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"Clinical Profile of Patientswith Acute Kidney Injuryrequiring Hemodialysis".

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ABSTRACT: Background: Acute kidney injury (AKI) refers to a syndrome encompassing kidney damage from mild injury to total loss of function that seriously disturbs the homeostasis of fluid and electrolyte balances. Acute kidney injury (AKI) is a frequent complication. This study aims to describe the clinical, biological aspects of AKI in tertiary care center of Maharashtra, India. Method: A prospective observational study was performed in Maharashtra, India. All patients suffering Acute Kidney Injury who don't have any prior kidney disease. The diagnosis of AKI was made using the KDIGO criteria. Kidney ultrasound exam wasperformed in all patients to assess internal bleeding and morphological and structural abnormalities of the kidneys.Results:Mean age was 43.18years with majority in 3rd and 5th decade.Majority of the patients were male.Majority of the patients i.e. 88 of them had Oliguria as a main complaint.Majority of the patients i.e. 50 of them had fever as common complaint. Vasculotoxic snake bite induced AKI was most common cause.Conclusion: A patients with Acute Kidney Injury requiring hemodialysis early initiation of Hemodialysis is associated with improved survival. Increasing AKI severity was associated with increased mortality.

I. INTRODUCTION

Acute kidney injury (AKI) refers to a syndrome encompassing kidney damage from mild injury to total loss of function that seriously disturbs the homeostasis of fluid and electrolyte balance[1].A uniform definition for acute kidney injury has existed only since 2004,the Acute Dialysis Quality Initiative (ADQI) proposed the Risk, Injury, Failure, Loss, End-stage kidney disease (RIFLE) criteria for AKI[2].Since then two modifications of the RIFLE: Acute Kidney Injury Network (AKIN) (2007)[3], and Kidney Disease: Improving Global Outcomes (KDIGO) (2012)have emerged[4]. All of the three modern definitions are based on changes in serum or plasma creatinine (Cr) and urine output (UO).

Clinical symptoms may be scarce in the early stages of AKI. As the kidney injury progresses and affects the glomerular filtration rate (GFR) Cr starts to rise. Oliguria or anuria may develop early, but sometimes the UO remains intact for quite long. Later in the course of AKI the severely diminished GFR manifests as electrolyte and acid-base disturbances, most often as elevated potassium and acidosis. Several different pathways have been proposed and studied; none of which seems to explain the big picture alone.The arising consensus suggests that AKI is a syndrome with several different predisposing factors and mechanisms of pathophysiology. A growing amount of data supports the idea that risk for AKI increases with a growing "burden of illness" whether chronic or acute.

The traditional division of kidney failure to pre- and post-renal causes has been widely abandoned as the complex nature of the kidney injury syndrome has unfolded. Extra renal causes, without actual kidney damage, such as depletion of fluids or urinary track obstruction naturally still exist but are rare causes for AKI in the intensive care environment. Also these causes, when identified, are quite easy to treat and usually without long-term damage to the kidney or other organs. In the ICU, AKI is usually multifactorial with both chronic conditions and acute events contributing to the development of kidney injury[5]. Sepsis is the most common single underlying cause for AKI.

In the diagnosing and staging of AKI, serum creatinine and urine output act as surrogates for glomerular filtration rate, however prominent weaknesses in both as kidney injury markers exist. A vigorous search for new kidney injury biomarkers has been going on for several years. A hope of easily measurable markers that would be more sensitive and specific to actual injury in the kidneys, would react earlier in the course of AKI, and would be less prone to bias in different physiological situations remains[6].

AKI has significant consequences. It is associated with morbidity and permanent loss of

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kidney function.All severity stages of AKI are associated with significantly higher short and long-term mortality.AKI increases hospital expenses up to two-fold and achieving quality adjusted life years in the treatment of AKI patients is expensive.

The aim of this study was to study the clinical profile and the different etiologies of patients with AKI requiring hemodialysis and the outcome of patients with AKI requiring hemodialysis.

II. MATERIAL & METHODS

Type of study - Prospective Case Series Study
Period of study -Duration from Sep 2018 to Aug 2020.
Sample size -The present study comprises of 110 patients whodevelop acute kidney injury requiring hemodialysis.

Patient data collection and evaluation:

Due informed consent of patients was taken. Meticulous history, clinical examination, laboratory and radiological investigations were done in all patients. Data was collected from all patients, irrespective of their gender/background /socio economic status on admission and later, on daily basis till the patients were discharged from hospital or had expired whichever was case. The patients were evaluated and followed up according to protocol.

INCLUSION CRITERIA

1. Patients >14years of age.
2. Patient with acute kidney injury not responding to Conservative management.
3. Patient with manifestation of uraemia, fluid over load, Hyperkalaemia, etc.

Exclusion criteria:

1. Patients of age <14years.
2. Patients with Pre -exiting renal disease.

III. RESULTS

Table no 1: Gender wise distribution of Patient

Gender	Total	Percentage (%)
Male	69	62.72
Female	41	37.27

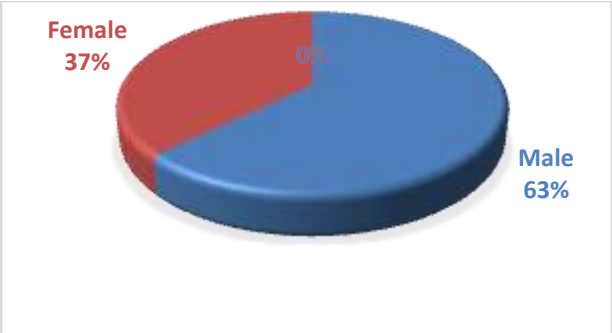

Chart no 1: Gender wise Distribution of patients.

Table no 2: Age wise distribution of Patients

Age group (In Years)	Number of patients	Percentage (%)
14-20	10	9.09
21-30	29	26.36
31-40	16	14.54
41-50	17	15.45
51-60	9	8.18
61-70	22	20.00
>71	7	6.36
Total	110	100

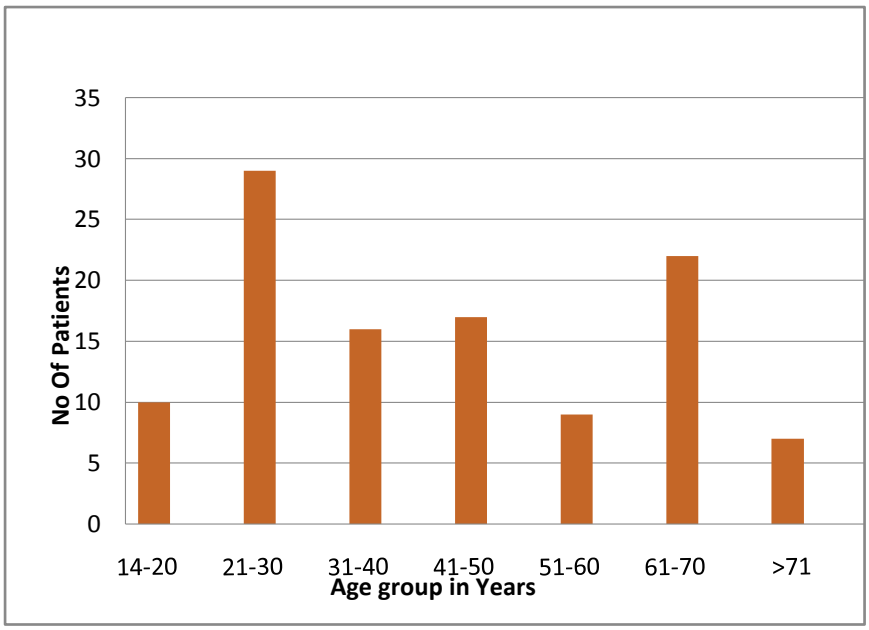

Chart no 2: Age wise Distribution of patients.

