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Original Research Article

An Observational Cross Sectional Study on the Relationship between Estimated Creatinine Clearance and Acid Base Status in CKD Patients

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Abstract

Background: Chronic renal failure (CRF) is a prevalent condition that is typically detected later in its course. It is a condition that leads to end-stage renal disease and causes a long-term deterioration in renal function. Congenital and genetic abnormalities, renal artery disease, glomerular and tubular problems, and physical factors like stones, trauma, etc. are the main causes of CRF. Our goal was to identify the variables measuring acid-base balance in patients with chronic renal failure (CRF) and the correlation between those variables and creatinine clearance.

Methods: From January 2022 to December 2022, this cross-sectional study was carried out at Department of Medicine, Katihar Medical College and Hospital, Katihar, Bihar. This study included 220 CKD cases overall who visited the Department of Medicine during the study period.

Results: With the exception of H+ (r= -0.280) and Aniongap (r= -0.266), which have a negative correlation with creatinine clearance, all acid-base balance parameters in the current study had a positive connection with creatinine clearance (correlation coefficient r = 0.282 to 0.378). Only two of them, arterial sodium and potassium (r = 0.078 and 0.055 respectively; p values of 0.247 and 0.420 respectively), did not correlate with creatinine clearance.

Conclusion: In this work, we attempted to close the knowledge gap about the relationship between electrolyte and acid-base disorders and eCrCl.

Keywords: CKD, CRF, Creatinine clearance, GRF.

Introduction

Assessment of renal damage and tracking the development of renal disease are two clinical settings where estimation of renal function is crucial. [1]

A specialised capillary network makes up the high-pressure filtration system known as the glomerulus. It produces an ultrafiltrate that is devoid of cells and is composed primarily of plazama, with the exception of heavy protein molecules. The kidneys' capacity to excrete metabolic waste products from the blood into the urine is impacted by renal injury or changes in glomerular function.[1] For the regular care of patients, precise renal function measurement is essential. [2]

The rate (volume per unit of time) at which the glomerulus forms ultrafiltrate is known as the glomerular filtration rate (GFR). On average, 120 mL are produced every minute. Renal function is directly measured by the GFR. It decreases prior to the appearance of renal failure symptoms and is correlated with the degree of structural abnormalities in chronic renal illness. When the GFR drops to less than 10-15 mL/min, it is highly capable of predicting the uraemia symptoms and indications. Sadly, it is not the best index because it is challenging to directly measure and occasionally insensitive for diagnosing renal illness. [1]

Using inulin injection and kidney clearance as the gold standard, GFR can be measured.[3] Unfortunately, using inulin is a time-consuming, expensive, and intrusive process. As an alternative, the biochemical marker creatinine, which may be present in both serum and urine, is frequently employed to calculate GFR.[4] The amount of blood plasma that is cleared of creatinine per unit of time is known as creatinine clearance (CrCl). For determining renal function, it is a quick and economical approach. The comparison of the levels of creatinine in blood and urine can be used to calculate both CrCl and GFR. GFR can be predicted and is referred to as eGFR. An estimate of GFR is provided by creatinine clearance (Crcl). Yet, because creatinine is released by the proximal tube, Crcl is slightly greater than real GFR (in addition to being filtered by the glomerelus). GFR and eCrcl are nearly equal. The Cockroft-Gault formula yields eCrcl.

On the other hand, people with chronic renal disease frequently have metabolic acidosis (CKD). [5,6] Metabolic acidosis affects 15% of CKD patients to some extent.[7] As glomerular filtrate rate (GFR) dips below 40 ml/min/1.73m2, metabolic acidosis prevalence starts to develop and rises over time.[8] Many negative consequences of metabolic acidosis include bone loss, insulin resistance, muscular atrophy, and the advancement of CKD.[9] DeFronzo *et al* classic euglycemic clamp tests amply proved that insulin resistance in CKD is caused by poor skeletal muscle glucose uptake rather than by a malfunctioning liver's glucose uptake. Hence, static tests that assess insulin sensitivity in CKD patients cannot substitute for dynamic testing that measure muscle glucose disposal.

