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| Indian | A ST CON | TUDY OF CLINICAL PROFILE OF PATIENTS WITH AMUNITY VERSUS HOSPITAL ACQUIRED ACUTE NEY DISEASE | KEY WORDS: Acute kidney injury (AKI), Community acquired (CA-AKI), Hospital acquired (HA- AKI) |
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| ABSTRACT | For Acute Dialysis Qua KDIGO clinical practice community acquired AF Methods: This is a pro- irrespective of their co- function at the time of considered as HA-AKI. RESULTS: Frequency of values at admission, af statistically significant (61.1% in CA-AKI and H (26.3%). Patients with were seem in patients outcome of patients an Conclusion: The freque common in ICUs, where CA-AKI have better sho | dney injury (AKI) depicts the abrupt decline in renal function mostly of lity Initiative (ADQI) Modified RIFLE criteria & AKIN criteria were p guidelines. We aim to evaluate the characteristics and prognosis (I (CA-AKI) patients who were admitted in the hospital for their clinic ospective cross sectional hospital based study design on comparis morbities & falling under Kidney Disease: Improving Global Outce admission to the hospital was considered as CA-AKI. AKI develop f CA-AKI was significantly higher as compared to HA-AKI. (p<0.00 ter 48 hrs and at 7th day from admission in both, CA–AKI as wel o < 0.001). Highest percentage of patients were in KDIGO stage 3, IA-AKI respectively. Need for dialysis was higher in patients with HA CA-AKI had significantly higher rate of complete recovery than H, with HA-AKI (38.8%) as compared to CA-AKI (4.9%). Thus, there d type of AKI (p<0.001). ency of community acquired AKI was much higher as compared eas CA-AKI is common in medical wards. Higher mortality was found rt and long-term outcomes as compared to HA-AKI. The reason may volume depletion which could be corrected easily. | proposed earlier, are now replaced by s of hospital acquired AKI (HA-AKI) & cal profile and outcome. son of HA-AKI & CA-AKI patients AKI omes (KDIGO) criteria. Impaired renal ping after 48 h of hospitalization was 1). The Difference in serum creatinine II as HA-AKI patients was found to be in both the groups of AKI (50.9% and A-AKI (44.4%) as compared to CA-AKI A-AKI patients. Higher mortality rates e was significant association between d to hospital acquired AKI. HA-AKI is d in HA-AKI than CA-AKI. Patients with |

INTRODUCTION

Acute kidney injury depicts the abrupt decline in renal function mostly occuring over the course (hours to days) and ends in retention of metabolic waste products and dysregulation of fluid, electrolytes & acid-base homeostasis.^{1,2} The etiological spectrum of AKI is notably different between developing and developed nations. It is related to environmental, social and economic circumstances. Sepsis, surgery and trauma are the most common causes of AKI in developed countries. In developing countries, acute diarrheal diseases and tropical diseases still prevail.³ The aetiology, course, and outcome of AKI differ in various parts of India.^{4,5} In 2004, the Acute Dialysis Quality Initiative (ADQI) group, comprising of experts in the fields of nephrology and critical care medicine, published the RIFLE classification⁶⁻⁸. This was a new consensus and evidence-based definition for AKI.⁶ In 2007, a modified version of the RIFLE criteria was published by the AKI Network (AKIN), an international collaboration of nephrologists and intensivists, known as the AKIN criteria.9 This existing classification scheme for AKI has been replaced by KDIGO clinical practice guidelines (2012) which incorporates the better elements of both RIFLE and AKIN criteria.

The kidneys are relatively unique among other organs of the body due to its ability to recover from almost complete loss of function. AKI may develop in a wide variety of settings including ambulatory, outpatients, hospitalized & particularly critically ill patients. AKI is associated with substantial morbidity and mortality.²

Although recovery of renal function occurs in majority of patients surviving an episode of AKI, many patients remain dialysis dependant or are left with severe renal impairment. More recently it has been recognized that even patients who have complete or near complete recovery of renal function are at increased risk of CKD and that superimposition of AKI on CKD is associated with acceleration in the rate of progression to ESRD.

