiologies.

Thecausesmaybe

Pre Renal

- Hemorrhage
- Hyperemesisgravidarum
- Congestiveheartfailure

Renal

- Sepsis
- Acute tubular necrosis(ATN)
- Pyelonephritis
- Renal cortical necrosis
- Thromboticmicro angiopathy
- Pre eclampsia
- HELLPSyndrome
- Acute fatty liver of pregnancy(AFLP)
- Glomerulonephritis

Medications

Post Renal

- Obstruction

In developing countries like India, AKI remains a frequent and grave complication of pregnancy associated with maternal and fetal mortality. There has been decline in the incidence due to improvement in the antenatal care, earlydiagnosis and also of the legalization of abortion.[2]

The aim of this study is to analyse the various causes of acute kidney injury in

pregnancy, the factors affecting its course and to determine the outcome of pregnancy among the pregnant women treated at Mysore medical college and research institute, Mysore.

NeedforStudy

Pregnancy induced acute kidney injury not only negatively affect pregnancy outcomes but have a relevant effect on the future health of affected mothers and their children. Pregnancy induced acute kidney injury merits special attention because it involves risk to two lives.

Pregnancy induced acute kidney injury is of concern because it is associated with high rates of maternal morbidity and a case fatality rate of 2.9%.[3]

Pregnancy induced acute kidney injury is also associated with significant fetal mortality and morbidity. The odds of perinatal mortality increases 4 fold when compared with pregnancies without pregnancy induced acute kidney injury.[4]

Studies from India have reported high perinatal mortality of 20% to 45% due to intrauterine death,still birth, and pre maturity.[5]

Good antenatal care, early identification and correction of problem leading to acutekidneyinjurybyappropriatetertiarycaremanagementwillreducetheincidence of acute kidney injury in pregnancy.[6]

OBJECTIVES

1. Tostudy etiological profile ofacutekidneyinjuryinpregnancy.

2. Studytheclinicalcourseofacute kidneyinjuryin pregnancy.

METHODOLOGY

Sourceofdata

Primarysourceofinformation-PregnantpatientswithAKIadmittedtotertiary care hospital, Mysore.

Secondary source of information from published articles journals, books, related websites are used in planning and developing synopsis and during dissertation as supporting documents.

StudyDesign: Prospectivecohortstudy.

StudyPeriod: AperiodofoneandahalfyearbetweenJan2020toJuly2021.

Sample Size: 53 cases

SAMPLESIZE

Samplesizewasdeterminedbasedon

- WhenpropotionwithsepsisamongAKIis25%

Description

- Theconfidencelevelisestimatedat95%
- Withaz-valueof1.96
- The confidence interval or margin of error is estimated at ±5, when proportion with sepsis among AKI is 25% Assuming p% =25% and q%=75%

$n=p\%xq\%x[z/e\%]^2 n=53$

Inclusion Criteria

- All pregnant patients with acute kidney injury admitted to tertiary care hospital,Mysore.

Exclusion Criteria

Allpatientswithpreviousshistoryof

1. Hypertension
2. Diabetes mellitus
3. Renal disease

METHOD

DATACOLLECTION

- AllpatientswithPRAKIattendingourinstitutionoveraperiodofoneanda half-years were included in the study.
- Patientswithpre-existingrenaldiseasewereexcludedfromthe study.
- AKI:serumcreatinineincreasedabout1.5timesfromthebaselinewhichisknowntohave occurredwithinpriorsevendaysorincreaseinserumcreatinineby >0.3mg/dlwithin48hoursorwhentheurineoutputdecreasedtolessthan <0.5ml/kg/hourforsix hours

StatisticalAnalysis

Data obtained from the study will be entered in excel sheets and it will be double checked. Data analyzed using SPSS software version 22.0 and will be presentedasdescriptivestatisticsintheformoffrequencytables,figuresand graphs.

SAMPLESIZEESTIMATION

Samplesizeisestimatedbasedontheformula

$$n=p\%xq\%x[z/e\%]^2$$

The confidence level is estimated at 95% with a z-value of 1.96. Confidence intervalormarginoferrorisestimatedat±5,whenproportionwithsepsisamongAKI is 25% Assuming p% =25% and q%=75%.

n=53

RESULTS

Table1:Distributionofthestudyparticipantsaccordingtotheirage(N=53)

Agegroup(years)	Number	Percentage
19-25	20	37.73
26-30	23	43.39
31-35	9	16.99
36-40	1	1.89
Total	53	100

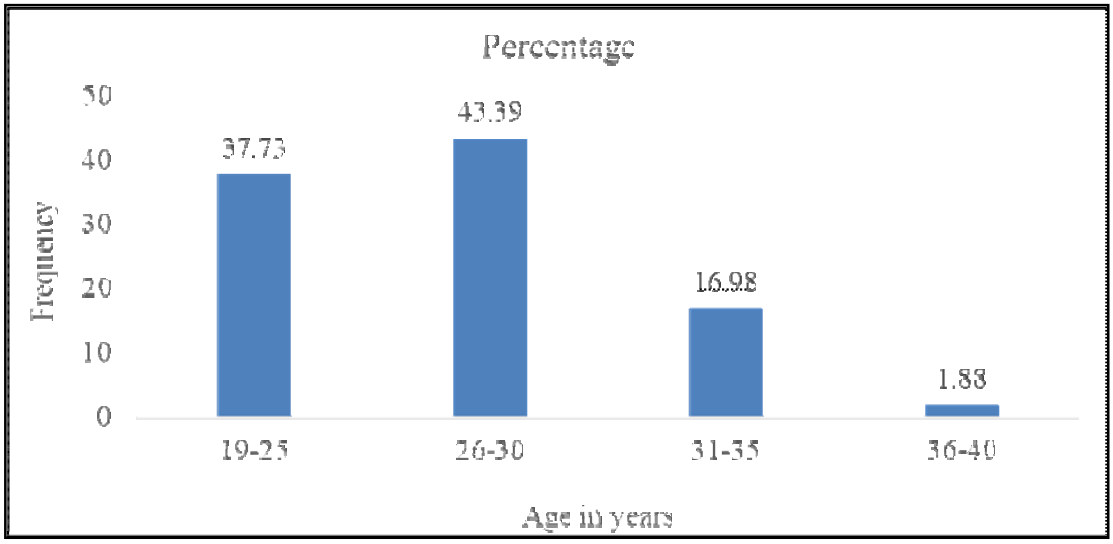

Figure1:Distributionofthestudyparticipantsaccordingtotheirage (N=53)

Majoritypatientsinourstudywasinagegroup26-30years(43.39%)and 19-25 years (37.73%), followed by 31-35 years (16.98%) and 36-40 years (1.88%).