The main causes of increased nephrotic syndrome are progressive hyperperfusion and hyperfiltration.[10] Moreover, people with CRF receiving the most clinical care frequently have acid-base problems. Other studies in this cohort showed the incidence of acid-base problems and their relationship to the results, with a primary focus on chronic metabolic acidosis. [11]

Because the body is an open system, acid-base balance is dynamic and subject to change as a result of both internal and external variables, including cellular metabolites and external inputs like food and water. Nonetheless, the buffer system and some important organs, particularly the kidneys, work to quickly restore the acid-base equilibrium. [12]

The kidneys expel acid produced by metabolism and reabsorb bicarbonate to maintain acid-base balance. Bicarbonate glomerular filtration absorption is crucial for human health.[13] It should be noted that CRF is one of the significant causes of disturbances of the acid-base balance. A number of extracellular pH-dependent metabolites are impacted when disturbed. The acid-base balance system also makes an effort to achieve a normal pH in this line.

While the buffers function swiftly, they are hindered by kidney-related delayed responses that have a longer and more lasting effect. [14] If the primary cause of the acid-base balance issue is not addressed, it will

gradually worsen and result in permanent harm to the patients.

The relationship between eCrcl and metabolic acidosis in CKD has not yet been determined, despite its importance for understanding disease progression, managing acidosis, and connecting high anion gap metabolic acidosis (HAGMA) and non-anion gap metabolic acidosis (NAGMA). As a result, NAGMA is associated with hyperkalemia, which frequently denotes progressive renal failure from NAGMA to HAGMA.

Material and Methods

From January 2022 to December 2022, this cross-sectional study was carried out at Department of Medicine, Katihar Medical College and Hospital, Katihar, Bihar. This study included 220 CKD cases overall who visited the Department of Medicine during the study period. Total 220 CKD patients who were not undergoing treatment/dialysis and aged between 18-70 years who attended the Department of Medicine, during the study period were included in this study. Written informed consent was taken from the patients or relatives.

Chronic renal illness, recurrence, edema, anaemia, hypertension, proteinuria, urine casts, decreased creatinine clearance (below 60 mL/min), urea, blood creatinine, and elevated uric acid. Patients medical histories, clinical examinations, blood tests, including haematological, biochemical, and urine testing, were all measured at the start of the study.

The Statistical Package for Social Science (SPSS) version 21.0 was used to analyse all data. In terms of continuous variables, mean and standard deviation (+SD) were used. The t-test or one-way ANOVA were used to examine two or more continuous variables. Frequency and percentage were used to characterise categorical variables, and the chi-square test was used to assess two or more category variables. Scattered diagrams and the Pearson Correlation coefficient were employed, and regression analysis was conducted where necessary.

Results

Table 1: Age Distribution

Age Group	No. of cases	Percentage
18-30 years	36	16.4%
31-40 years	35	15.9%
41-50 years	24	10.9%
51-60 years	39	17.7%
61-70 years	86	39.1%
Total	220	100.0%
Mean Age	50.34 ±15.68	

The age distribution of the study participants is shown in Table 1. The participants in the current study ranged in age from 19 to 70. 39.1% of the participants, or the majority, were in the age range of 61 to 70. The participants' average age was 50.34±15.68 years.

Table 2: Sex Distribution

Sex	No. of cases	Percentage
Male	170	77.3%
Female	50	22.7%
Total	220	100.0

Table 2 displays the study participants gender distribution. In the current study, men made up the vast majority of participants (77.3%).

Table 3: Anthropometric Variables

Variables	No. of cases	±SD
Height	161.78	±5.59
Weight	59.24	±5.90
BMI	22.65	±1.65

Table 3 shows the mean values for anthropometric measurements like height, weight, and BMI. The average weight and BMI were 59.24±5.90 kg and 22.65±1.6 kg/m^2 respectively. The average height was 161.78±5.59 cm.