AKI maybe community acquired (CA-AKI) or hospital acquired (HA-AKI). CA-AKI is classified as patients diagnosed with impaired renal function at admission to the hospital. HA-AKI is classified as any patient developing AKI after 48 hours of admission to the hospital. Common causes of community acquired AKI include volume depletion, adverse effects of medications and obstruction of the urinary tract. The most common clinical settings for hospital acquired AKI are sepsis, major surgical procedures, critical illness involving heart or liver failure, intravenous iodinated contrast administration and nephrotoxic drug administration². AKI complicates approximately 5-7% of hospital admission & 30% of admission to ICU patients. The risk of AKI is contributed by the acute insult and background morbidity. Acute insult may be in the forms of sepsis and hypoperfusion, toxicity, obstruction & parenchymal kidney disease. Background morbidities in the form of elderly, CKD, cardiac failure, liver failure, diabetes mellitus, vascular disease, nephrotoxic medication also contribute to insult.¹⁰Since the implementation of Kidney Disease: Improving Global Outcomes (KDIGO) guidelines to diagnose and upgrade AKI, very few studies have evaluated the impact of its early recognition and management on subsequent outcome. There is lack of data to assess the impact of early AKI management on mortality, length of hospital stay, need of renal replacement therapy (RRT) or subsequent kidney function. In this study, we therefore aim to evaluate the characteristics and prognosis of hospital acquired AKI (HA-AKI) and community acquired AKI (CA-AKI) patients who were admitted in the hospital for their clinical profile and outcome.

AIMS AND OBJECTIVES

- 1) To study the clinical profile of patients with community and hospital acquired acute kidney injury.
- 2) To determine the frequency of community and hospital acquired acute kidney injury.

METHODS AND MATERIAL Source of Data

The study was conducted in patients coming with elevated serum creatinine as per KDIGO guidelines and symptoms suggestive of acute kidney injury in the medical wards of Geetanjali Hospital, Udaipur

Sample Size: 120 cases Sample procedure: Cross-sectional study Duration: January 2017 – October 2018

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INCLUSION CRITERIA

- All patients between 18 yrs to 75 yrs of age developing AKI irrespective of their co-morbities.
- Diagnosing criteria for AKI According to Kidney Disease: Improving Global Outcomes (KDIGO) criteria

EXCLUSION CRITERIA

- Age less than 18 years.
- Age more than 75 years.
- Diagnosed as Chronic kidney disease (CKD).
- CKD on maintenance hemodialysis (MHD).
- Pregnant females.

Study Design:

All the patients of age more than 18 years, admitted during the study period with acute kidney injury (AKI) or who developed acute kidney injury during the hospital stay have been included. Diagnosing criteria for AKI was according to KDIGO criteria. Patients below 18 years and above 75 years of age have been excluded, as KDIGO criteria has limitations for inclusion of these patients.

Impaired renal function at the time of admission to the hospital was considered as CA-AKI. AKI developing after 48 h of hospitalization was considered as HA-AKI.

- CA-AKI = Community Acquired Acute Kidney Injury
- HA-AKI = Hospital Acquired Acute Kidney Injury

OBSERVATION AND RESULTS

A total number of 120 cases of acute kidney injury (by KDIGO criteria), admitted in Geetanjali Medical College and Hospital, Udaipur, who met the inclusion criteria were studied. The following observations were noted:

TABLE 2: PREVALENCE OF AKI AND ITS DISTRIBUTION

| Type of AKI | No of Patients | Z TEST | P VALUE |
|-------------|----------------|--------|---------|
| CA-AKI | 102 (85%) | 5.99 | <0.001 |
| HA-AKI | 18 (15%) | | |
| Total | 120 (100%) | | |

Out of the 120 patients of AKI, 102 (85%) patients were classified as having CA-AKI and 18 (15%) patients as having HA-AKI. Thus, the frequency of CA-AKI was statistically significantly higher as compared to HA-AKI (P<0.001).

