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RESEARCH ARTICLE

ASSESSMENT OF ACID BASE AND ELECTROLYTE STATUS IN PATIENTS WITH CHRONIC KIDNEY DISEASE

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ABSTRACT

Background: Chronic kidney disease (CKD) encompasses a spectrum of pathophysiologic processes associated with abnormal kidney function. The risk of worsening CKD is related with the GFR and the amount of albuminuria. Acid-base and electrolyte homeostasis is vital for proper functioning of numerous metabolic processes and organ functions and kidneys play a critical role in the maintenance and regulation of this homeostasis. Kidney diseases and dysfunction (chronic kidney disease, CKD) results in alterations of electrolyte and acid-base balances. Therefore, we aimed to assessment of Acid Base and Electrolyte Status in Patients with Chronic Kidney Disease for an early detection of CKD and to identify the factors associated with it. ***Methods:*** A Prospective observational study was conducted from Aug 2022 to July 2023 among all CKD patient of different stages and different etiology admitted in medicine and nephrology department of MBGH and associated hospital of R.N.T. Medical college Udaipur, Rajasthan. For albuminuria urine ACR was used. For acid base status (H+,PH, HCO) arterial blood gas analysis (ABG) was done as per standard protocol. Venous samples were taken for analysis of H, PH, HCO3 Na, K, CI. Ca, Mg, and Phosphorus. Urine sample sent for urinary cast and complete urine analysis. All data statistically analysed by SPSS version 17. P value <0.05 were considered as statistically significant. ***Results:*** This study found acidosis in 76.7% patients, in which Maximum 48.2% belongs to stage G5 followed by stage 4,stage G3b, 12.5% in each, 3.5% belongs to stage G3a. In acidosis Hypertension and diabetes was most responsible factor in 28 patient either independent or with combined effect. 5.3% were due to obstructive uropathy, 3.5% each were due to autoimmune and PCKD. Infectious cause were also associated with 3.5%. Hyperkalemia was found in 37.5% while 5.3% were hypokalemic. In hyperkalemia group maximum 25% patient was in stage G5 followed by G4(5.3%), G3B (5.3) in equal then in stage G3a(1.7%). In hypokalemia 3.5% belongs to stage G3b and 1.7% in stage G3a. in study hyponatremia was found in 19.4% patient and hypernatremia in 5.3%. Hypermagnesemia found in 25% patients and hypomagnesaemia in 5.3%. ***Conclusions:*** It is imperative to monitor hypertension, metabolic disorders (DM,etc.) and serum electrolytes concentration in renal dysfunction patients to slow the progression of CKD to end-stage renal disease (ESRD) and other serious complications because maximum patient in our study were in stage G5 and mostly caused by either hypertension or diabetes or both. Metabolic acidosis, hyperkalemia, hyponatremia, hypomagnesemia, hyperchloremia, hypocalcemia and hyperphosphatemia were the most common electrolytes imbalance in CKD.

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INTRODUCTION

The kidneys are functioning to maintain the electrolyte and water balance in the tissue fluids of the body, necessary for survival. As a begin in the tubular structures may present primarily with electrolyte disorders or disorders of dilution and concentration of the urine. In both glomerular and tubular disorders, upon progressing to chronic disease, the distinction is more difficult because glomerular diseases eventually affect the tubular interstitium and tubular diseases progress to glomerular dysfunction and scarring.[1]Acute kidney injury (AKI) is defined by the impairment of kidney filtration and excretory function