The above table shows distribution of study population based on different age groups. Majority of AKI patients i.e.26.36%were between 21-30years followed by 20% in 61-70 years and followed by 15.45% from 41-50 years and followed by 14.54% from 31-40 and followed by 09.09% from 14-20 and 8.18% from 51-60 years, 6.36% from >71 years.

Table No.3: Distribution of patients with respect to gender.

14-20	6	4	10
21-30	15	14	29
31-40	10	6	16
41-50	11	6	17
51-60	5	4	9
61-70	17	5	22
>71	5	2	7

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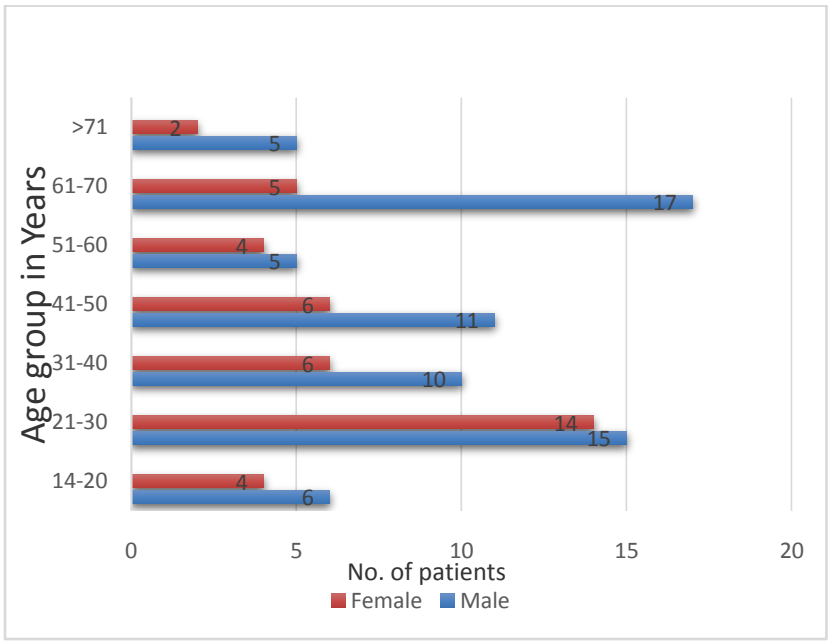

Chart No.3: Distribution of patients with respect to gender.

The table shows distribution of 110 patients with respect to age and gender. Age was categorized with an interval of 10. Out of 110, 69 were males and 41 females. Most of the males belonged to the age group of 61 to70 years. Most of the females belonged to the age group of 21 to 30 years.

Table No.4: Distribution of patients with respect to etiology and Age

Etiology	**14-20 years**	**21-30 years**	**31-40 years**	**41-50 years**	**51-60 years**	**61-70 years**	**>71 years**	**Total**
Vasculotoxic Snake Bite	3	19	13	10	3	9	2	59
Sepsis	2	2	1	6	5	9	5	30
Obstetric causes	5	6	1	0	0	0	0	12
Hepatic Cause	0	0	2	1	0	2	0	5
Obstructive Cause	0	0	0	0	1	2	0	3
Paraquat Poisoning	1	0	0	0	0	0	0	1
Total	11	27	17	17	9	22	7	110

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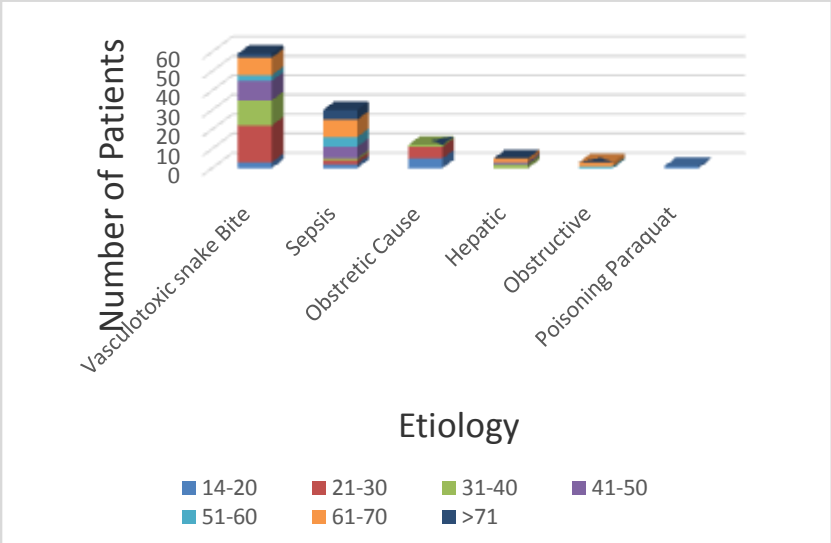

Chart No. 4: Distribution of patients with respect to etiology and age.

The Table depicts various causes of AKI distributed across the age groups. Most common cause of AKI in the study is vasculotoxic snake bite, followed by Sepsis and Obstetrical. Age-group wise, the 3 most common causes in the age group of 21 to 30 yearsare:vasculotoxic snake bite and obstetric causes. And sepsis Vasculotoxic snake bite is the most common causes in AKI patients aged between 21 and 50 years.

Table no 5: Different Causes of Sepsis

Cause of sepsis	Number of Patients
Urosepsis	21
Cellulitis	6
Pneumonia	3
Total	30

Among the Sepsis Urosepsis is the most common cause fallowed by Cellulitis followed by Pneumonia. Overall the Sepsis is most commonly seen in age group 40-70. All 6 Patients of cellulitis underwent for debridement.

Table no 6: Different Obstetric causes of AKI

Cause of Obstetrics	Number of Patients
Puerperal Sepsis	6
HELLP Syndrome	2
Pre- Eclampsia	2
Abruptio Placenta	1
Postpartum Hemorrhage	1
Total	12

Among the Obstetric causes of AKI Puerperal Sepsis is the most common cause fallowed by HELLP Syndrome followed by Pre-Eclampsia Abruptio Placenta and Postpartum Hemorrhage. Overall the Puerperal Sepsis is most commonly seen in age group 20-30.