TABLE 3: AGE WISE DISTRIBUTION

| Age (years) | CA-AKI | HA-AKI | No of patients | | |
|-------------|---------------|--------------|----------------|--|--|
| 18-30 | 11 (10.7%) | 0 | 11 (9.16%) | | |
| 31-40 | 15 (14.7%) | 1 (5.5%) | 16 (13.33%) | | |
| 41-50 | 20(19.6%) | 6 (33.3%) | 26 (21.66%) | | |
| 51-60 | 26 (25.4%) | 9 (50%) | 35 (29.16%) | | |
| 61-70 | 23 (22.5%) | 2 (11.1%) | 25 (20.83%) | | |
| >70 | 7 (6.8%) | 0 | 7 (5.83%) | | |
| Total | 102 (85%) | 18 (15%) | 120 (100%) | | |
| Mean Age | 51.54 ± 14.93 | 53.22 ± 7.22 | 51.94 ± 14.11 | | |
| T value | 0.467 | | | | |
| P value | 0.642 | | | | |

Out of the 102 patients with CA-AKI, maximum i.e. 26 patients (25.4%) were from the age group of 51-60 years, followed by, 23 patients (22.5%) between 61-70 years, 20 patients (19.6%) between 41-50 years, 15 patients (14.7%) between 31-40 years and 11 patients (10.7%) between 18-30 years. Out of the 18 patients with HA-AKI, maximum i.e. 9 patients (50%) were from the age group of 51-60 years, followed by, 6 patients (33.3%) between 41-50 years, 2 patients (11.1%) between 61-70 years and 1 patient (5.5%) between 31-40 years. The mean age of patients with CA-AKI was 51.54 ± 14.93 and HA-AKI was 53.22 ± 7.22 , which was statistically insignificant. Thus, the maximum number of patients with CA-AKI were in the 6th and 7th decades of life and in the 5th and 6th decades with HA-AKI.

| TABLE 4: SEX WISE DISTRIBUTION | | | | | | |
|--------------------------------|------------|------------|----------------|--|--|--|
| Sex | CA-AKI | HA-AKI | No of patients | | | |
| Male | 59 (57.8%) | 11 (61.1%) | 70 (58.3%) | | | |
| Female | 43 (42.1%) | 7 (38.8%) | 50 (41.6%) | | | |
| Total | 102 (85%) | 18 (15%) | 120 (100%) | | | |
| x ² value | 0.067 | | | | | |
| P value | >0.05 | | | | | |

Out of 120 patients of AKI, 70 patients (58.3%) were males, while 50 (41.6%) were females. The sex ratio was 1.4:1. Out of 102 patients with CA-AKI, 59 (57.8%) were males whereas, 43 (42.1%) were females. Out of 18 patients with HA-AKI, 11 (61.1%) were males while, 7 (38.8%) were females. Thus, CA-AKI and HA-AKI both have male predominance, but statistically there was insignificant association of gender and type of AKI (P>0.05).TABLE 5: DISTRIBUTION OF CLINICAL PARAMETERS OF AKI

| Clinical Parameters | CA-AKI | HA-AKI | No of patients |
|----------------------------|------------|------------|----------------|
| Diarrhea | 57 (55.8%) | 0 | 57 (47.5%) |
| Vomiting | 61 (59.8%) | 8 (44.4%) | 69 (57.5%) |
| Oliguric Output | 58 (56.8%) | 15 (83.3%) | 73 (60.8%) |
| Breathlessness | 12 (11.7%) | 8 (44.4%) | 20 (16.6%) |
| Altered Sensorium | 5 (4.9%) | 7 (38.8%) | 12 (10%) |
| Edema | 8 (7.8%) | 2 (11.1%) | 10 (8.3%) |
| Non-specific Uremic | 23 (22.5%) | 4 (22.2%) | 27 (22.5%) |
| Symptoms | | | |
| Jaundice | 5 (4.9%) | 6 (33.3%) | 11 (9.2%) |
| x ² value | 44.85 | | |
| P value | <0.001 | | |