over days to weeks (generally within 7 days), resulting in the retention of nitrogenous and other waste products normally cleared by the kidneys. AKI ranges from asymptomatic and transient changes in laboratory parameters of glomerular filtration rate (GFR), to rapidly fatal derangements in the ability of the kidney to maintain effective circulating volume regulation, excrete nitrogenous wastes, metabolic toxins, and maintain electrolyte and acid-base composition of the plasma.[2] Chronic kidney disease (CKD) encompasses pathophysiologic processes associated with deranged kidney function, often with a progressive decline in glomerular filtration rate (GFR). The risk of worsening CKD is closely linked to both the GFR and the amount of albuminuria. The term end stage renal disease represents a stage of CKD where the accumulation of toxins, fluid, and electrolytes normally excreted by the kidneys leads to death unless the toxins are removed by renal replacement therapy, using dialysis or kidney transplantation.[3]There has been a wealth of published data highlighting the risk of adverse consequences and outcomes in people with albumin excretion rate (AER) 430 mg/24 hours and/or glomerular filtration rate (GFR) <60 ml/min/1.73 m2 (GFR categories G3a-G5), irrespective of the etiology or duration of reduced kidney function. Description of the relationship between GFR, albuminuria and prognosis has significantly improved the understanding of CKD in multiple populations. Widespread use of albumin- ratio (ACR) and reagent strip urine testing to detect elevated albuminuria together with reporting of estimated GFR (eGFR) has led to easier identification of people with CKD. Acid-base and electrolyte homeostasis is important for proper functioning of metabolic processes and organ functions in the human body. Kidneys play a main role in the maintenance and regulation of this homeostasis.

Kidney diseases and dysfunction (chronic kidney disease, CKD) compromise the regulatory functions, resulting in electrolyte and acid-base imbalances that can be life-threatening. Metabolic acidosis can be broadly classified into (1) high anion gap (AG) acidosis and (2) normal AG acidosis. The ionic environment of the blood is neutral with the addition of cations always equal to the sum of anions. The cations (Na + and K +) exceed the total anions (Cl – and HCO 3 –) resulting in an artificial AG. Serum albumin is the major contributor to the gap. The prevalence of metabolic acidosis increases with progression of CKD Normal AG acidosis is predominant in early stages of CKD; high AG acidosis occurs in Late stages (GFR <30 mL/min/1.73 m 2) due to retention of anions such as sulfate, phosphate, and urate. Although less common than metabolic acidosis, metabolic alkalosis can occur in patients with CKD. CKD patients are commonly on diuretics as well as calcium carbonate or citrate which can result in hypokalemia and alkalosis. Hyperkalemia is the most common electrolyte disorders. Dysnatremia occurs in CKD due to compromised renal water regulation. Hyperkalemia prevalence increases as CKD Decreased glomerular filtration and ability of tubular K + secretion, often in combination with a diet generous in K +, are the major cause of hyperkalemia. Other causes of hyperkalemia pertinent to CKD patients are (1) medications that further reduce the already limited capacity of distal nephron K + excretion such as renin angiotensin aldosterone system inhibitors, K + -sparing diuretics and calciurene inhibitors, (2) transcellular K + shift due to insulin deficiency, mineral metabolic acidosis, and tissue breakdown (hemolysis, rhabdomyolysis, tumor lysis syndrome), and (3) hyporeninemic hypoaldosteronism (type IV) RTA. Hypernatremia (serum [Na +] >145 mEq/L) is relatively common with a reported incidence of 1–3.4% in hospitalized patients.

Hypernatremia relates total body water deficiency relative to total body sodium. Common manifestations include intense thirst, fatigue, and lethargy, muscle weakness, slowing of mentation, confusion, and coma. Hypernatremia is an increased osmolality state. Thus, measurement of serum osmolality is, in general, not required. Measurement of urine osmolality is important in differentiating renal water loss such as diabetes insipidus (inappropriately dilute urine) from extra renal water loss (concentrated urine).Magnesium (Mg 2+) is the 2nd most important intracellular cation with more than 99% located intracellularly (53% in bones, 46.5% in soft tissues) and less than 0.5% located extracellularly. In early stages of CKD, decreased filtration of Mg 2+ is balanced by reduced renal tubular reabsorption; hence, dysmagnesemia is uncommon. In advanced CKD, hypermagnesemia can be enhanced by Mg 2+ -rich diet and Mg 2+ -containing medications.