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Table No. 6: Distribution of patients with respect to clinical features

	Clinical symptoms	No. of patients	Percentage (%)
	Fever	50	45.45
Urinary symptoms	Oliguria	88	80.00
	Anuria	16	14.54
	Dysuria	10	9.09
	Pyuria	5	4.54
Respiratory symptoms	Dyspnea	24	21.81
	Cough	3	2.72
Gastrointestinal symptoms	Nausea	14	12.72
	Vomiting	10	9.09
Central Nervous System Symptoms	Altered Sensorium	11	10.00
	Convulsion	1	0.90

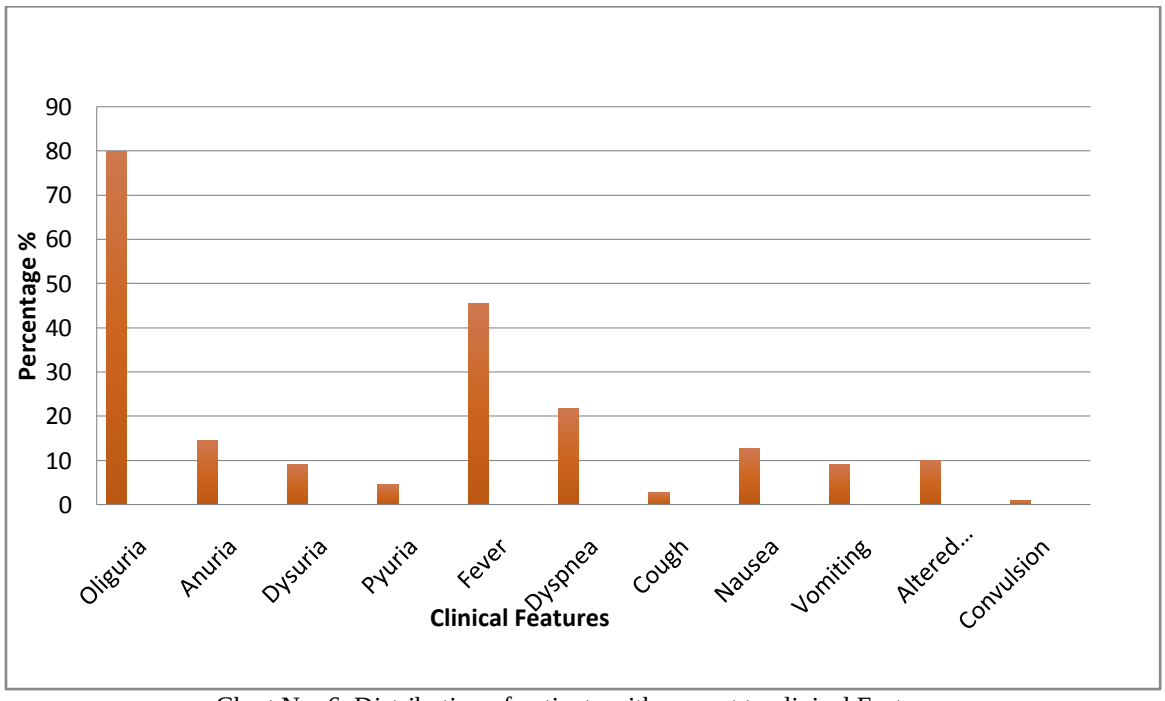

Chart No. 6: Distribution of patients with respect to clinical Features

Most common clinical symptom with which the patients presented were Oliguria (80%) fallowed by Fever (45.45%).The major system involved is Renal system, complaining of Oliguria(80%), Anuria (14.54%), Dysuria (9.09%), Pyuria (4.54%). Another Major System involved is Respiratory System Complaining of dyspnea (21.81%), 2.72% complaining of cough. Gastrointestinal symptoms are Seen Nausea (12.72%) and vomiting (9.09%). And among central nervous system symptoms, 10% had alteredsensorium and 0.90% had Convulsion.

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Table No. 7: Distribution of patient with respect to General examination

General examination	Number of patients	Percentage (%)
Pallor	78	70.90
Icterus	8	7.27
Tachycardia	14	12.72
Cellulitis	6	5.45
Cellulitis due to Snake Bite	8	7.27

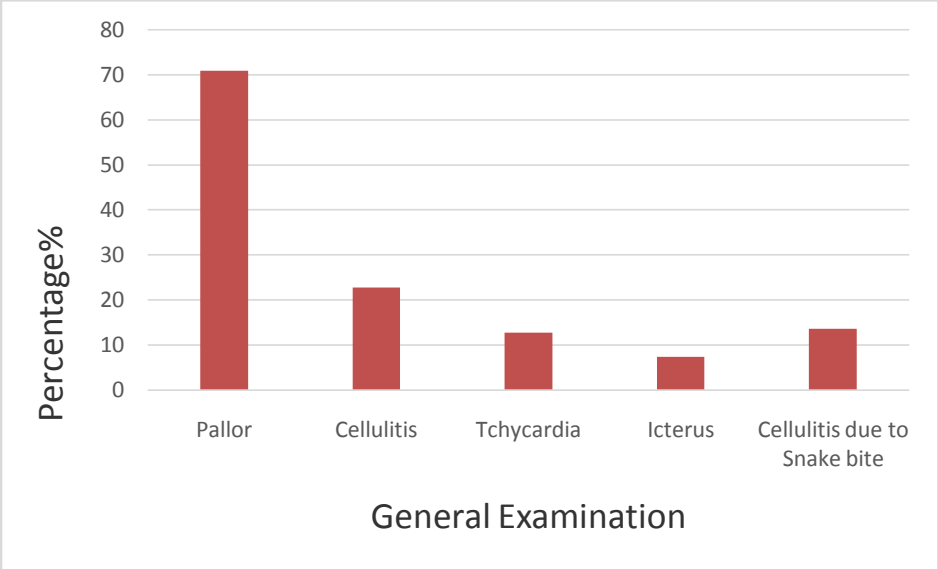

Chart No. 11: Distribution of patients with respect to General examination

The table shows distribution of patients with respect to General Physical Examination. Pallor was present in 70.90%(78) and7.27%(8) Patients have cellulitis secondary to snake bite, Cellulitis were present in 5.45%(6). And Tachycardia in 12.72%(14) patient, icterus was present in 7.27%(8) patients.

Table No. 8-A: Distribution of patients with respect to CBC

Parameters		No of Patients	Percentage%
WBC Count	>11000	49	44.54
	<11000	61	55.45
Hemoglobin Level	>10	35	31.81
	<10	75	68.18

In 44.54% patients WBC's count were raised more than 1100. And in 55.45 % WBC's count were Normal. In 31.81% patients Hemoglobin level were more than 10. And in 68.18 % Hemoglobin level were less than 10.

Table No.8-B: Distribution of patients with respect to Renal function test.

Lab Parameter		No of Patients	Percentage%
Creatinine Level	2-5	44	40.00
	6-10	56	50.90
	11-15	8	7.27
	>16	2	1.81
Blood Urea Level	40-100	36	32.72
	101-150	40	36.36
	151-200	17	15.45
	201-250	8	7.27
	251-300	7	6.36
	>300	2	1.81

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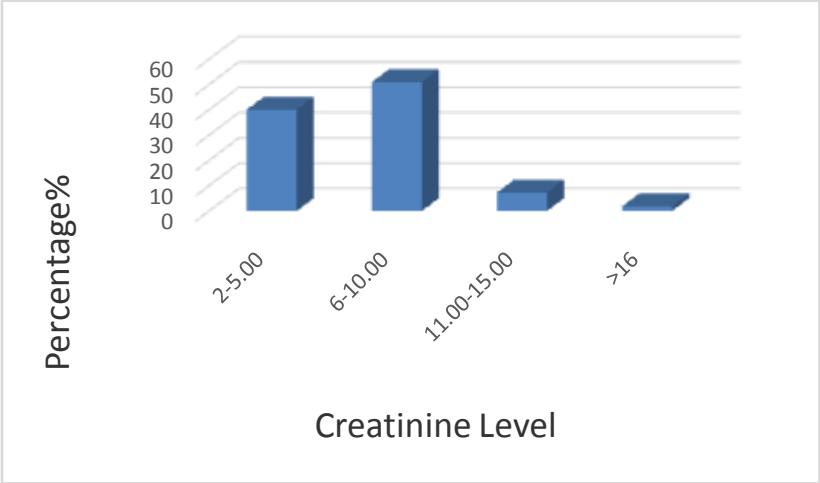

Chart No.14: Distribution of patients with respect to Creatinine Level.