Table 4: Presenting Complaints

Presenting complaints	No. of cases	Percentage
Constitutional Symptom	165	75.0%
Bilateral lower limb Swelling	145	65.0%
Shortness of Breath	100	45.5%
Nausea / Vomiting	85	38.6%
Facial Puffiness	75	34.1%
Abdominal Distension	70	31.8%

The information about the complaints made by the study subjects is shown in Table 4. 75% (165) of the study participants had constitutional symptoms, whereas 65.9% (145) of the cases had bilateral lower limb edoema, and 45.5% (100) of the cases had shortness of breath. Abdominal distension (31.8%), face puffiness (34.1%), and nausea or vomiting (38.6%) were less frequent symptoms.

Table 5: Incidence of Co-morbidities

Co-morbidities	No. of cases	Percentage
Hypertension	110	50.0%
Diabetes	100	45.5%
None	75	34.1%

In terms of co-morbidities, we discovered that diabetes and HTN were present in nearly 50% of the research subjects, respectively (50% and 45.5%). Only 34.1% of research participants were co-morbidity-free. Table 5 provides information.

Table 6: Vital Parameters

Vital Parameters	Mean	±SD
DBP (mmHg)	92.67	±11.86
SBP (mmHg)	150.95	±23.64
Pulse Rate/ min	99.37	±16.93
SpO2 (%)	96.75	±2.32
Respiratory Rate /min	24.09	±2.45
Body Temperature (ºF)	97.8	±0.81

The average values for key parameters among the study participants are shown in Table 6. DBP and SBP levels were respectively 92.67±11.86 mmHg and 150.95± 23.64 mmHg on average. The

average heart rate was 99.37±16.93 beats per minute, and the average respiratory rate was 24.09±2.45 beats per minute.

Table 7: Fluid Intake and Output

Fluid Intake and Output	Mean	±SD
Input (ml/24 hr)	1677.27	±551.80
Output (ml/24 hr)	812.50	±575.45
Statistical Inference	p value = <0.01	

Data on fluid intake and urine output are shown in Table 7. Mean fluid consumption for 24 hours was 1677.27±551.80 ml, while average urine output was 812.50±575.45 ml. The 24-hour urine output average was significantly lower than the 24-hour fluid intake average, with a p value of less than 0.01.

Table 8: Complete Blood Count

Variables	Mean	±SD
Hb (gm/dl)	7.52	±1.34
MCV (fl)	88.87	±7.27
Platelet (10^3)	186.22	±86.98
TLC (10^3)	8.56	±2.52
Neutrophil (%)	72.57	±11.53
Eosinophil (%)	3.35	±1.90
Lymphocyte (%)	18.13	±8.71
Monocyte (%)	5.55	±3.59

Table 8 presents the data regarding the mean levels of variables of complete blood count.

Table 9: ECG Findings

Variables	No. of cases	Percentage
Normal Sinus Rhythm	220	100.0%
Tall T wave	40	18.2%
Other Findings	15	6.8%

Table 9 displays the ECG results among research participants. While large T waves were found in 18.2% of participants and additional abnormalities were observed in 6.8% of patients, all of the patients in the study had normal sinus rhythm.

Table 10: USG Findings

Variables	No. of cases	Percentage
Bilateral Small Kidney	190	86.4%
Other Findings	25	11.4%

The majority of the study participants had bilateral small kidneys, according to the USG findings, whereas 11.4% of patients reported different findings. Data are displayed in Table 10.

Table 11: Grading of CKD

Grading of CKD	No. of cases	Percentage
Stage IIIa	3	1.4%
Stage IIIb	5	2.3%
Stage IV	33	15.0%
Stage V	179	81.4%
Total	220	100.0%

The distribution of research participants by CKD grade is shown in Table 11. The bulk of the study patients (81.4%) fell into stage V of the CKD classification, 15% (33) into stage IV, and 8 (3.7%) into stage III.