Parity	Number	Percentage
Primi	25	47.17
G2	19	35.85
G3	9	16.98
Total	53	100

Table2:Distributionofthestudyparticipantsaccordingtotheir Parity(N=53)

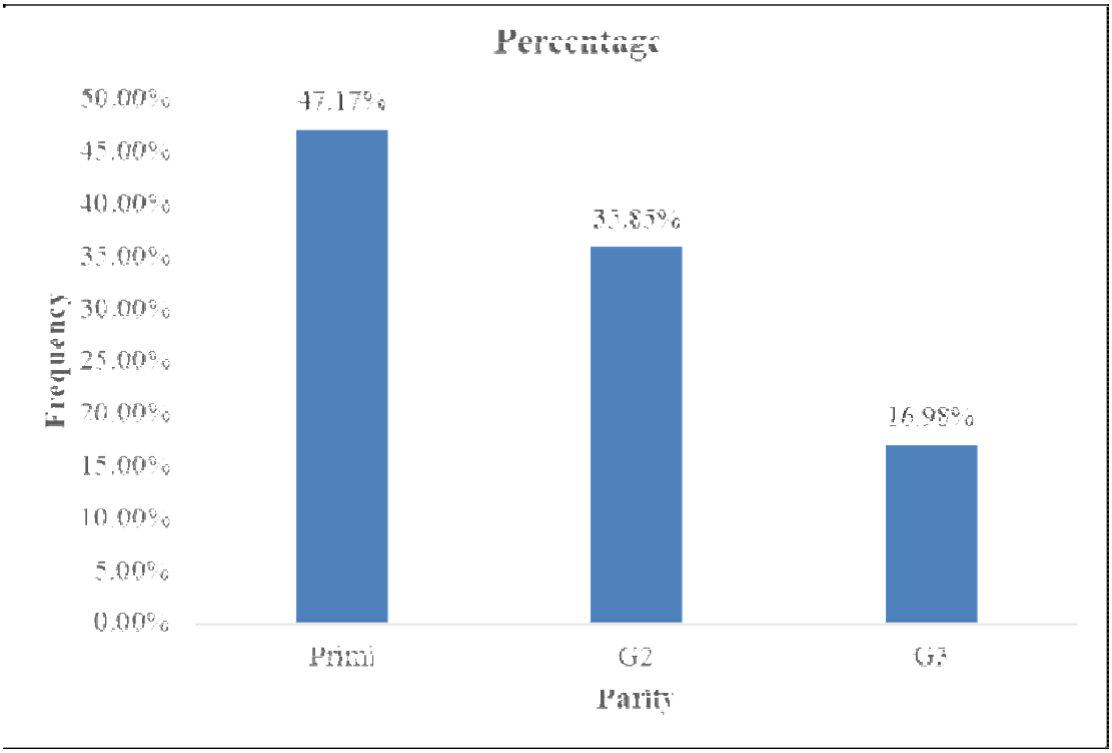

Figure2:Distributionofthe studyparticipants accordingtotheirParity(N=53)

Majority of patients in this study belonged to primi 47.17% and G 2 35.85%followed by G 3 16.78%.

Table3:Distributionofthestudyparticipantsaccordingtotheir Clinical symptoms (N=53)

Clinical symptoms	Number	Percentage
Oliguria	20	37.74
Fever	11	20.75
Abdominalpain	6	11.32
Vomiting	18	33.96
Loose stools	3	5.66
Bleeding PV	15	28.30

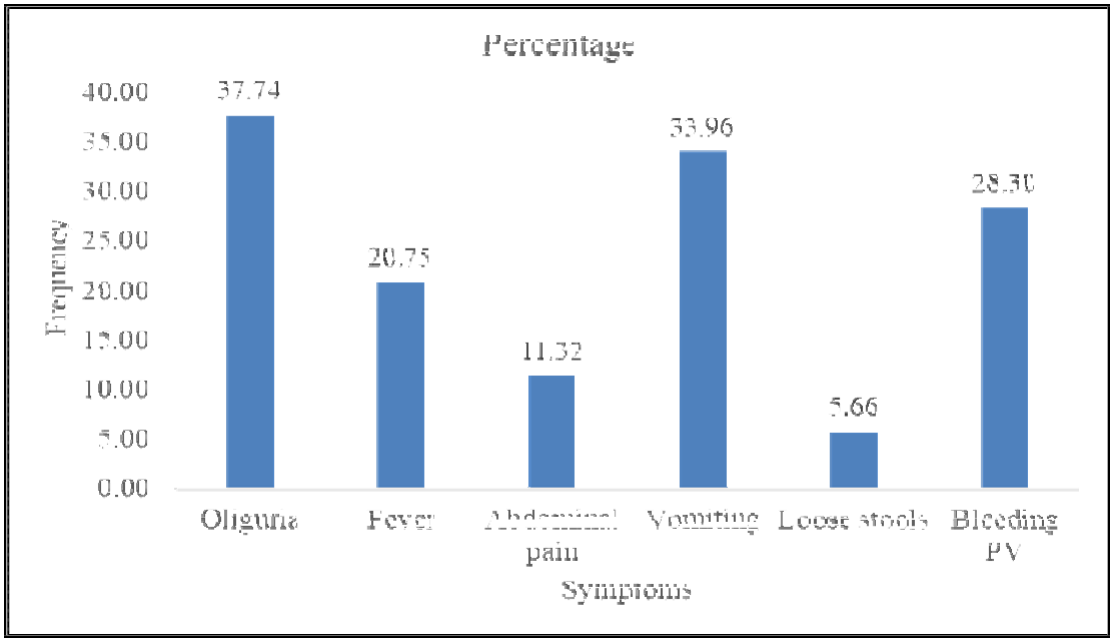

Figure3:Distributionofthestudyparticipantsaccordingtotheir Clinical symptoms (N=53)

Majority of the patients in the study had oliguria as their major symptom (37.74%), followed by vomiting (33.96%), bleeding PV (28.3%), and fever (20.75%) whereas the least common symptom being loose stools.