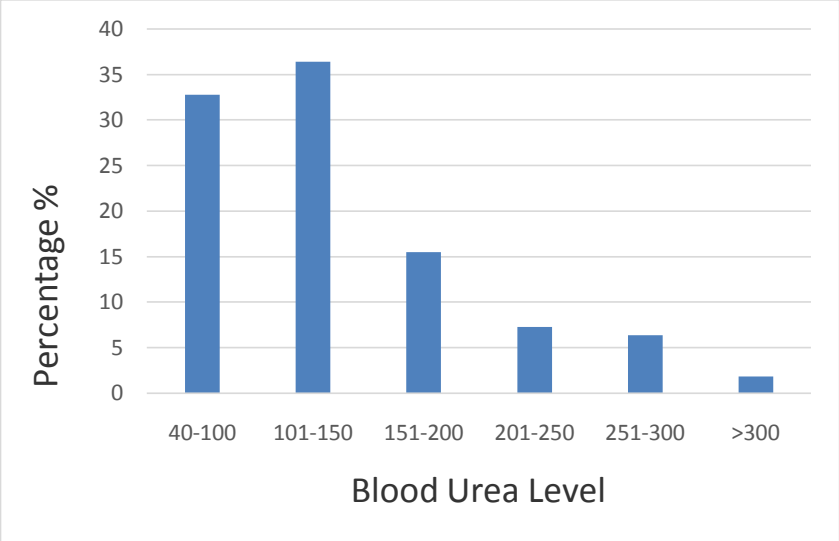

Chart No.15: Distribution of patients with respect to Blood Urea Level.

Most of the patient 50.90% were between range 6-10 of creatinine, followed 40% from range 2-5, followed by 7.27% from range 11-15, followed by 1.81% from >16.

Most of the patient 36.36% were between range 101-150 of Blood Urea Level, followed 32.72% from range 40-100, followed by 15.45% from range 151-200, followed by 7.27% from range201-250, followed by 6.36% from range 251-300, followed by 1.81% >300.

Table No.8-C: Distribution of patients with respect to Serum Electrolytes Level

Serum Electrolyte		**No of Patients**	**Percentage%**
Serum Sodium Level	Hypernatremia	5	4.54
	Normal	52	47.27
	Hyponatremia	53	48.18
Serum Potassium Level	Hyperkalemia	7	6.36
	Normal	83	75.45
	Hypokalemia	20	19.09

Most of the patients are have electrolyte abnormality. Hyponatremia is seen in 48.18% patients. Hypernatremia is seen in 4.54% patients. And 47.27% patients have normal serum sodium level.

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Hypokalemia is seen in 18.18.18% patients. Hyperkalemia is seen in 6.36% patients. And 75.45% patient had normal serum potassium level.

Table No.8-D: Distribution of patients with respect to Liver Function Tests

Lab Parameters		No of Patients	Percentage%
Serum Bilirubin Level	2-4.5	6	5.45
	4.6-10	3	2.72
	10.1- 15.5	2	1.81
Serum Aspartate Aminotransferase Level (SGOT	40-100	14	12.72
	101-200	3	2.72
	201-300	1	0.90
	301-400	2	1.81
	>400	1	0.90
Serum Glutamic Pyruvic Transaminase Level (SGPT)	40-100	18	16.36
	101-200	4	3.63
	201-300	1	0.90
	301-400	2	1.81

Hyperbilirubinemia is seen in 10% patients. 5.45% Patients have bilirubin between 2-4.5 which is more common group, followed by 2.72% from 4.6-10%, followed by 1.81% from 10.1-15.

Serum Aspartate Aminotransferase Level (SGOT) is deranged in 19.09% patients. Most common range is 40-100, 12.72%.

Serum Glutamic Pyruvic Transaminase Level (SGPT) is deranged in 22.72% patients. Most common range is 40-100, 16.36%.

Table No.9: Distribution of patients with respect to outcome

Outcome	No. of patients	Percentage (%)
Survival	89	80.90
Death	21	19.09

In this study patients with Acute Kidney Injury requiring hemodialysis, out of 110, 89 is survived and 21 expired. Percentage wise 80.90% survived and 19.09 expired

Table No.10: Distribution of etiology and outcome of AKI patients requiring hemodialysis with respect to mortality.

Etiology	No. of Patients	Outcome		Totalmortality rate%
		Survived	Expired	
Vasculotoxic snake bite	59	51	8	13.55
Sepsis	30	22	8	26.66
Obstetric	12	10	2	16.66
Hepatic	5	2	3	60.00
Obstructive	3	3	0	00.00
Paraquat Poisoning	1	1	0	00.00

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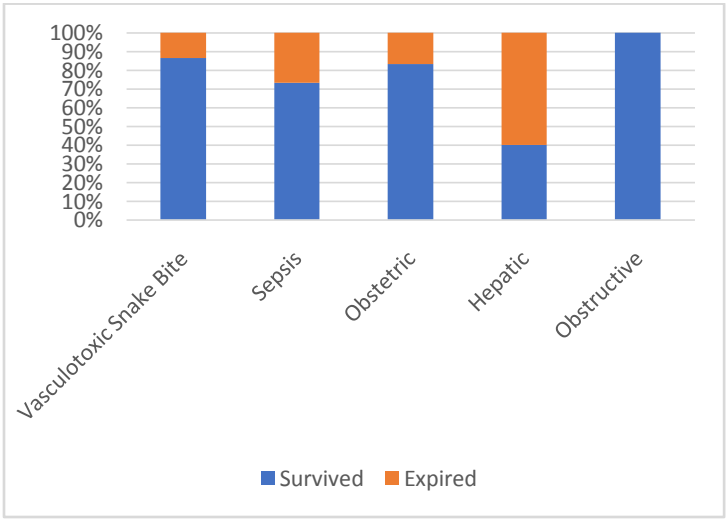

Chart No.11: Distribution of etiology and outcome of AKI patients requiring hemodialysis with respect to mortality.

IV. DISCUSSION:

The incidence rate of acute kidney injury (AKI) around the world is not well known. Recent studies in the United States and Spain have shown incidences varying between an average of 23.8 cases per 1000 discharges with an 11% yearly increase between 1992 and 2001, to an increase from 61 to 288 per 100,000 population between 1988 and 2002. More recently reported a high incidence of 1811 cases of AKI per million population during 2003[7]. The relatively wide disparity in reported incidence rates and the increasing frequency of the condition raise concerns as to the real magnitude of the problem. In addition to a real increase in worldwide incidence, large differences in the definition of AKI and case mix likely underlie such differences. It is recognized that the epidemiology of AKI in developing countries differs from that of the developed world in many important ways. Whereas in developed regions elderly patients predominate, in developing countries, AKI is a disease of the young and children, in whom volume-responsive "prerenal" mechanisms are common. Although overall mortality seems to be lower than in developed countries, this finding is not true across all age groups: In these regions, AKI affects predominantly the young and children and mortality is high. The difficulties of defining the incidence of AKI are especiallynotable when one searches for data on developing countries, the place of residence of more than 50% of the world's population. AKIis a potentially fatal, but reversible renal disease. The etiology, course, outcome differ in various parts of the world and also within India due to its climatic and geographic diversity and the variable standards of medical care.