Table 12: Comparison of Biochemical Parameters according to CKD staging

Variables	Stage IIIa (n=3)	Stage IIIB (n=5)	Stage IV (n= 33)	Stage V (n= 179)	p value
SGOT (U/L)	54.00±5.23	38.20±14.44	34.31±13.12	37.41±10.50	0.026
SGPT (U/L)	39.00±6.85	39.40±2.88	31.30±8.32	35.23±9.65	0.080
Blood Urea (mg/dl)	85.50±12.45	83.60±5.58	109.71±38.66	174.46±12.82	<0.0001
Serum Creatinine (mg/dl)	1.43±0.23	1.93±0.50	4.02±2.81	9.43±4.30	<0.0001
eCrCl (ml/min)	52.66±2.88	37.20±9.09	19.87±5.39	8.84±3.30	<0.0001
Spot ACR (mg/g)	306.68±6.28	267.79±74.12	380.59±382.75	294.47±114.85	0.090

According to the CKD staging, Table 12 compares various biochemical indicators of renal and hepatic function. In the present study we discovered all the variables of except SGPT and spot ACR exhibited significant difference according to CKD stage. Patients with CKD stage V had significantly higher mean levels of blood urea and serum creatinine than those with previous stages of the disease, although their mean levels of creatinine clearance were significantly lower.

Table 13: Diagnosis of Acid base Balance Disorders

Acid base balance disorders	No. of cases	Percentage
Normal Anion Gap Metabolic Acidosis (NAGMA)	31	14.1
High Anion Gap Metabolic Acidosis (HAGMA)	29	13.2
Respiratory Alkalosis	55	25.0
NAGMA + Respiratory Alkalosis	80	36.4
HAGMA + Respiratory Alkalosis	10	4.5
Respiratory Acidosis + Metabolic Alkalosis	5	2.3
No Acid Base Disorder	10	4.5
Total	220	100.0

There are four different types of acid-based disorders: metabolic acidosis, respiratory alkalosis, and respiratory acidosis. Regarding the diagnosis of an acid-base disorder, we discovered that respiratory alkalosis alone was the second most prevalent disorder, followed by metabolic acidosis with respiratory alkalosis, and that the incidence of HAGMA alone was also lower than NAGMA alone. In the current investigation, only 4.5% of individuals lacked an acid-base balance problem. Table 13 provides information.

Table: 14. Diagnosis of Acid base Balance Disorders according to CKD staging

Variables	Stage IIIa (n=3)	Stage IIIB (n=5)	Stage IV (n= 33)	Stage V (n= 179)
(NAGMA)	3	2	2	24
(HAGMA)	0	0	6	23
Respiratory Alkalosis	0	0	3	32
NAGMA + Respiratory Alkalosis	0	3	17	80
HAGMA + Respiratory Alkalosis	0	0	0	10
Respiratory Acidosis + Metabolic Alkalosis	0	0	4	1
No Acid Base Disorder	0	0	1	9

Acid-based disorders were tabulated in table number 14 based on CKD stage. The bulk of the study patients (81.4%) fell into stage V of the CKD classification, 15% (33) into stage IV, and 8 (3.7%) into stage III. There were no stage I or stage II patients in this study. The three stage IIIa patients exclusively have NAGMA. There are only 5 patients in stage IIIB, 2 of whom solely have NAGMA (40%) and 3 of whom also have respiratory alkalosis and NAGMA (60%) together. At stages IIIa and IIIB, there is no HAGMA. Out of the 33 stage IV patients, 17 have respiratory alkalosis and NAGMA (51.51%), 6 have respiratory acidosis and metabolic alkalosis (18.18%), 4 have respiratory acidosis and metabolic alkalosis (12.12%), 3 have respiratory alkalosis alone (9.09%), 2 have NAGMA alone (6.06%), and 1 has no acid-base disorder (3.03%). The majority of the 220 individuals with CKD (179 patients, 81.36%) are at stage V. Out of 179 stage V CKD patients, 80 have respiratory alkalosis with NAGMA+ (44.69%), 32 have respiratory alkalosis alone (17.8%), 24 have NAGMA alone (13.4%), 23 have HAGMA alone (12.84%), 10 have HAGMA+ respiratory alkalosis (5.58%), 9 have no acid base disorder (5.05%), and 1 has respiratory+ metabolic alkalosis (0.5%).