Table4:Distributionofthestudyparticipantsaccordingtotheir BloodPressure(N=53)

Bloodpressure	Number	Percentage
Hypotensive	11	20.75
<140/90	23	43.40

≥140/90	19	35.85
Total	**53**	**100**

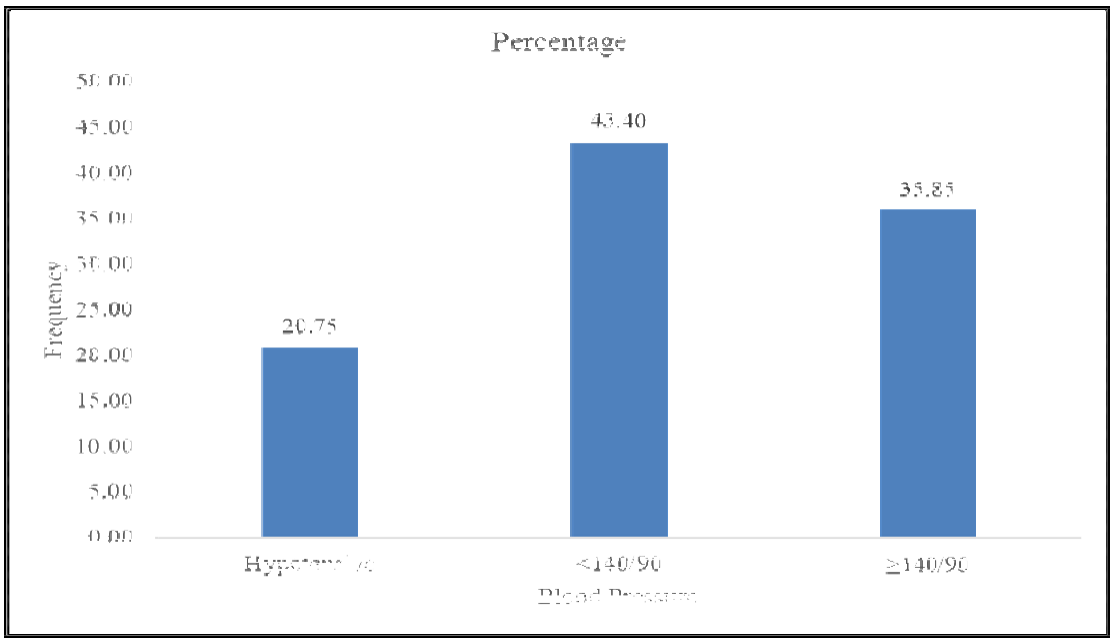

Figure4:Distributionofthestudyparticipantsaccordingtotheir Blood Pressure (N=53)

Majority of the patients in the study were normotensive (43.4%), followed by 35.85% of the participants being hypertensive.

Table5:DistributionofthestudyparticipantsaccordingtotheirHemoglobinlevels(N=53)

Hemoglobin(g/dL)	Number	Percentage
≥11	14	26.42
10-10.9	13	24.53
7-9.9	26	49.06
Total	**53**	**100**

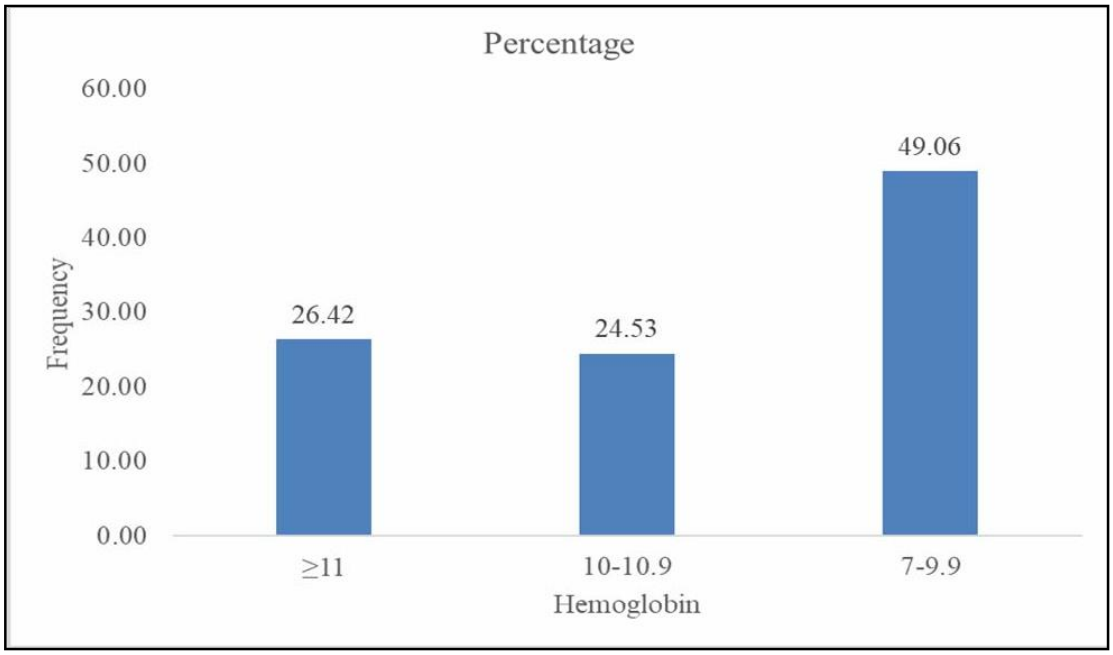

Figure5: Distribution of the study participants according to their Hemoglobinlevels (N=53)

Majority of the patients in the study were anemic with hemoglobin being inthe range of 7-9.9 (49.06%).

Table6:DistributionofthestudyparticipantsaccordingtotheirTotalcount (N=53)

Totalcount(/cumm)	Number	Percentage
3000-5000	3	5.66
5000-6000	5	9.43
6000-8000	12	22.64
8000-10,000	14	26.42
10,000-15,000	11	20.75
15,000-20,000	7	13.21
20,000-25,000	1	1.89
Total	**53**	**100**