Table No. 1: Acute kidney injury with respect to gender

In present study population, 62.72% individuals were male while 37.27%were female. Similarly, in a study by Paulo Roberto Santos et al[8] 66.3% were male while 33.7% female. Study by Xin Wang et al[9] showed that 64% were males and also in Eswarappa Met al[10] study AKI was more common in males accounting 63.6% Study by Luao X[11] showed majority were males. In study P S Priyamvada et al[12] 71.20% were male. However in a study conducted by Kai Singbartl et al[13], gender was not found to alter AKI susceptibility.

Table No. 2: Acute kidney injury with respect to age factor

In our study 110 patients were analyzed. There were 69 males and 41 females. Mean age of occurrence was 43.18 years. Maximum number of cases occurred in 2nd and 3rd decade due to it is working population majority works in farm so incident of snake are high in this age group. In study P S Priyamvada et al[12] mean age was 42.20 years very close to present study. In a study by Kai Singbartl et al[14], age was consistent risk factor. Even in a study by Eswarappa M et al[10] showed that median age of the patients was 55.5 years, lying in the age group of 51-60 years. This study highlighted the potential risk of AKI in elderly patients especially above the age of 60 years. In a study by Paulo Roberto Santod etal[15] mean age was 42.5. In a study by Koza Y[16] mean age was 42.5

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years. Average age was 70.54 years in a study by Xin Wang et al[17].

Etiology:

In the present study, there are various etiologies of AKI; 53.63% were vasculotoxic snake bite, followed by Sepsis27.27%, followed by obstetric causes 10.90%, hepatic causes 4.5%, followed by obstructive cause 2.72% and poisoning 0.90%.

Sepsis is the most common cause of AKI followed by hypovolemia in the above-mentioned studies. However, vasculotoxic snake bite induced AKI is the first most common cause in our study, followed by sepsis is second most common cause in our study. Hypovolemic shock is other common cause of AKI in many above mentioned studies. Other significant causes included obstetrical diseases, hepatic cause, obstructive, and poisoning.

The incidence of snake bite cases are high in our study area because majority of population lives in village and main occupation is farming and lots of cases of snake bite come to our hospital as it is only tertiary care center present in study area. Seasonal peaks of snakebite incidence are usually associated with increases in agricultural activity or seasonal rains, perhaps coinciding with unusual movement and activity by snakes. Snakebites and snakebite fatalities peak during the monsoon season in India and worldwide, probably reflecting agricultural activity, flooding, increased snake activity, and abundance of their natural prey. All the snake bite accidents in the current study had occurred in rural areas and majority of them happened during monsoon months.

Table No 18: Causes of Acute Kidney Injury requiring hemodialysis in Present and other different studies

Studies	Vasculotoxic snake bite	Sepsis	Obstetrical	Hepatic	Obstructive
Eswarappa et al[18]	37.2%	38.6%	8.4%	-	-
D Gupta et al[19]	8.1%	53.1%	3.2%	-	-
Oluyomi O. Okunola et al[20]	13.3%	35.5%	13.5%	-	-
Grace Igiraneza[20]	-	35%	26.9%	-	-
Shigehiko Uchino et al[21]	-	47.5%	-	5.7%	2.6%
Sanjay Vikrant et al[22]	82%	-	-	-	-
R Arul et al[23]	83%	-	-	-	-
Siva Kumar et al[24]	43%	-	-	-	-
Tarun k[25]	38%	-	-	-	-
Tushar B Patil[26]	57%	-	-	--	
Sanjay Vikrant et al[27]	-	53%	-	-	-
Present Study	53.63%	27.27%	10.90%	4.50%	2.72%

Vasculotoxic snake bite and Acute Kidney Injury:

Snake bite continues to be an important public health problem in tropical countries. No direct estimates on the incidence of snake bite are available in India as many of the cases get underreported. Extrapolated data suggest that India has the largest number of snake envenomation in the world with 80,000 envenomations every year with case fatality rate of.7–20%.[28] Recently it was reported that snake bite was responsible for 45,900 deaths in India in 2005 with only 23% of death occurring in hospital or healthcare facility. Snake bite can cause acute kidney injury (AKI) and the incidence of AKI varies country wise. In India the reported incidence is 13–32%,[28]4–8 while in Burma, Russell's viper bite is the most common cause of AKI. Even though there are studies on snake bite and AKI, they are short term and confined only till hospital stay. The long-term outcome of these patients who develop severe AKI and survived is not well described in literature.

Outcome of patients with AKI was considered good. In recent times there has been few studies showing that the long-term outcome is not benign as thought before. However, most of the long-term studies had included mainly patients with sepsis, nephrotoxic medication- induced AKI, or postsurgical AKI.[29]We could not come across any long-term outcome studies of snake bite-induced AKI. It is not known whether the long-term outcome is any different from other causes of AKI. Here we report our center's experience on outcome of 59 such patients who had dialysis-requiring AKI following snake bite. In our study persistent renal abnormalities in the form of either functional derangement, hypertension, or proteinuria were detected. A very significant percentage of the patients were lost to follow- up. This continues to be a problem in a developing country like India where many of the patients earn their livelihood on a daily basis and cannot afford follow-up.

Nephropathy is usually caused by bites by snakes with hemotoxic or myotoxic venoms. AKI is mainly observed following bites by the Viperidae group, sea snakes, but the substantial proportion of these cases result from viper bites. A number of factors contribute to AKI i.e. bleeding, hypotension, circulatory collapse, DIC, microangiopathic hemolytic anemia and direct nephrotoxicity of venom. Tubular necrosis and cortical necrosis are main cause of AKI[30]. Acute interstitial nephritis has also been described[30].

Recommended first-aid: reassurance, immobilization of the whole patient, especially their bitten limb, accelerated transport to medical care (summoned by emergency telephone helpline) ideally in recovery position. Unless neurotoxic elapid bite can be excluded, apply pressure-pad (simpler, more practicable than pressurebandage immobilization). Never use, recommend or condone tight (arterial) tourniquets. Investigations/laboratory tests: 20minute whole blood clotting test (20WBCT) is a simple, informative bedside test requiring only a new, clean, dry, ordinary glass tube, bottle, vial or syringe. Positive (non-clotting) result indicates severe consumption coagulopathy and need for immediate antivenom treatment. Indian "polyvalent anti-snake venom serum" (ASV) is raised against venoms "big four" species: spectacled cobra (N. naja); common krait (B. caeruleus), Russell's viper (D. russelii), saw-scaled viper (E. carinatus), but other species, now recognized as being important, are not covered. Antivenom treatment is indicated if/when patients with proven/ suspected snakebite develop one or more of the following signs. Systemic envenoming: haemostatic abnormalities [spontaneous systemic bleeding, coagulopathy Positive non-clotting 20WBCT, INR >1.2, or prothrombin time >4-5 seconds longer than control), or thrombocytopenia; neurotoxicity (bilateral ptosis, external ophthalmoplegia, paralysis etc.); cardiovascular abnormalities (hypotension, shock, cardiac arrhythmia, abnormal ECG); Acute kidney injury (oliguria/anuria, rising blood creatinine/ urea); haemoglobin-/myoglobin-uria (dark brown/black urine, +ve urine dipsticks, other evidence of intravascular haemolysis/generalized rhabdomyolysis); supporting laboratory evidence[31]. Local envenoming: local swelling involving more than half bitten limb (in absence of tourniquet) within 48 hr of the bite; swelling after bites on digits; rapid extension of swelling beyond wrist/ankle within few hours of bites on hand/foot); enlarged tender lymph node draining bitten limb Antivenom should be given only when benefits exceeds risks[31]. Antivenom reactions: many patients develop early (within a few hours) or late (after 5 days or more) reactions. Depending on type of antivenom and dose, the incidence may be as high as 81% (43% severe) of early anaphylactic or pyrogenic reactions or as low as 3.5%. IgE-mediated Type I hypersensitivity after previous exposure to equine serum is uncommon. Early anaphylactic reactions (1 - 180 minutes after starting antivenom) can have all classic features of anaphylaxis from urticaria to life-threatening shock, bronchospasm and angio-oedema. Most are attributable to complement activation by IgG aggregates/residual Fc fragments, or stimulation of mast cells/basophils by antivenom proteins. Pyrogenic (endotoxin) reactions (within 1-2 hours) involve rigors, fever (risk of febrile convulsions in children), vasodilatation, hypotension and a fall in blood pressure. Attributable to pyrogen contamination during manufacture. Late (serum sickness-type) reactions (1-12, mean 7, days after treatment) involve fever, nausea, vomiting, diarrhoea, itching, recurrent urticaria, arthralgia, myalgia, lymphadenopathy, periarticular swellings, mononeuritis multiplex, proteinuria, immune complex nephritis, rarely encephalopathy. Prediction: skin and conjunctival "hypersensitivity" tests, often recommended in "package inserts" predict acquired IgE-mediated Type I hypersensitivity to horse/sheep proteins, but not large majority of early (anaphylactic) or late (serum sickness type) antivenom reactions. Their use is strongly discouraged. Prevention of reaction A powerful, well-designed study in Sri Lanka showed that adrenaline (0.25 ml/ mg of 0.1% solution subcutaneously) given before antivenom