The common clinical features observed in our study were oliguria (60.8%), vomiting (57.5%), diarrhea (47.5%), non-specific uremic symptoms (22.5%) breathlessness (16.6%), altered sensorium (10%), jaundice (9.2%) and edema (8.3%). Among patients of HA-AKI we have found high parameters of oliguria (83.3%), followed by vomiting (44.4%) and breathlessness (44.4%), while, in CA-AKI we have found high parameters of vomiting (59.8%), followed by oliguria (56.8%) and diarrhea (55.8%).

It has been observed that there was significant association between clinical features and type of AKI i.e. there was change in clinical feature with type of AKI (p<0.001).

TABLE 6: DISTRIBUTION OF PATIENTS ACCORDING TO CO-MORBIDITIES

| | | Ha-aki | | X ² | Р |
|-------------|-------------|-------------|------------|----------------|-------|
| morbidities | | | | value | Value |
| PRESENT | 15 (14.71%) | 6 (33.33%) | 21 (17.5%) | 2.5 | 0.11 |
| ABSENT | 87 (85.29%) | 12 (66.67%) | 99 (82.5%) | | |
| Total | 102 (100%) | 18 (100%) | 120 (100%) | | |

Out of 102 CA-AKI patients, 15 (14.71%) had co-morbidities whereas, out of 18 patients of HA-AKI, 6 (33.33%) had co-morbidities. Thus, it has been observed that there was insignificant association between the presence of co-morbidities and type of AKI (p=0.11).

The percentage of co-morbidities in HA-AKI patients (6 out of 18 patients i.e. 33.33%) as compared to CA-AKI (15 out of 102 patients i.e. 14.71%) was more and this difference in proportion was statistically significant (P<0.001).

TABLE 7: TYPE OF CO MORBIDITY AND ITS DISTRIBUTION

| Co-morbidities | CA-AKI | HA-AKI | No of patients |
|-----------------------|-------------|------------|----------------|
| DM | 5 (4.9%) | 5 (27.7%) | 10 (8.3%) |
| Hypertension | 6 (5.8%) | 0 | 6 (5%) |
| CAD | 2 (1.9%) | 1 (8.3%) | 3 (2.5%) |
| CVA | 1 (0.9%) | 0 | 1 (0.8%) |
| COPD | 1 (0.9%) | 0 | 1 (0.8%) |
| Total | 15 (14.71%) | 6 (33.33%) | 21 (17.5%) |
| 2 value | 5.48 | | |
| P value | 0.241 | | |

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Co-morbidities were present in 21 (17.5%) patients of AKI. Out of these 21 AKI patients, 15 (71.43%) patients had CA-AKI and 6 (28.57%) patients had HA-AKI. Among the co-morbidities, diabetes was present in 10 (8.3%) patients, hypertension was present in 6 (5%) patients and CAD was present in 3 (2.5%) patients. CVA and COPD was present in 10(.8%) patient each. The commonest co-morbidity in patients with CA-AKI was HTN (5.8%), while in HA-AKI was DM (27.7%). It has been observed that there was insignificant association between co-morbidities and type of AKI (p = 0.241).