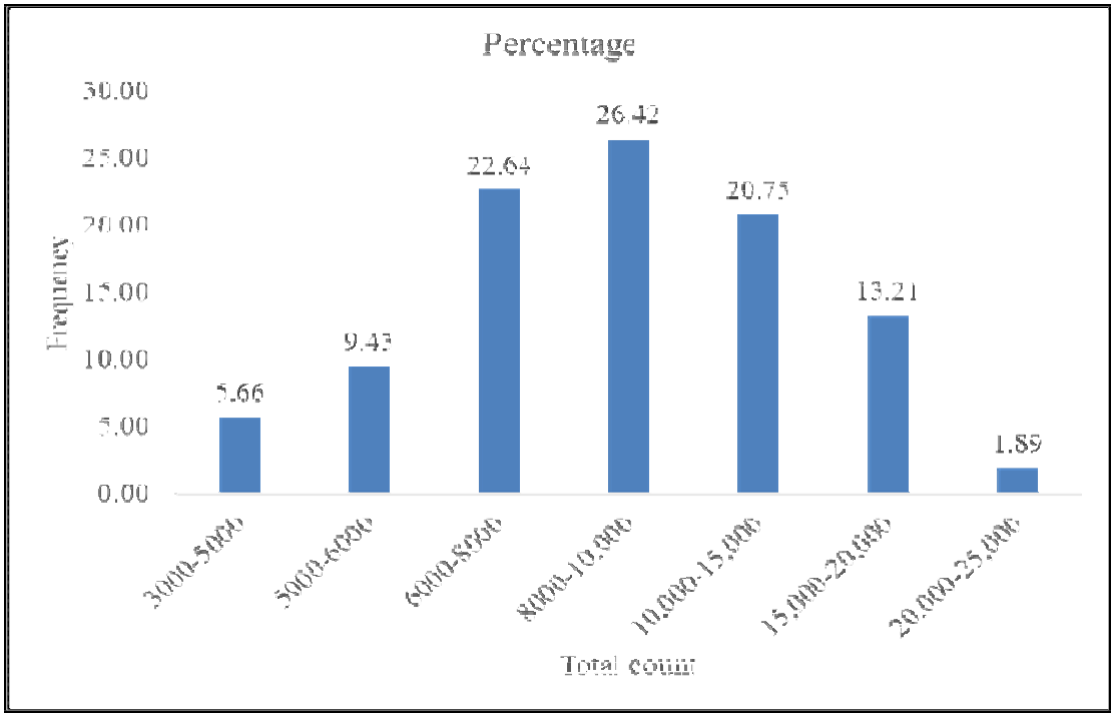

Figure 6: Distribution of the study participants according to their Total Count (N=53)

Majorityofthepatientshadbloodcountswithinnormalrange3000-10,000 (64.15%), whereas 35.85% of the participants had elevated blood counts level.

Table 7: Distribution of the study participants according to their Platelet count (N=53)

Platelets(/cumm)	Number	Percentage
50,000-1,00,000	4	7.55
1,00,000-1,50,000	20	37.74
1,50,000-2,00,000	19	35.85
2,00,000-2,50,000	6	11.32
2,50,000-3,00,000	2	3.77
3,00,000-3,50,000	2	3.77
Total	**53**	**100**

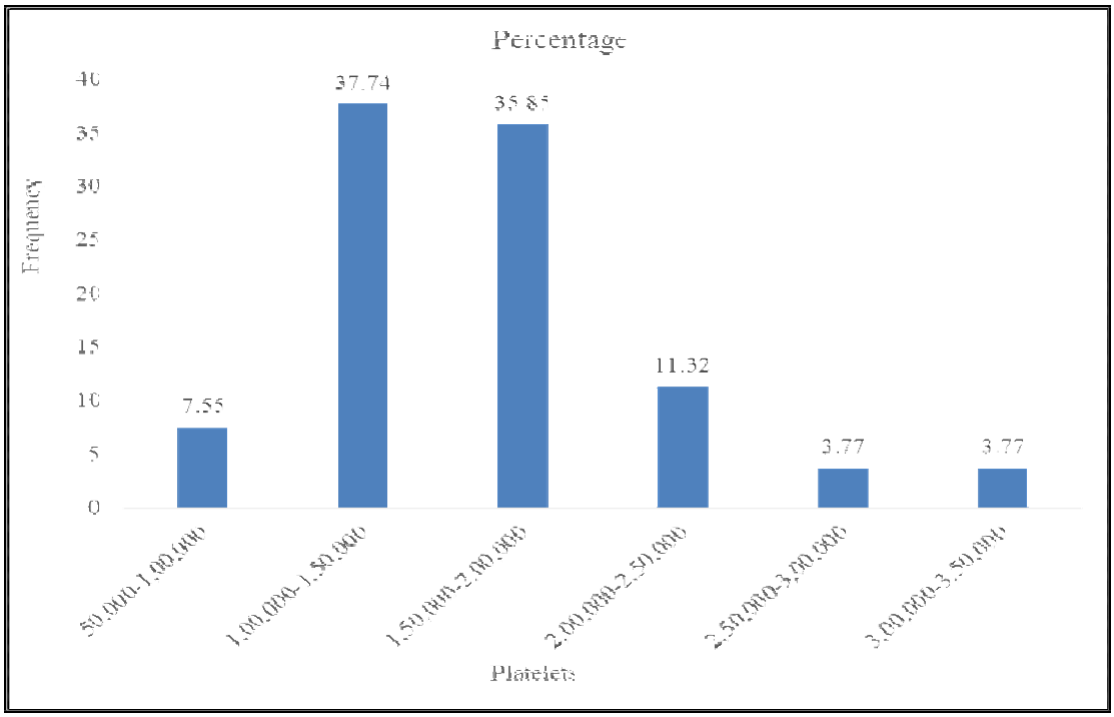

Figure7:DistributionofthestudyparticipantsaccordingtotheirPlatelet count (N=53)

The study participants showed no evidence of thrombocytopenia in the study, although majority of patients had platelet count within 1,00,000-1,50,000(37.74%), followedbyplateletscountswithintherangeof1,50,000-2,00,000(35.85%).Theleast numberofpatientshadplateletcountwithintherefencerangeof2,50,000-3,00,000& 3,00,000-3,50,000(3.77% in each group)

Table8:DistributionofthestudyparticipantsaccordingtotheirESRLevels (N=53)

ESR	Number	Percentage
5-7	18	35.6
7-9	14	31.1
9-11	10	15.6
11-13	7	4.4
13-15	2	8.9
15-17	1	2.2
17-19	1	2.2
Total	**53**	**100**