was started reduced severe reactions by 43% (95% CI 25–67) at 1 h and by 38% (95% CI 26–49) up to and including 48 h after antivenom administration; hydrocortisone (200 mg intravenously) and promethazine (25 mg intravenously) were ineffective. Hydrocortisone negated benefit of adrenaline. Dilution and slow infusion (10-120 minutes) does not alter risk of reactions. At the earliest sign of early anaphylactic antivenom reaction, temporarily suspend antivenom administration and giveintramuscular injection of 0.1% adrenaline, initial adult dose 0.5 ml/mg (0.01 mg/kg body weight for children). Since severe, life-threatening anaphylaxis can evolve rapidly, adrenaline must be available at the bed-side and should be given at the first sign of anaphylaxis (e.g. when itching, tachycardia, restlessness develop and a few spots of urticaria appear). Repeat adrenaline every 5-10 minutes if reaction persists or worsens. Adrenaline safe in pregnancy. In case of asthmatic symptoms, given salbutamol bronchodilator. After adrenaline, give intravenous antihistamine anti-H1 blocker (e.g. chlorphenamine maleate, adults 10 mg, children 0.2 mg/ kg intravenously) and hydrocortisone[31].

In present study 53% patients with snake bite induced AKI required hemodialysis. Out of 8 patient of post snake bite cellulitis 3 patients required debridement, AKI was present before debridement.

Sepsis and Acute Kidney Injury: Acute kidney injury (AKI) is frequently associated with sepsis. Its incidence varies from 11 to 42% [32, 33]. Sepsis is the most common cause of AKI in critical care patients, accounting for 50% of cases in the ICU[33]. AKI incidence rate and severity correlate with the severity of the underlying sepsis[34]. Septic AKI is a hallmark of severe sepsis and septic shock and is associated with worse outcomes including prolonged hospital length of stay, fewer ventilator-free days and increased mortality when compared to patients with non-septic AKI[33,34]. It appears that septic AKI is different than non-septic AKI with respect to the underlying contributing factors, and severity of injury and outcomes. Septic patients develop more severe AKI than in non-septic patients and even patients with non-severe infections (e.g. pneumonia) have a significantly higher incidence of AKI. Recent studies have identified several pathophysiological mechanisms that are discussed in detail elsewhere in this journal. Several factors have been implicated in the pathogenesis of septic AKI. Hemodynamic changes in the macro circulation (i.e.: vasodilatation and increased cardiac output), and systemic and renal microcirculation contribute to renal hyperemia coupled with inefficient cellular oxygen extraction. The renal medulla is particularly sensitive to these hemodynamic perturbations and resultant hypoxemia, since it is already functioning at a lower PaO2 level, especially in the nephrons of the cortico-medullary junction. Sepsis is also associated with systemic inflammation and endothelial dysfunction, which also have been shown to contribute to renal injury and enhance microcirculation perturbations. The stress response is altered in sepsis; the earliest phase characterized by a short-lived hypo-responsiveness, which is followed by a dramatic phase of hyper-responsiveness. In the hyper-reponsive phase, both pro- and anti-inflammatory cytokines are released in the systemic circulation, and endothelial exposure of local adhesion receptors leads to platelet aggregation with microthrombi formation and enhanced leucocyte recruitment. This excessive immune response with deregulation between the pro- and anti-inflammatory mediators contributes to further downstream or distant organ damage such as AKI. The later phase of sepsis is characterized by hypo-functionality of the immune system, which may last from several days to weeks, and increases susceptibility to new or recurrent infections. The complex interplay of various factors during the course of sepsis makes it difficult to identify the exact mechanism and pathways in septic AKI.

Clinical manifestations of sepsis with AKI depend on many factors. The sequence of insults (AKI preceding sepsis, versus sepsis preceding AKI, or simultaneous presentation) may influence the patient's initial clinical features. One must therefore keep sepsis features in mind when evaluating a patient with AKI and conversely evaluate for AKI when a patient presents with sepsis.

Signs and symptoms of sepsis vary not only with organ involvement, but also from one individual to another due to patient- and disease-specific characteristics and susceptibilities. Signs of sepsis reflect the phase of the disease and range from features limited to the primary organ (e.g. pneumonia) to severe multi-organ dysfunction syndrome (MODS) and septic shock. Caregivers must therefore be alert for any signs of infection, sepsis or septic shock when evaluating patients for renal failure, and conversely it is important to, frequently monitor renal function (along with other organ involvement) in patients with documented or suspected sepsis.

Septic AKI is defined by AKI in the presence of sepsis without another significant

contributing factor explaining AKI. Recent diagnostic and staging criteria for AKI included an absolute increase of serum creatinine of 0.3mg/dl over 48 hours, a relative change in serum creatinine 1.5-1.9 times baseline over 7 days, or a urine output of less than 0.5 ml/kg/h for six hours. Severity of septic AKI may be classified using the well documented consensus KDIGO criteria for AKI staging, and outcomes appear to be correlated with the presence and severity of AKI as defined by this classification system[34].Several pitfalls are associated with the use of serum creatinine and urine output for the diagnosis of septic AKI. Serum creatinine is a late, insensitive marker of renal injury, for a number of reasons. Because of the half-life of circulating creatinine, increments in serum creatinine lag decrements in glomerular filtration rate (GFR) by hours. Furthermore, the time to achieve a new steady state concentration that fully reflects the degree of GFR loss is delayed by multiples of a prolonged serum creatinine half-life, reflected in changes over days rather than hours. Additionally, in critically ill septic patients hemodilution in hypotensive patients receiving aggressive fluid resuscitation with positive fluid balance masks serum creatinine increments, and has been shown to delay AKI diagnosis by a further day. Sepsis has also been show to reduce muscular production of creatinine, even without weight loss, further reducing the utility of serum creatinine as a marker of septic AKI.