TABLE 8: ETIOLOGICAL PROFILE OF AKI

| Etiological | CA-AKI | HA-AKI | No of patients |
|-----------------------------------|------------|------------|-------------------|
| Acute Diarrheal Disease | 59 (57.8%) | 0 | 59 (46.6%) |
| Snake Bite | 25 (24.5%) | 0 | 25 (20.8%) |
| Sepsis | 0 | 12 (66.6%) | 12 (10%) |
| Tropical Acute Febrile Illness | 12 (11.7%) | 0 | 12 (10%) |
| Acute Glomerulonephritis | 1 (0.9%) | 3 (16.6%) | 4 (3.3%) |
| Ureteric calculi | 3 (2.9%) | 0 | 3 (2.5%) |
| Super vasmol Poisoning | 2 (1.9%) | 0 | 2 (1.6%) |
| Pigment Nephropathy | 0 | 2 (11.1%) | 2 (1.6%) |
| NSAID Induced | 0 | 1 (5.5%) | 1 (0.9%) |
| Total | 102 (100%) | 18 (100%) | 120 (100%) |
| x ² value | 114.12 | | |
| P value | <0.001 | | |

The most common cause of AKI was acute diarrheal disease (46.6%), followed by snake bite (20.8%), sepsis (10%), acute febrile illness due to tropical diseases (10%), acute glomerulonephritis (3.3%) and ureteric calculi (2.5%). In addition, higher frequency of sepsis (66.6%) was found in patients with HA-AKI, followed by, acute glomerulonephritis (16.6%), pigment nephropathy (11.1%) and NSAID induced (5.5%). Whereas, in patients of CA-AKI, higher frequency of acute diarrheal disease (57.8%) was found, followed by, snake bite (24.5%), acute febrile illness due to tropical diseases (11.7%) and ureteric calculi (3.9%) respectively. It has been observed that there was significant association between etiological profile and type of AKI (p < 0.001).

TABLE 9: HAEMATOLOGICAL AND BIOCHEMICAL PARAMETERS IN AKI

| Biochemical | CA-AKI | HA-AKI | Т | Р |
|-----------------------------|-------------------|-------------------|-------|--------|
| parameters | | | value | value |
| Hemoglobin | 9.93 ± 1.58 | 9.56 ± 2.04 | 0.88 | 0.38 |
| TLC (mm3) | 7029.37 ± 2102.32 | 7264.28 ± 4160.39 | 0.37 | 0.71 |
| APC (lakhs) | 3.03 ± 0.74 | 2.71 ± 0.95 | 1.62 | 0.11 |
| RBS (mg/dl) | 105.9 ± 5.85 | 105.72 ± 4.35 | 0.12 | 0.90 |
| Serum Creatinine (mg/dl) | 2.94 ± 1.21 | 1.13 ± 0.14 | 6.32 | <0.001 |
| Blood Urea (mg/dl) | 96.05 ± 44.71 | 107.17 ± 64.1 | 0.90 | 0.37 |
| Serum Sodium (meq/l) | 135.67 ± 4.99 | 135.94 ± 4.88 | 0.21 | 0.83 |
| Serum Potassium (meq/l) | 4.16 ± 0.86 | 3.88 ± 0.65 | 1.32 | 0.19 |
| Total Protein (g/dl) | 6.23 ± 0.7 | 6.45 ± 0.75 | 1.22 | 0.23 |
| Serum Albumin(g/dl) | 2.95 ± 0.51 | 3.1 ± 0.68 | 1.09 | 0.28 |

In the present study, the most significantly affected biochemical parameter in CA-AKI as well as HA-AKI was serum creatinine (2.94 \pm 1.21 mg/dL vs 1.13 \pm 0.14 mg/dL; p<0.001) as compared to the other parameters.

TABLE 10: Creatinine and AKI Distribution

| | - | HA-AKI N = 18 | | P value |
|------------------------------|-------------|------------------|------|---------|
| S. Creatinine (At admission) | 2.94 ± 1.21 | 1.13 ± 0.14 | 6.32 | <0.001 |

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| S. Creatinine (After 48 hrs) | 2.95 ± 1.34 | 4.11 ± 2 44 | 2.93 | 0.004 |
|------------------------------|-------------|----------------|------|--------|
| S. Creatinine (At 7th day | 1.02 ± 0.28 | | 3.66 | <0.001 |
| from the day of admission) | | 0.63 | | |