AIM AND OBJECTIVE

- To study acid base analysis in different stages of chronic kidney disease (CKD).
- To study serum electrolytes (K^+, Na^+, Cl^-, Mg^{+2}, $Ca^{+2}$and phosphorus) indifferent stages of chronic kidney disease (CKD).

MATERIAL AND METHODS

STUDY DESIGN: Prospective observational study.

STUDY POPULATION: All CKD patient (irrespective of sex) of different stages and different etiology (before hemodialysis or peritoneal dialysis) admitted in various wards of medicine department and nephrology department of MBGH and associated hospital R.N.T. Medical college Udaipur, Rajasthan will be enrolled in the study after taking informed consent.

STUDY PERIOD: June 2022-june 2023 after approval of ethical committee.

Inclusion criteria*:* CKD Patients from 15 to 70 year age.

Exclusion criteria

- Patient below 15 year and above 70 years age.
- Patients with Renal carcinoma.
- Renal transplant patient.
- Patient on dialysis.

Study method: Study will be conducted in all CKD patient (irrespective of sex) of different stage and different etiology (before hemodialysis is or peritoneal dialysis) admitted in various wards of medicine department and nephrology department of MBGH and attached hospitals of RNT medical college Udaipur Rajasthan from June 2022 to June 2023 after approval of ethical committee .Those full filling the inclusion and exclusion criteria will be enrolled in Study after taking written informed consent. Detailed history which includes duration of disease history of hemodialysis or peritoneal dialysis, and detailed drug history as well history of co-morbidities like hypertension, IHD, diabetes mellitus, autoimmune disease, obstructive uropathy etc. will be taken. then General physical examination and systemic examination will be done. CKD will be defined as per recent KDIGO (Kidney Disease Improving Global Outcomes) guideline and staging of CKD will be done with same guideline which is validated guideline internationally.[4]

STAGING OF CKD

CKD staging will be done based on GFR category.

All-relevant investigation which include routine testing's like CBC, RFT, LFT, blood sugar, lipid profile, ECG, X-RAY, USG and then specific investigation. Venous samples will be taken for analysis of Serum electrolytes. The sampling parameters included in the study will be H^+, PH, HCO^{-3} Na^+, K^+, Cl^-. Ca^{+2}, and Mg^{+2}, Urine sample will be send for urinary cast and complete urine analysis.

Table 1. Mean Age and SD

	No. of patients	Mean age ±S.D.
Total	56	54.6 ±12.71
Male	42	54.5 ±12.56
Female	14	55 ±13.13

We enrolled 56 patient of during study period out of 56, 42 patient (75%) were male while 14(25%) patient were female.

Table 2. Patient distribution according to CKD stages

Stage	Male	Female	Total
G1 (≥90)	00	00	00
G2 (60-89)	01	00	01
G3a (45-59)	03	00	03
G3b (30-44)	06	05	11
G4 (15-29)	08	00	08
G5 (<15)	24	09	33
TOTAL	42	14	56

We categorized serum sodium in different stages and found hyponatremia in 10 (19.4%) patient out of study population and hypernatremia in 3 patient (5.3%) while rest patient were in normal sodium ranges. in hyponatremia group 5 patient(8.9%) were in stage G5,4 patient(7.1%) were in stage G3B,2 patient(3.5%) in stage G4 and one patient belongs to stage G1(1.7%). We found hyperkalemia in 21 patient, hypokalemia in 3 patient and 32 patient was with normal potassium level in hyperkalemia group maximum patient (14=25%)was in stage G5 followed by G4(5.3%), G3B (5.3)in equal then in stage G3a(1.7%).in hypokalemia 2 patient belongs to stage G3b(3.5%) and one patient (1.7%)in stage G3a. While calculating serum magnesium imbalance in different stages of CKD we found 14 patient (25%)in hypermagnesemia group,3 patient(5.3%) in hypomagnesaemia group while maximum 39 patient were in normal range of magnesium. In hypermagnesemia group maximum patient were in G5 stage (12=21.4%) follower by G4 and G3a stage one patient(1.7%) each. In hypomagnesemia group early ckd stages were involved as stage G3b,G3a,and G2 one patient(1.7%) in each. After calculating serum calcium level in different stages we found that majority of patient (31=55.3%) were hypercalcemic. 3 patient (5.3%)had calcium above normal value and 22 patient had calcium value in normal range .In hypocalcemic group we found that most patient were in advanced stages of CKD. As 23 in G5(41%), 4 IN G4(7.1%)3 IN G3B(5.3%) and only one patient(1.7%) was belongs to G2 stage. High calcium were found in one patient each in stage G4, G3b,G3a.