In present study 27.27% patients with sepsis induced AKI required hemodialysis.

Obstetrical causes and Acute Kidney Injury: The incidence of acute kidney injury in pregnancy (P-AKI) has declined significantly over the last three decades in developing countries. However, it is still associated with significant fetomaternal mortality and morbidity. The diagnosis of P-AKI is based on the serum creatinine increase. The usual formulas for estimating glomerular filtration rate (GFR) are not validated in this population. During the first trimester of gestation, AKI develops most often due to septic abortion or hyperemesis gravidarum. Septic abortion related AKI with respect to total AKI was not found in our study. Prevention of unwanted pregnancy and avoidance of septic abortion are keys to eliminate abortion associated AKI in early pregnancy. However, we have not seen AKI on account of hyperemesis gravidarum in our study. In the third trimester, the differential diagnosis of AKI in association with pregnancy specific conditions namely preeclampsia/HELLP syndrome, acute fatty liver of pregnancy and thrombotic microangiopathies of pregnancy (P-TMA) is more challenging, because these 3 conditions share several clinical features of thrombotic microangiopathy which makes the diagnosis very difficult on clinical grounds. It is imperative to distinguish these conditions to make appropriate therapeutic decisions. Typically, AFLP and HELLP syndrome improve after delivery of the fetus, whereas plasma exchange is the first-line treatment for pregnancy associated thrombotic microangioathies (P-TMA). We observed that puerperal sepsis is the most common cause of AKI in late third trimester and postpartum periods followed by HELLP syndrome and postpartum hemorrhage. Pregnancy-associated thrombotic microangiopathies (aHUS/TTP) and AFLP are rare causes of AKI during pregnancy in developing countries. Acute kidney injury that occurs during pregnancy or in the post-partum period (PR-AKI) is a serious obstetric complication with risk of significant associated maternal and fetal morbidity and mortality. Recent data indicates that the incidence of PR-AKI is increasing, although accurate calculation is limited by the lack of a uniform diagnostic criteria that is validated in pregnancy. Hypertensive and thrombotic microangiopathic disorders of pregnancy have been identified as major contributors to the burden of PR-AKI. As is now accepted regarding preeclampsia, HELLP syndrome and atypical hemolytic uremic syndrome, it is believed that PR-AKI may have long-term renal, cardiovascular and neurocognitive consequences that persist beyond the post-partum period. Further research regarding PR-AKI could be advanced by the development of a pregnancy-specific validated definition and classification system; and the establishment of refined animal models that would allow researchers to further elucidate the mechanisms and sequelae of the disorder.

In our study, obstetrical causes included puerperal sepsis, HELLP syndrome, Postpartum hemorrhage and abruptio placenta.In study by Grace Igiraneza[35] pregnancy related causes of AKI found to be 26.9%. Another study by Oluyomi O. Okunola et al[20]pregnancy related causes of AKI found to be 13.5%. In our study pregnancy related causes of AKI were seen in 10.90% of AKI cases.

Hepatic Cause and Acute Kidney Injury: The pivotal prognostic role of renal function in cirrhosis is reflected by the inclusion of serum creatinine (sCr) in the Model for End Stage Liver Disease (MELD) Score, which is currently used for assessment of severity of liver disease and prioritization of patients with advanced liver disease for liver transplantation. As a consequence of systemic and splanchnic arterial vasodilatation

and consecutive reduction in effective circulating blood volume, renal perfusion may be critically impaired in patients with advanced cirrhosis and portal hypertension. As a result, patients with cirrhosis are prone to developing renal dysfunction.

Acute kidney injury (AKI), defined by a significant reductionin glomerular filtration rate (GFR) over a short time period, is a common and severe complication in patients with cirrhosis and is often triggered by a precipitating event can be overdose of diuretics, large-volume paracentesis without albumin replacement, gastrointestinal bleeding, bacterial infections. AKI has an estimated prevalence of approximately 20–50% among hospitalized patients with cirrhosis and patients with cirrhosis are more likely to develop renal failure compared to individuals without liver disease. AKI is associated with poor prognosis and represents an important predictor for short-term mortality in patients with cirrhosis The spectrum of causes for AKI in cirrhosis includes (i)prerenal AKI (i.e. hypovolemia due to gastrointestinal bleeding, aggressive diuretic treatment, lactulose-induced diarrhea or infections), (ii) the hepatorenal syndrome-type AKI (HRS-AKI), which is defined as a potentially reversible deterioration of renal function unresponsive to volume resuscitation, caused by renal vasoconstriction in the absence of alternative identifiable causes (iii) intrinsic causes such as acute tubular necrosis and, although very rare, (iv) postrenal causes[36]. With a yearly incidence of 8–12%, HRS-AKI is quite common in decompensated cirrhosis with ascites. The correct classification of AKI is essential since HRS-AKI, representing one of the most lethal complications of portal hypertension, requires a specific treatment approach. However, despite adequate treatment mortality is still about 60% and higher. HRS-AKI is a diagnosis by exclusion and thus, often difficult to establish.

The most recent definition criteria were published in 2015 byboth a community of hepatologists (ICA) together with the Acute Dialysis Quality Initiative (ADQI), a community of nephrologists, and reclassified the former type 1HRS as a special entity of acute kidney injury: the 'HRS type of AKI' (HRS-AKI)[36].

AKI in cirrhosis is defined as an acute increase in serum creatinine of0.3 mg/dL within 48hours or by50% from a stable baseline serum creatinine (sCr) within 3months (presumed to have developed within the past 7 days when no prior readings are available[36]. The main modifications over the former, rather stringent criteria that were based on absolute serum creatinine level, was abandoning the arbitrary threshold of sCr 1.5 mg/dL to diagnose AKI, since milder degrees of renal failure in patients with cirrhosis had often remained underdiagnosed[36]. In addition, the use of urine output as part of the diagnostic criteria was eliminated, since many patients with cirrhosis and ascites maintain a preserved renal function despite being oliguric due to sodium and water retention.

Obstructive cause and Acute Kidney Injury: Aobstruction anywhere along the urinary tract—from the kidneys, where urine is produced, to the urethra, through which urine leaves the body—can increase pressure inside the urinary tract and slow the flow of urine. An obstruction may occur suddenly or develop slowly over days, weeks, or even months[37]. An obstruction may completely or only partially block part of the urinary tract. Sometimes only one kidney is affected, but obstruction may affect both kidneys.

The prevalence of urinary tract obstruction ranges from five in 10,000 to five in 1,000 depending on the cause. In Men, particularly those older than 60, are also more likely to be affected because, as men age, the prostate gland tends to increase in size.

Normally, urine flows out of the kidneys at extremely low pressure. If the flow of urine is obstructed, urine backs up behind the point of blockage, eventually reaching the small tubes of the kidney and its collecting area (renal pelvis), swelling (distending) the kidney and increasing the pressure on its internal structures. Such kidney distention is called hydronephrosis. The elevated pressure due to the obstruction may ultimately damage the kidney and can result in loss of its function. When the flow of urine is obstructed, stones (calculi) are more likely to form. An infection may develop when the flow of urine is obstructed because bacteria that enter the urinary tract are not flushed out. If both kidneys are obstructed, kidney failure may result.

Long-standing distention of the renal pelvis and ureter can also inhibit the rhythmic muscular contractions that normally move urine down the ureter from the kidney to the bladder (peristalsis). Scar tissue may then replace the normal muscular tissue in the walls of the ureter, resulting in permanent damage.