The majority of patients at all stages (80, 36.4%) exhibit NAGMA+ respiratory alkalosis. The only patients with your respiratory alkalosis were those in stages IV and V. HAGMA was only found in IV and V. Only in stages IV and V was there a lack of acid-based disorders.

Discussion

The participants in the current study ranged in age from 19 to 70. 39.1% of participants were between the ages of 61 and 70. The participants average age was 50.34±15.68 years (Table 1). Male participants made up 77.3% of the study participants (77.3%) (Table 2). The average weight was 59.24±5.90 kg, the average height was 161.78±5.59 cm, and the average BMI was 22.65±1.6 kg/m^2 (Table 3).

According to Chih-Cheng Hsu *et al*., renal failure can occur at any age. Due to the comorbidity of other diseases like diabetes and hypertension, it is more likely to affect the senior population [15,16]. According to the current survey, 39.1% of participants were between the ages of 61 and 70. The data from the study mentioned above agree with the findings from my study.

In their study, Pham Van T *et al*. look at individuals with chronic renal failure who have problems with acid-base balance and the association between its parameters and creatinine clearance. According to their study on demographic factors, 300 CRF patients between the ages of 50 and 81 were involved (180 men and 120 women). The patients average age was 51.95±17.03. The majority of patients (20.8%) were over the age of ≥71. The youngest and oldest patients were both

20 years old [17]. The youngest patient in my study was 20 years old, and the oldest was 74. So, the above study supports my study.

In my research, the average BMI was 22.65±1.6 kg/m^2. The majority of earlier research that looked at the link between BMI and risk of ESRD were carried out in Europe and North America, where the average BMI is greater than it is in Asian populations. Moreover, Asians have been found to have higher diabetes and cardiovascular disease risks at lower BMI cutoffs. According to a Chinese study, those who are underweight have a higher risk of developing ESRD than people who are in the normal weight range. Thus, research in Asian populations is required to examine the relationship between low range BMI and the risk of getting ESRD [18] My research so supports the Asian study.

75% (165) of the study participants had constitutional symptoms (such as fever, extreme fatigue, and bone or joint pain), whereas 65.9% (145) of the cases had bilateral lower limb edoema and 45.5% (100) had shortness of breath. Abdominal distension (31.8%), face puffiness (34.1%), and nausea or vomiting (38.6%) were less frequent symptoms (Table 4). A median of six symptoms were reported by 96% of individuals, with a 95% confidence interval of 93.2 to 98.0. The three most common complaints were excessive tiredness (81%; 76.0-85.6), sleep disturbance (70%; 64.3-75.3), and joint and bone discomfort (69%; 63.4-74.6). [19] As a result, my study and the prior study agree.

In my study, HTN and Diabetes were discovered in nearly half of the study participants (50% and 45.5%, respectively). Only 34.1% of research participants were co-morbidity-free (Table 5). Hypertension, diabetes, and hyperlipidemia were the three most prevalent comorbidities. The prevalence of hypertension in CKD patients with at least three comorbidities was close to 90%. [20]. Hence, my study is consistent with the earlier study.

SBP and DBP averages were 150.95 mmHg±23.64 and 92.67±11.86 mmHg, respectively (Table 6). 30% of the general adult population and up to 90% of those with CKD are affected by hypertension, which is defined by the European Society of Cardiology and the European Society of Hypertension (ESC/ESH) as a blood pressure (BP) of ≥140/80 mmHg or above. As 50% of the patients in my research had HTN, it cannot be compared to the previous study. The fact that NAGMA was 14.1% in my study could be the reason of this.