In the present study, the serum creatinine values of HA-AKI and CA-AKI patients at admission was 1.13 ± 0.14 and 2.94 ± 1.21 respectively, in which the difference was found to be statistically significant; p<0.001. After 48 hours of admission, serum creatinine of CA-AKI was 2.95 \pm 1.34 and HA-AKI was 4.11 \pm 2.44, which was also found to be statistically significant; p=0.004. Even, the difference in serum creatinine values of patients with HA-AKI and CA-AKI on 7th day from the day of admission was statistically significant (CA-AKI = 1.02 \pm 0.28 and HA-AKI = 0.69 \pm 0.63; p<0.001).

Therefore, the difference in serum creatinine values at admission, after 48 hrs and on 7^{th} day from admission, in both, CA–AKI as well as HA-AKI patients was found to be statistically significant (p<0.001, p=0.004, p<0.001 respectively).

TABLE 11: KDIGO and AKI Distribution

| | CA-AKI | HA-AKI | x² value | P value |
|---------|------------|------------|----------|---------|
| KDIGO 1 | 32 (31.4%) | 3 (16.6%) | 1.61 | 0.45 |
| KDIGO 2 | 18 (17.6%) | 4 (22.2%) | | |
| KDIGO 3 | 52(50.9%) | 11 (61.1%) | | |
| Total | 102 (85%) | 18 (15%) | | |

In the present study, out of the 102 patients with CA-AKI, 52 (50.9%) were found to be in KDIGO stage 3; 32 (31.4%) in KDIGO stage 1 and 18 (17.6%) in KDIGO stage 2. Out of the 18 patients with HA-AKI, 11 (61.1%) were found to be in KDIGO stage 3; 4 (22.2%) in KDIGO stage 2 and 3 (16.6%) in KDIGO stage 1. Thus, the highest percentage of patients were in KDIGO stage 3, in both the groups of AKI.

It has been observed that there was insignificant association between stages of KDIGO and Type of AKI (p = 0.45)

TABLE 12: KDIGO STAGE AND ITS ETIOLOGICAL DISTRIBUTION

| Etiological | KDIGO-1 | KDIGO-2 | KDIGO-3 | Total |
|-----------------------------------|------------|------------|------------|---------------|
| Acute Diarrheal Disease | 18 (30.5%) | 13 (22%) | 28 (47.4%) | 59 (46.6%) |
| Snake Bite | 9 (36%) | 2 (8%) | 14 (56%) | (40.070) |
| | , , | , , | . , | |
| Sepsis | 2 (16.7%) | 3 (25%) | 7 (58.3%) | 12 (10%) |
| Tropical Acute Febrile Illness | 2 (16.7%) | 2 (16.7%) | 8 (66.6%) | 12 (6.6%) |
| Acute Glomerulonephritis | 2 (50%) | 1 (25%) | 1 (25%) | 4 (3.3%) |
| Ureteric calculus | 2 (66.7%) | 0 | 1 (33.3%) | 3 (2.5%) |
| Super vasmol Poisoning | 0 | 1 (50%) | 1 (50%) | 2 (1.6%) |
| Pigment Nephropathy | 0 | 0 | 2 (100%) | 2 (1.6%) |
| NSAID Induced | 0 | 0 | 1 (100%) | 1 (0.8%) |
| Total | 35 (29.1%) | 22 (18.3%) | 63 (52.5%) | 120 (100%) |
| x ² value | 12.68 | | | |
| P value | 0.696 | | | |

In the present study of 120 patients of AKI with the above etiologies, 35 (29.1%), 22 (18.3%) and 63 (52.5%) patients respectively were found in the KDIGO 1, KDIGO 2 and KDIGO 3 stages respectively. Thus, among the 3 stages of AKI by KDIGO criteria, maximum percentage of patients was in stage 3. It has been observed that there was insignificant association between stages of KDIGO and etiological profile (p = 0.696).