In present study Most Common cause was Benign prostatic hyperplasia (BPH), prostate cancer, followed by renal calculi. In present study 2.72% AKI cases were because of obstructive cause.

Poisoning and Acute Kidney Injury: The chemical composition of paraquat is C12H14N2 and its molecular weight is 186.3. The lipid

hydroperoxide induced by paraquat destroys cell membranes and kills the tissue of green plants via an oxidative process, which is initiated by the formation of superoxide as a result of the suppressed reduction of NADP in the course of photosynthesis when exposed to this herbicide. However, outside the living plant, this powerful superoxide is changed into a relatively inert chemical at high temperature or when exposed to soil mineral. Consequently, when paraquat is sprayed in the environment, it quickly loses its toxicity. Severely poisoned patients usually die of circulatory collapse quickly, although if the clinical course is prolonged, pulmonary fibrosis can develop. A paraquat dose of 30 mg/kg may be fatal. This is equivalent to 7-8 mL of the 24.6% solution sold commercially. With a dose of less than 7-8 mL, the survival rate is about 13% with no treatment, and increases to 73% with active treatment. Even with a dose exceeding 7-8 mL, the recent survival rate is as high as 50% with active treatment[38]. Most paraquat is absorbed in the jejunum; nevertheless, only 17.6% of it gets absorbed. This means that most resorption through the intestine is at a low rate.

Despite this sluggish process, resorption through the small intestine may cause death. Therefore, urgent gentle gastric lavage is required to reduce the absorption and this should be followed by the administration of Fuller's earth and activated charcoal to inactivate the poison in the stomach. To expedite its excretion, laxatives, hydration, and diuretics should be started as soon as possible. As the Proud foot curve predicts, the plasma paraquat concentration is the factor with the greatest influence on the prognosis. The plasma paraquat concentration peaks within 2 hours and then decreases. As the curve shows, the initial decrease is faster. This is called the distribution phase, and has a half life of about 5 hours and volume of distribution of 1.2-1.6 L/kg. The half life in the subsequent elimination phase is about 84 hours.

Paraquat intoxication is characterized by multi-organ failure, causing substantial mortality and morbidity. Many paraquat patients experience acute kidney injury (AKI), sometimes requiring hemodialysis. In the study by Cheng-Hao Weng[39] mortality rate was 80% in AKI patients requiring hemodialysis. In study Cheng-Hao Weng[40] mortality rate was 54% in AKI patients requiring hemodialysis. In present study mortality rate was 0.00%.

OUTCOME: In Present study, out of 110 AKI patients 89 of them survived and 21expired. Total Mortality was 19.09%. Out of 21 deaths, 8 were related to vasculotoxic snake bite, 8 were of septic AKI death, 3 related to hepatic diseases, 2related to obstetrics cause. The mortality rate of each etiology is as follows,

Table No 19: Etiology wise Mortality percentage.

Etiology	Mortality in Percentage%
Vasculotoxic Snake Bite	7.27
Sepsis	26.66
Hepatic Cause	60
Obstetric Cause	16.66

Table no 20: Total mortality Percentage in different studies.

Studies	Mortality in Percentage%
Sandeep Mahajan et al[41]	64.40
Fanny Garnier et al[42]	47
Eswarappa et al[43]	37.6
Riccardo Maria et al[44]	36
Grace Igiraneza et al[35]	34.1
Selby et al[45]	21.9
Present Study	19.09

In present study Vasculotoxic snake bite induced AKI requiring hemodialysis cases were highest that is 59/110(53.63%), out of which 51/59survived and 8/59 of them died. In our study Vasculotoxic snake bite is most common cause of AKI requiring hemodialysis. Early administration of adequate antisnake venom has reduced complications like AKI, DIC. All 59/110 patients of vasculotoxic snake bite induced AKI before initiation of dialysis were managed conservatively with use of diuretics and appropriate fluid management. But all of them did require renal replacement therapy along with antisnake venom, diuretics and fluid management.

Sepsis induced AKI were second highest number of cases. Out of110 cases 30 were sepsis related. 22 of these cases survived and 8 of them expired.Sepsis is second Major causes of sepsis in our study were due to infections like Urosepsis, Cellulitis, Pneumonia, Pyelonephritis. Appropriate antibiotics were administered as per the clinical manifestations and culture and sensitivity reports. Early use of appropriate antibiotics did show better results and survival. Although with vigilance use of antibiotics and supportive treatment 8 patients of sepsis died. Late presentation of patients was one of the most important factors, along with older age were contributing to the deterioration of clinical condition of the patients and death.

Obstetric cause induced AKI requiring hemodialysis in our study were 12 cases. Puerperal sepsis were 8, 2 of them were due to HELLP syndrome, 1 due to abruption placenta and 1 due to postpartum hemorrhage. Out of 12 cases 2 of them died attributing 16.66% of the mortality. Before initiation of dialysis were managed conservatively with use of diuretics and appropriate fluid management. But all of them did require renal replacement therapy along with a rational antibiotic according to sensitivity, diuretics and fluid management. Abruption placenta case did show improvement and had normal urine output and renal function test at the time of discharge. One patient of HELLP syndrome did not show improvement and died due to uremic complications. Another patient of puerperal sepsis did not response expired due to Sepsis and Uremic complications.

In Present study Mortality due to hepatic diseases was 60%. Total cases of hepatic disease-causing AKI were 5.Out of 5, 2 of them survived and 3 of them died contributing 60% to the mortality. All cases were liver cirrhosis causing AKI. Due to reduced renal perfusion AKI developed in liver cirrhosis patients. Salt restriction, appropriate fluid management, use of diuretics, albumin and terlipressin did show some improvement in the patient. These supportive management measures just prolonged the life of the patients but was not the definitive management (liver transplantation) and hence patients expired.

In Present study there were 3 patient of Obstructive cause AKI requiring Hemodialysis. One of Ca Prostate, second was BPH, third was renal calculi. All of them survived, mortality was 0.00%.

In Present study there was one case of Paraquat compound poisoning induced AKI requiring hemodialysis, he survived and there was 0.00% mortality.

Conclusion: AKI is a worsening problem, From a worldwide perspective, there is a clear need to understand the epidemiology of AKI more accurately. Use of standardized definitions and descriptions of existing at-risk and high-risk populations, both in community and institutional settings, are the first step to improve outcomes.

A patients with Acute Kidney Injury requiring hemodialysis early initiation of Hemodialysis is associated with improved survival. Increasing AKI severity was associated with increased mortality. In snake bite induced AKI, Viper bite induced AKI showed poor outcome, the time lag for administration of ASV had worse outcomes, those who completed ASV before the onset of renal failure had better prognosis. Cellulitis and regional lymphadenopathy indicated the amount of toxin released and presence of fang marks showed maximum envenomation. Presence of Proteinuria, hematuria, anemia and thrombocytopenia were associated with poor outcomes. Signs of DIC were associated with worse outcomes.

Septic AKI exerts an important and independent increase in the risk for hospital death. In survivors, septic AKI is associated with prolonged ICU and hospital stays but also a trend toward greater recovery of kidney function.Pregnancy is an independent risk factor of AKI among women of childbearing age. Puerperal sepsis is the most common risk factor for developing AKI in pregnancy. Pregnancy-related AKI is associated with increased risk of mortality, higher medical. Results from this study may help to increase the awareness of pregnancy-related AKI and to improve AKI-related care.

Chronic liver disease is associated with reduced chances for recovery may be due to compromised renal perfusion in the setting of hepatorenal physiology.

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