The average heart rate was 99.37±16.93 beats per minute (Table 6). According to testing for cardiovascular reflexes and heart rate variability, chronic kidney disease (CKD) is linked to elevated sympathetic tone and cardiac autonomic neuropathy [21,22].

The average respiration rate was 24.09±2.45/min (Table 6). Pulmonary edema and respiratory muscle dysfunction become more frequent as glomerular filtration rate decreases as a result of fluid retention and metabolic changes. Pulmonary hyperventilation is brought on by an increase in the patient's breathing rate, which results in increased oxygenation of the alveoli. As oxygen enters the alveoli, a diffusion process occurs. Diffusion between O_2 and CO_2 increases as more oxygen is introduced. This condition has an impact on increasing carbon in the blood so that it triggers changes in the metabolic state towards acidosis [23]. My study matches with the above study.

SpO_2 on average was 96.75±2.32%. According to CH Konge *et al*., mixed venous blood in the patients had an oxygen saturation of 53 +/- 8% compared to 79 +/- 2% in the control participants, indicating some degree of tissue hypoxia. The oxygen saturation of mixed venous blood in renal failure would be as low as 40% if oxygen uptake were not

decreased [24]. Due to the fact that I used arterial blood, my study data does not match the data shown above.

Mean fluid consumption for 24 hours was 1677.27±551.80 ml, while average urine output was 812.50±575.45 ml. When compared to 24-hour fluid intake, the average level of urine production was substantially lower (p value = <0.01) (Table 7). The urine production varies, ranging from oliguria to normal or even above normal amounts, even when the GFR is very low. These results are connected to the fact that the difference between the GFR and the rate of tubular reabsorption rather than only the GFR determines urine output. If, for example, a patient with advanced acute or chronic kidney failure has a GFR of 5 L/day (versus the normal of 140 to 180 L/day), the daily urine output will still be 1.5 L if only 3.5 L of the filtrate is reabsorbed [25]. My study matches with the above study.

In my investigation, every patient had a healthy sinus rhythm. 18.2% of participants had tall T waves, and 6.8% of patients had additional abnormalities like Q waves, tachycardia, LA and RA enlargement. 18.6% of patients had high potassium levels, and this discovery of a tall T wave is related to the patients' hyperkalemic condition (Table 9). Patients with CKD frequently experience abnormal electrocardiographic (ECG) results. However, there is conflicting evidence in the literature on how frequently ECG abnormalities occur in CKD patients, and there is little data on the local population [26]. The results of my investigation support those from the other studies.

According to USG findings, 86.4% of participants had B/L small kidneys, whereas 11.4% of patients had various conditions (ADPKD, Hydronephrosis, Renal Agenesis, Ectopic Kidney, etc). (Table 10). In a 1985 study by Päivänsalo M *et al*, they found that aberrant sonographic results were present in 67% of CKD cases [27]. The study mentioned above does not support my observation. In my study, 81.4% of the participants had ESRD (Stage V). This could be the reason why the data are different.

In my study, all of the subjects had normocytic, normochromic anaemia. Most of the metrics, including Platelet count, TLC, and DLC, were within normal bounds (Table 8, Figure 8). This is consistent with the results of the earlier research by (Bengre ML *et al* 2012) [28]. This may be because the majority of patients (81.4%) had advanced stage (Stage V) CKD.

In my study it was found that all the biochemical variables showed significant difference according to different CKD staging except SGPT and spot ACR. Mean level of SGPT (U/L) in stage IIIa is 39.00 ± 6.85, in stage IIIb is 39.40 ± 2.88, stage IV is 31.30 ± 8.32, stage V 32.23 ± 9.65 (p value 0.080). SGOT (U/L) in stage IIIa is 54.00 ± 5.23, in stage IIIb is 38.20 ± 14.44, stage IV is 34.31 ± 13.12, stage V 37.41 ± 10.50 (p value 0.026). Taking normal lab value of SGPT (7-56 U/L) and SGOT (5-40), the present study showed near normal range of SGOT in stage IIIb, IV, V and increased value in stage IIIa. Nevertheless, SGOT & SGPT values less than twice the range of normal are not significant for chronic liver disease. SGPT was within the near-normal range in all stages, with no discernible variation based on CKD staging. Patients with chronic kidney disease (CKD) frequently have serum aminotransferase levels that are close to the lower end of the normal value range [29].