TABLE 13: MANAGEMENT OF AKI

| Renal Replacement Therapy | CA- AKI | HA-AKI | Total |
|------------------------------|------------|------------|------------|
| Conservative | 75 (69.6%) | 10 (55.5%) | 85 (70.8%) |
| Hemodialysis | 21 (20.5%) | 6 (33.3%) | 27 (22.5%) |

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| Peritoneal Dialysis | 6 (5.8%) | 2 (11.1%) | 8 (6.6%) |
|---------------------|-----------|-----------|------------|
| Total | 102 (85%) | 18 (15%) | 120 (100%) |
| x² value | 2.430 | | |
| P value | 0.297 | | |

In the present study, out of the 85 patients (70.8%) who were managed conservatively, 75 (69.6%) had CA-AKI and 10 (55.5%) had HA-AKI. From the 27 patients (22.5%) who required hemodialysis, 21 patients (20.5%) had CA-AKI and 6 patients (33.3%) had HA-AKI. Whereas, from the 8 patients (6.6%) in which peritoneal dialysis was done, 6 patients (5.8%) had CA-AKI and 2 patients (11.1%) had HA-AKI. Thus, it was found that compared to CA-AKI (26.3%), the need for dialysis was higher in patients with HA-AKI (44.4%). It has been observed that there was insignificant association between type of renal replacement therapy and type of AKI (p = 0.297).

TABLE 14: ETIOLOGY WISE MANAGEMENT DISTRIBUTION

| Etiological Renal Replacement Therap | | | ' |
|--------------------------------------|--------------|--------------|------------------------|
| | Conservative | Hemodialysis | Peritoneal Dialysis |
| Acute Diarrheal Disease | 46 (38.3%) | 9 (7.5%) | 4 (3.3%) |
| Snake Bite | 14 (11.6%) | 9 (7.5%) | 2 (1.6%) |
| Sepsis | 7 (5.8%) | 3 (2.7%) | 2 (1.6%) |
| Tropical Acute Febrile Illness | 10 (11.7%) | 2 (1.6%) | 0 |
| Acute Glomerulonephritis | 3 (2.5%) | 1 (0.8%) | 0 |
| Ureteric calculus | 2 (1.6%) | 1 (0.8%) | 0 |
| Super vasmol Poisoning | 2 (1.6%) | 0 | 0 |
| Pigment Nephropathy | 0 | 2 (1.6%) | 0 |
| NSAID Induced | 1 (0.8%) | 0 | 0 |
| Total | 85 (70.9%) | 27 (22.5%) | 8 (6.6%) |
| 2 value | 16.79 | | |
| P value | 0.399 | | |

In all, 35 (29.1%) patients required dialytic support, of which 27 (22.5%) were treated by hemodialysis and 8 (6.6%) by peritoneal dialysis. The remaining 85 patients (70.8%) were treated conservatively. It has been observed that there was insignificant association between type of renal replacement therapy and etiological profile (p = 0.399).

TABLE 15: OUTCOME WISE DISTRIBUTION

| Outcome | CA- AKI | HA-AKI | No of Patients |
|-------------------|------------|-----------|----------------|
| Complete Recovery | 94 (92.1%) | 9 (50%) | 103 (85.8%) |
| Partial Recovery | 3 (2.9%) | 2 (11.1%) | 5 (4.1%) |
| Mortality | 5 (4.9%) | 7 (38.8%) | 12 (10%) |
| Total | 102 (85%) | 18 (15%) | 120 |
| x² value | 23.292 | | |
| P value | <0.001 | | |

Regarding the outcome of AKI, 103 patients (85.8%) had complete recovery, of which, 94 (92.1%) had CA-AKI and 9 (50%) had HA-AKI. 5 patients (4.1%) had partial recovery. The overall in hospital mortality rate was 10%. Moreover, hospital acquired AKI has found significantly higher mortality (38.8%) then community acquired AKI (4.9%). It has been observed that there was significant association between outcome of patients and type of AKI (pc.0.001).