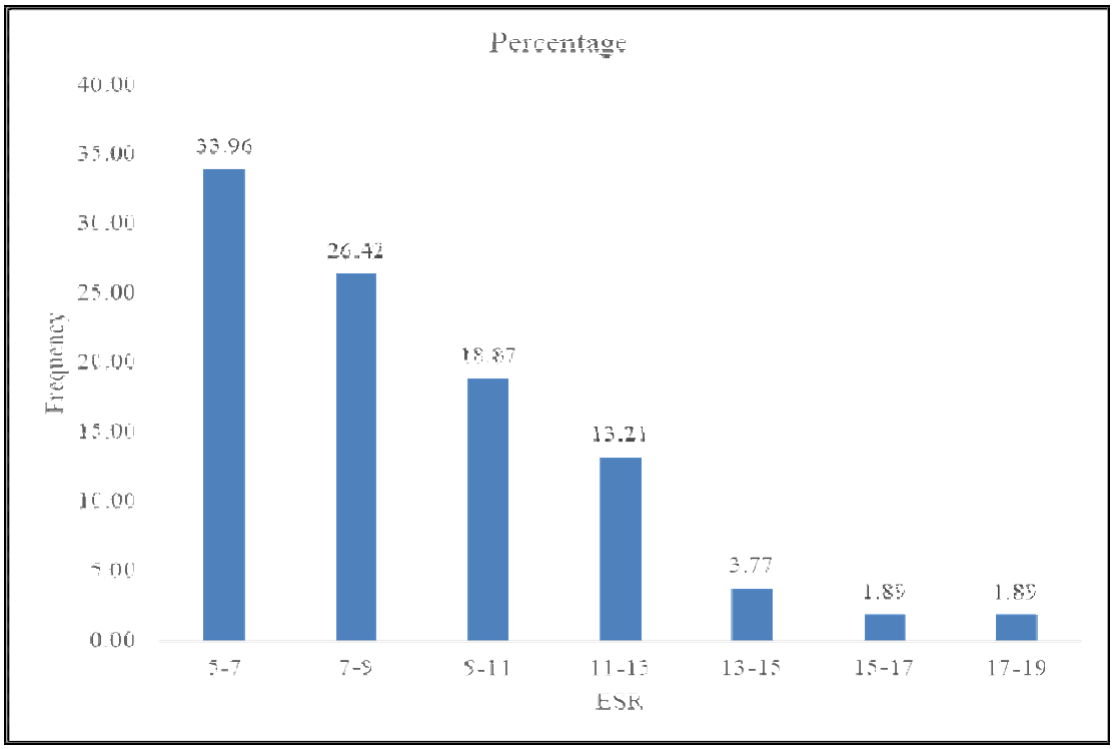

Figure 8: Distribution of the study participants according to their ESR Levels (N=53)

MajorityofthepatientsinthestudyhadnoevidenceofelevatedESR.

Table9:DistributionofthestudyparticipantsaccordingtotheirUrealevels (N=53)

BloodUrea(mg/dl)	Number	Percentage
<40	3	5.66
40-100	32	60.38
100-200	18	33.96
53	**100**	

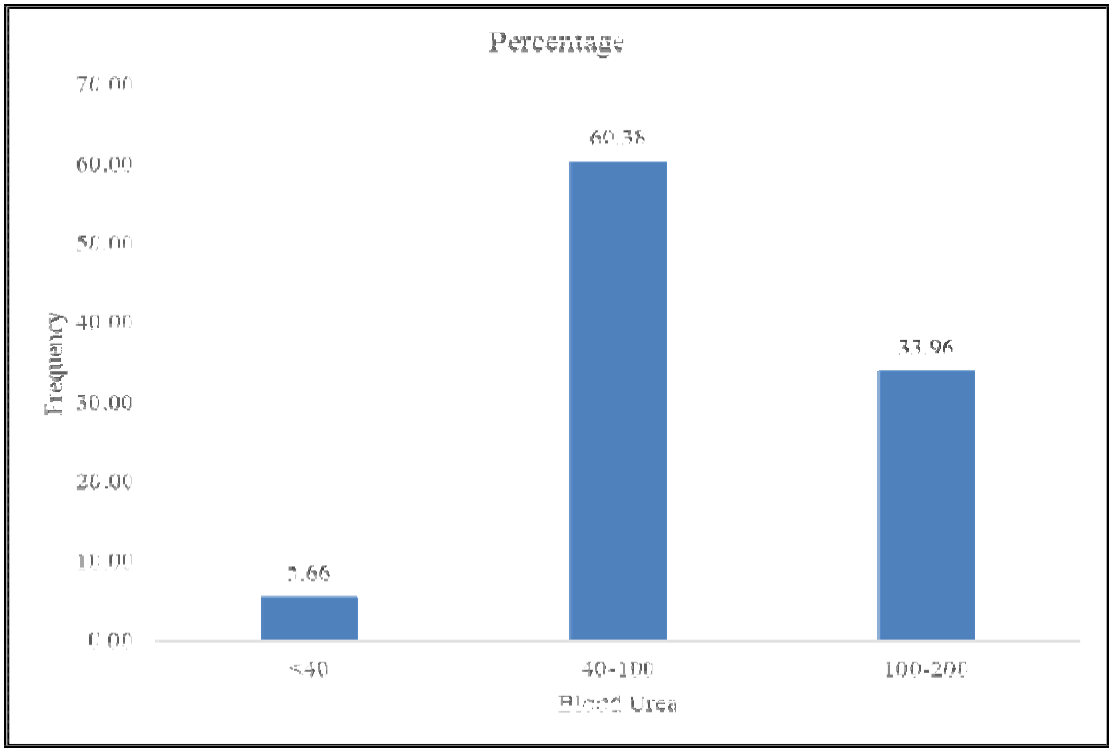

Figure 9: Distribution of the study participants according to their Urea levels(N=53)

Majorityofthepatientsinthestudyhadelevatedurealevelsbetween 40-100 (60.38%), followed by urea levels within 100-200(33.96%), whereas only 5.66% of study participants had normal urea levels

Table10:Distributionofthestudyparticipantsaccordingtotheir Blood creatinine levels (N=53)

BloodCreatinine(mg/dl)	Number	Percentage
1-2	25	47.17
2-3	12	22.64
3-4	11	20.75
4-5	5	9.44
Total	53	100