In the present study it was observed that mean levels of blood urea (mg/dl) in stage IIIa is 85.50 ± 12.45, in stage IIIb is 83.60 ± 5.58, stage IV is 109.71 ± 38.68, stage V 174.46 ± 12.82 (p value < 0.0001). Serum Creatinine (mg/dl) in stage IIIa is 1.43 ± 0.23, in stage IIIb is 1.93 ± 0.50, stage IV is 4.02 ± 2.81,

stage V 9.43 ± 4.30 (p value <0.0001) (Table 12). As CKD progresses, blood urea and serum creatinine rise as a result. Salman Shafi *et al*. claim that as the stage of CKD advances, blood urea and serum creatinine rise. Therefore, my research backs up the earlier study.

The bulk of the study participants (81.4%) were classified as having stage V of the CKD, while 15% (33) were classified as having stage IV, and 8 (3.7%) were classified as having stage III (Table 11).

In this study no patients of stage I and II were found.

All 3 patients of stage IIIa have 100% NAGMA only.

Out of 5 patients of stage IIIB, 2(40%) patients have NAGMA only and 3(60%) patients have both NAGMA+ respiratory alkalosis.

No HAGMA in both stage IIIa and IIIB.

Of of the 33 stage IV patients, 17 (51.51%) have respiratory alkalosis plus NAGMA, six (18.18%) have HAGMA, four (12.12%) have respiratory acibosis plus metabolic alkalosis, three (9.09%) have respiratory alkalosis alone, two (6.06%) have NAGMA alone, and one (3.3%) has no acid-base problem.

Out of 220 patients CKD 179 (81.36%) patients have stage V.

Out of 179 patients with stage V CKD, 80 (44.69%) have respiratory alkalosis plus NAGMA, 32 (17.8%) have respiratory alkalosis alone, 24 (13.4%) have NAGMA alone, 23 (12.84%) have HAGMA alone, 10 (5.58%) have HAGMA+ respiratory alkalosis, nine (5.05%) have no acid-base disorder, and one (0.5%) has respiratory+ metabolic alkalosis. A total of 3 (100%) stage IIIa patients out of 220 patients had NAGMA, which was detected in 14.1% of patients.

NAGMA + HAGMA was found in 27.2 % patients.

100 (45.45 %) patients have NAGMA+ respiratory alkalosis (80% patients in stage V, 17% patients in stage IV, 3% in stage III).

Respiratory alkalosis alone was present only in 3 % patients of (stage IV, 8.57% and stage V, 91.42%).

HAGMA alone was present in 13.2% patients (Stage IV and stage V) only.

HAGMA + Resp. Alkalosis was present in 4.5% in satge V.

2.3% patients had Respiratory acidosis + Metabolic alkalosis. 4.5% patients had no acid base disorder (stage IV and V) (Table 14).

No patients had HAGMA + Metabolic alkalosis.

According to this study, 25% of individuals only had pulmonary alkalosis. Patients with 17.7% had NAGMA that has changed to HAGMA. 4.5% of CKD patients in advanced stages still do not have an acid-base imbalance.

Conclusion

The majority of CKD patients primarily experience constitutional symptoms, along with bilateral lower limb edoema and shortness of breath. In terms of co-morbidities, we discovered that diabetes and HTN were present in roughly two-thirds of the study participants, while the remaining one-third were co-morbidity-free. Acid-base balance parameters and creatinine clearance in the current study have a positive link (correlation coefficient r = 0.282 to 0.378). Only two of them, arterial sodium and potassium (r = 0.078 and 0.055 respectively; p values of 0.247 and 0.420 respectively), did not correlate with creatinine clearance. The stage of renal failure can be predicted by creatinine clearance.

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