SUMMARY AND CONCLUSION

120 cases of acute kidney injury (classified by KDIGO criteria), who presented to Geetanjali Medical College and Hospital, Udaipur, from January 2017 to October 2018, were studied. These cases were further divided into CA-AKI and HA-AKI and studied regarding the demographic characteristics, clinical presentation, etiology and outcome.

• Out of the 120 patients of AKI, 102 (85%) patients were

classified as having CA-AKI and 18 (15%) patients as having HA-AKI. Thus, the frequency of CA-AKI was significantly higher as compared to HA-AKI. (p<0.001)

- The mean age of patients with CA-AKI was 51.54 ± 14.93 and HA-AKI was 53.22 ± 7.22 , which was statistically not significant. The maximum number of patients with CA-AKI were in the 6th and 7th decades of life whereas maximum patients with HA-AKI were in the 5th and 6th decades of life.
- Out of 120 patients of AKI, 70 patients (58.3%) were males, while 50 (41.6%) were females. The sex ratio was 1.4:1. Both, CA-AKI and HA-AKI had male predominance.
- The most common clinical feature was oliguria (83.3%) in HA-AKI and vomiting (59.8%) in patients with CA-AKI. Thus, we observed that there is significant association between clinical features and type of AKI i.e. there was change in the clinical feature with the type of AKI (p<0.001).
- Co-morbidities were present in 17.5% patients. We found that the commonest co-morbidity in patients with CA-AKI was HTN (5.8%) and in HA-AKI was DM (27.7%).
- We found that the commonest etiology in patients with HA-AKI was sepsis (66.6%) followed by acute glomerulonephritis (16.6%). In CA-AKI patients, the most common etiology was acute diarrheal disease (57.8%) followed by snake bite (24.5%). Thus, we observed that there was significant difference in etiological profile with type of AKI (p<0.001).
- We observed that the difference in serum creatinine values at admission, after 48 hrs and at 7th day from admission in both, CA–AKI as well as HA-AKI patients was found to be statistically significant (p < 0.001).
- The highest percentage of patients were in KDIGO stage 3, in both the groups of AKI (50.9% and 61.1% in CA-AKI and HA-AKI respectively).
- Maximum patients (70.8%) were managed conservatively while, 29.1% patients required dialytic support.
- We observed that the need for dialysis was higher in patients with HA-AKI (44.4%) as compared to CA-AKI (26.3%).
- We found that patients with CA-AKI had significantly higher rate of complete recovery than HA-AKI patients.
- We observed significantly higher mortality rates in patients with HA-AKI (38.8%) as compared to CA-AKI (4.9%). Thus, there was significant association between outcome of patients and type of AKI (p<0.001).

CONCLUSION

From the above study we concluded that the frequency of community acquired AKI was much higher as compared to hospital acquired AKI. Both community and hospital acquired AKI had male predominance. The clinical and epidemiological characteristics of community and hospital acquired AKI vary considerably. HA-AKI is common in ICUs, whereas CA-AKI is common in medical wards. Sepsis was the most common cause of HA-AKI, whereas, acute diarrheal disease and snake bite were frequent causes of CA-AKI. A major proportion of CA-AKI in the current study had preventable etiologies. Higher mortality was found in HA-AKI than CA-AKI. The underlying co-morbidities and vital organ dysfunction appears to be a major predictor of death.

Patients with CA-AKI have better short and long-term outcomes as compared to HA-AKI. The reason may be the difference in aetiology as most patients of CA-AKI had volume depletion which could be corrected easily.

AKI is a catastrophic disease and to deal with such a cataclysmic disease leading to high mortality and morbidity, frequent epidemiological studies from all parts of country are needed to devise the preventive and therapeutic strategies for this condition. Early recognition, early fluid resuscitation, effective anti-infective treatment, appropriate antidotes and timely referral of established AKI patients to centers with dialysis facilities can improve AKI outcome.

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