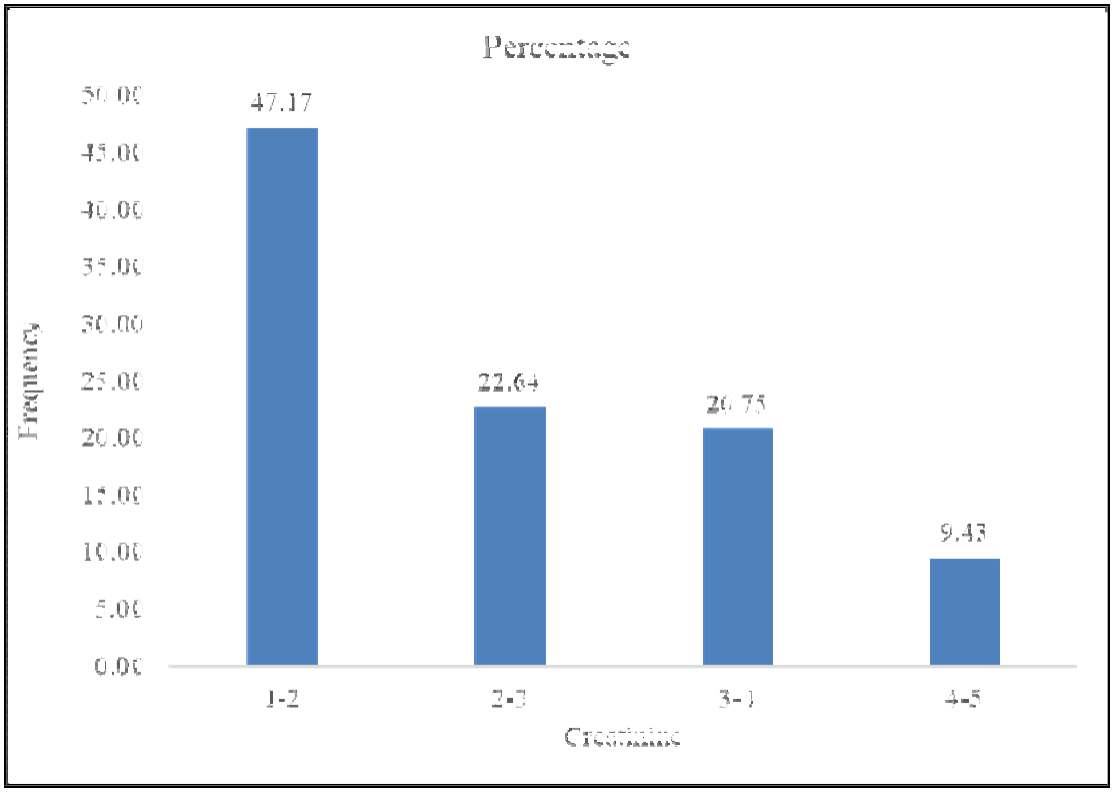

Figure10:Distributionofthestudyparticipantsaccordingtotheir Blood creatinine levels (N=53)

Majorityofthepatientsinthestudyhadelevatedserumcreatininelevelsof 1-2 (47.17%), followed by 2-3 (22.64%), 3-4 (20.75%). Only 9.44% of study participants had creatinine values within the range of 4-5.

Table11:DistributionofthestudyparticipantsaccordingtotheirBilirubin levels (N=53)

Total Bilirubin mg/dl	Number	Percentage
<1	20	37.74
1-1.2	20	37.74
1.2-3	10	18.87
3-5	3	5.66
Total	**53**	**100**

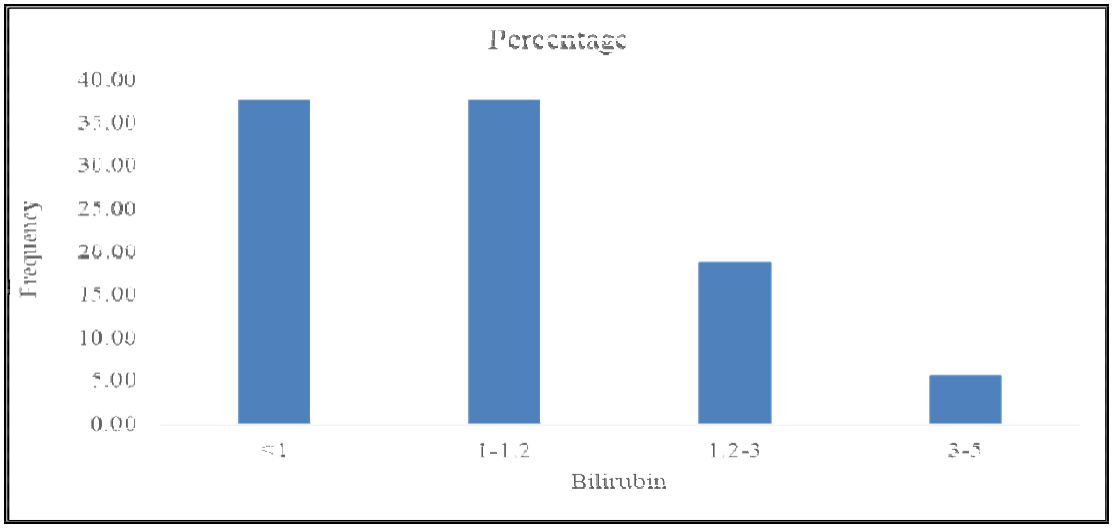

Figure11:Distributionofthestudyparticipantsaccordingtotheir Bilirubin levels (N=53)

Majority of the patients in the study had bilirubin levels <1 and 1-1.2 (37.74% each), followed by 1.2-3 (18.87%), and within 3-5 (5.66%).

Table12:DistributionofstudyparticipantsaccordingtotheirSGOTLevels (N=53)

SGOT(U/L)	Number	Percentage
5-40	37	69.82
40-100	13	24.52
100-300	3	5.66
Total	53	100

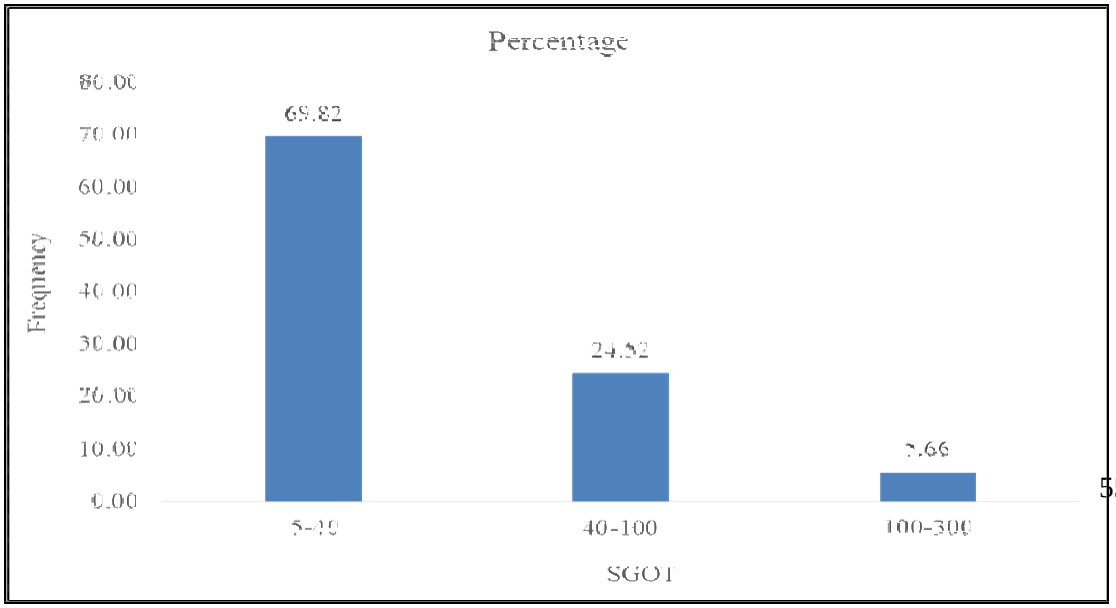

Figure12:DistributionofstudyparticipantsaccordingtotheirSGOTLevels (N=53)

Majority of patients had no elevated liver enzymes levels (69.82%), whereas 24.52% and 5.66% of patients had elevated SGOT levels elevated between 40-100and 100-300 respectively.

Table 13: Distribution of the study participants according to their SGPT Levels (N=53)

SGPT(U/L)	Number	Percentage
5-56	49	92.45
56-100	1	1.89
100-300	3	5.66
Total	53	100

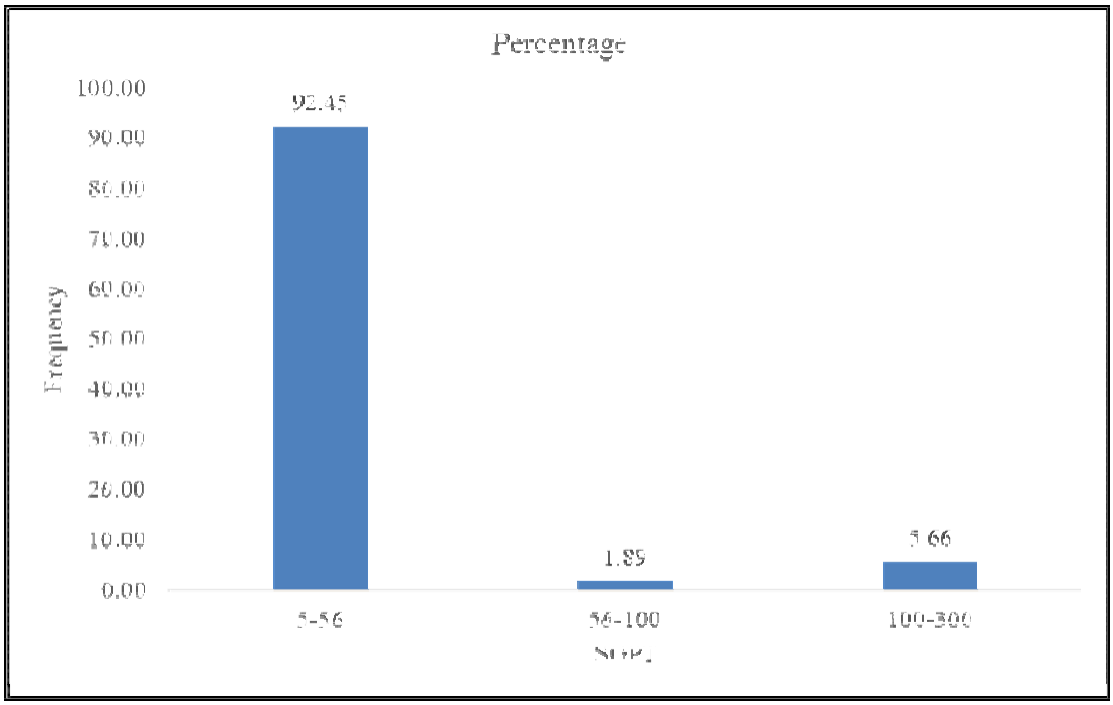

Figure 13: Distribution of the study participants according to their SGPTLevels (N=53)

MajorityofthepatientsinthestudyhadserumSGPTlevelswithin5-56(92.45%),whereas1%and3%ofstudyparticipantshadelevatedSGOTlevelswithin 56-100 and within 100-300 respectively

Table14:Distributionofthestudyparticipantsaccordingtotheir ALP Levels (N=53)

ALP(U/L)	Number	Percentage
20-140	52	98.11
≥140	1	1.88
Total	**53**	**100**

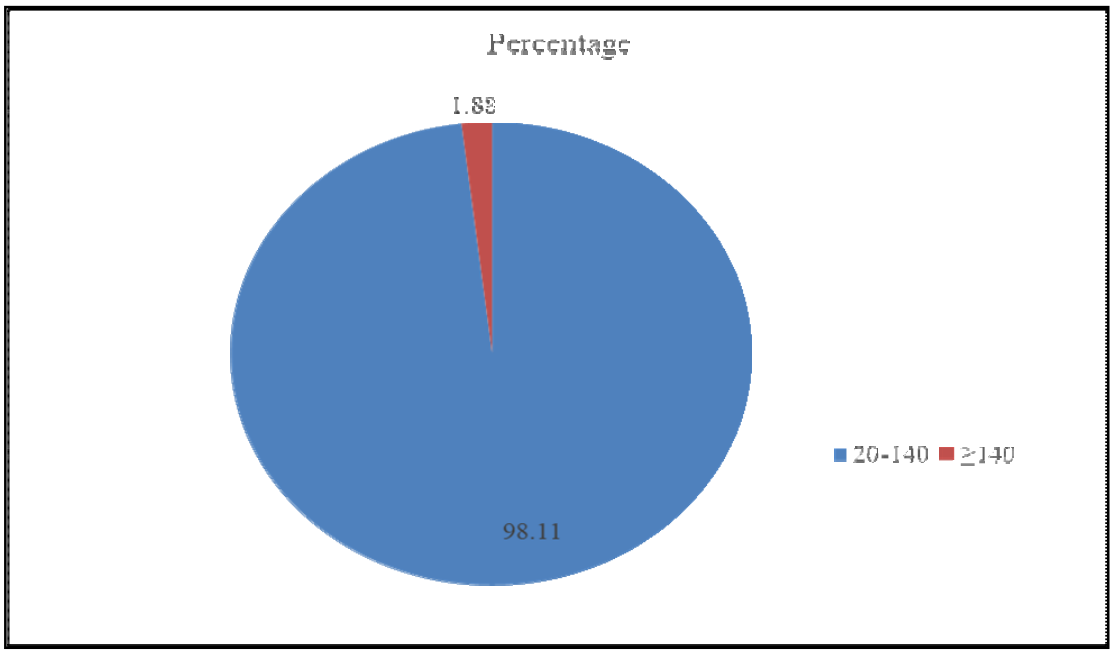

Figure14:Distributionofthestudyparticipantsaccordingtotheir ALP levels (N=53)

MajorityoftheparticipantsinthestudyhadnoelevatedALPlevelswhileonly 1.88% (1 study participant) had ALP levels >140.

Table15:Etiologyofacutekidneyinjury

Etiology frequency	Maternal
Hypertensive disorders	26
Post partum heamorrhage	10
Abruptio placenta	6
HELLPsyndrome	4
Sepsis	5

Hyperemesisgravidarum	2

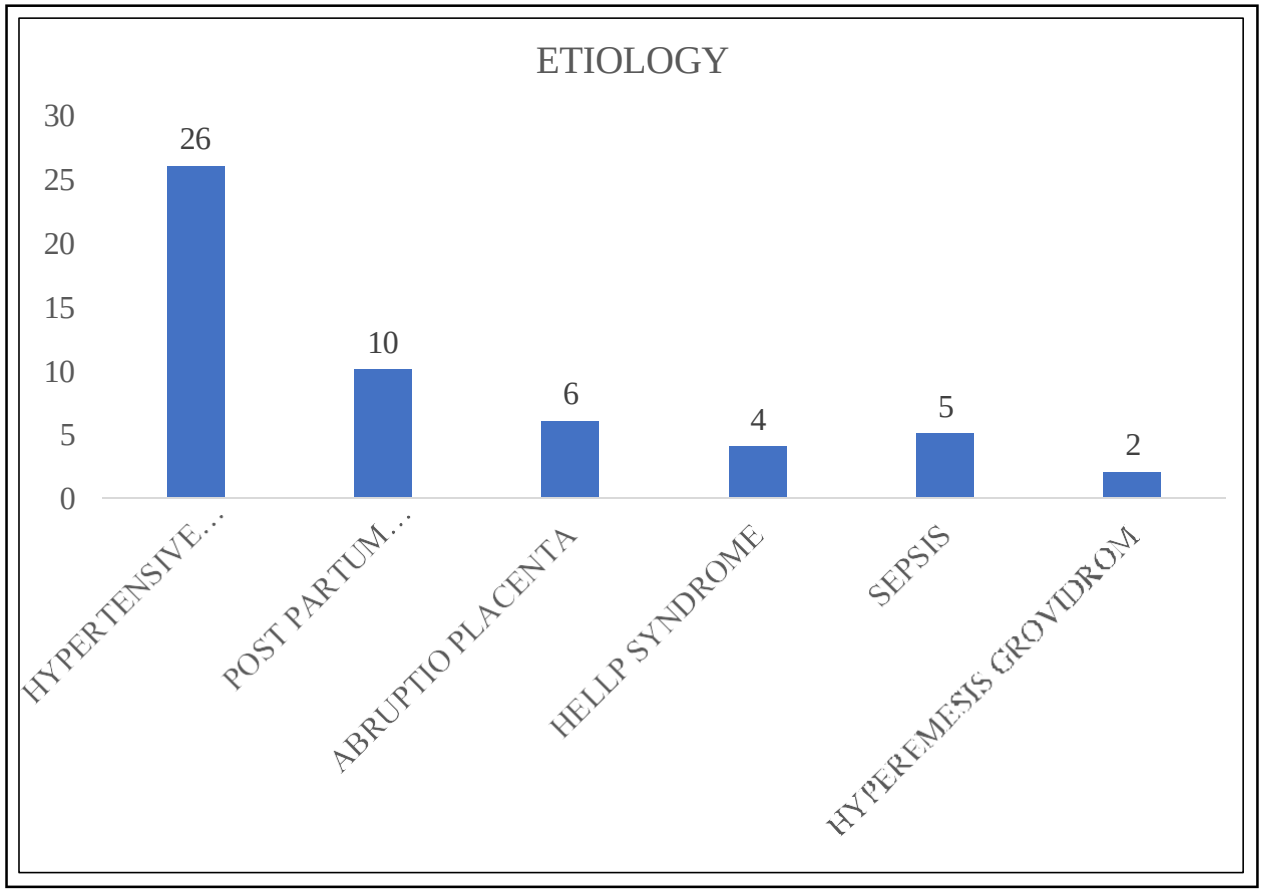

Figure15:Etiologyofacutekidney injury

Majority of patients had AKI etiology being hypertensive disorders of pregnancy (26 participants), followedby 10 participants with etiology of post partum haemorrhage,abruptioplacentabeingtheetiologyin6participants,sepsisin 5 participants,HELLPsyndrome and hyperemesis gravidarumin 4 and 2 participants respectively.

Table16:Maternaloutcome

Outcome	Number	Treatment	Total
Recovered	48	Supportivemeasures	41
		Dialysis	7
Death	5	Dialysis done	3
		Notdoneduetohemodynamic instability	2

DISCUSSION

Table17:Comparisonofagedistributionwithotherstudies

	Currentstudy	**MaheshPuri et al[7]**	**Gopalkrishna et al[8]**	**Godaraet al[9]**
Total	53	165	130	57
Age	26	25	28	22

The table shows a comparison between the current study with others studies formeanageofthetotalnumberofpatients.Thecurrentstudyhasatotalof 53 patients wish the mean age of 26 while the study by Mahesh Puri et al had a totalof165patientswithameanageof25.ThestudybyGopalkrishnaetalhad 130 patients with a mean age of 28 and Godara et al had 57 patients with a mean age of 22.

Table18:Comparisonofparitywithotherstudies

	Currentstudy	**MaheshPuri et al[7]**	**Krishnaetal[10]**	**Godaraet al[9]**
Total	53	165	98	57
parity	Primi	Primi	Gravida 2	Gravida 2

Table 19 shows the comparison of parity of our study with other studies. Our study with 53 patients had a mean parity of primi pregnancies. Mahesh et al had similarresults.StudiesbyKrishnaetalandGodaraetalshowedthemeanparitytobe Gravida 2 pregnancies.

Table19:ComparisonofcauseofAKIwithotherstudies

	Currentstudy	**Gopalkrishna et al[8]**	**PrakashJet al[11]**	**Godaraet al[9]**
Total	53	130	132	57

Most common	Pre eclampsia	Sepsis	Pre eclampsia	Sepsis

Table 20 compares cause of AKI in our study with other studies. The most common cause of AKI in our study and the study by Prakash J et al was pre eclampsia. The most common cause of AKI in the studies by Gopalkrishna et al and Godara et al was Sepsis.

CONCLUSION

Pregnancy related acute kidney injury (PRAKI) is a majority clinicalchallenge because it poses a risk to 2 lives mother and fetus. It is largely due to preventableobstetricalcomplications,butcanbecausedbycertainpregnancyspecific diseases. Pregnancy hypertensive complications, like preeclampsia are the leading cause of PRAKI. Puerperal sepsis and obstetrical hemorrhage still account forAKI in the postpartum period in addition to PE/HELLP syndrome. The implementation of specific intervention for the prevention and management of sepsis and hypertensive complications in pregnant women may decrease the burden of PRAKI.

Fortunately, with ongoing improvements in obstetrical care, multidisciplinary approaches, and new insights into the diagnosis and management of associated conditions such as preeclampsia, maternal and perinatal mortality in this setting are largely avoidable.

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