Assessment of chloride level suggestive that about 50 % cases have high chloride value while 10 patients were in low chloride group and remaining 18 patients found with normal serum chloride value. In hyperchloremic arm maximum 17 patient belonging to stage G5 followed by 7 patient in G4 and 2 patient each in G3A,and G3B GROUP. Out of 10 hypochloremic patient 6 patients were in stage G5 and 4 patients were in stage 4. Phosphorus estimate suggestive of almost all patient 52 out of 56 have high phosphorus no patient was found with low phosphorus rest 4 patient were in normal phosphorus range. Majority of high phosphorus group belongs to stage G5 (33) followed by G4(8),G3B(8),G3A(2) and G2(1). Out of 56 case 43 patient (76.7%) were in acidosis on admission only 1 patient were found alkali and rest 12 patient have PH in normal range .out of 43 acidosis patient maximum 27 belongs to stage G5 followed by stage 4, stage G3b 7 patient in each 2 patient belongs to stage G3a only 1 patient who found with alkalosis is in stage 5.

DISCUSSION

CKD, with its high prevalence, morbidity and mortality, is an important public health problem. With 3% of land mass, India hosts 17% of the Earth's population. Large numbers of patients below the poverty line, low gross domestic product, and low monetary allocations or health care have led to suboptimal outcomes. Moreover, CKD and other non-communicable diseases have often been ignored in the face of persistent challenges from and competition for resources for communicable diseases. On a typical diet, an adult generates approximately 0.8–1 mEq/kg body weight of nonvolatile acid [1] and 15,000 mEq of CO 2 (volatile acid) daily. Depending on the pCO 2 , a small fraction of CO 2 is dissolved in body fluids as carbonic acid (H 2 CO 3), a weak acid, while a large amount of CO 2 is eliminated through respiration. Non-volatile acids are buffered in the body to prevent acute systemic pH perturbations. HCO 3 – /H 2 CO 3 is the major buffer system which neutralizes nonvolatile acids at the cost of HCO 3.[5]The prevalence of metabolic acidosis increases with progression of CKD. Normal high AG acidosis is predominant in early stages of CKD; AG acidosis occurs in late stages (GFR <30 mL/min/1.73 m2) due to retention of anions such as sulfate, phosphate, and urate. It should be noted that net endogenous acid production is relatively unchanged in CKD[6]. In our study Out of 56 case 43 patient (76.7%) were in acidosis on admission maximum 27 patient (48.2%)belongs to stage G5 followed by stage 4, stage G3b 7 patient in each(12.5%), 2 patient (3.5%)belongs to stage G3a. In a cross-sectional analysis of the baseline data from the Chronic Renal Insufficiency Cohort (CRIC) study involving 3,900 patients in CKD stages 2–4, the prevalence of metabolic acidosis (serum HCO3 – <22 mEq/L) was 7% for CKD stage 2, 13% for CKD stage 3 and 33% for CKD 4 with an overall acidosis occurrence of 17.3%[7]. In our study maximum patient was in stage G5 so overall metabolic acidosis rate is so high. Although less common than metabolic acidosis, metabolic alkalosis can occur in patients with CKD. CKD patients are commonly on diuretics as well as calcium carbonate or citrate which can cause hypokalemia and alkalosis.

In our study only 1 patient out of 56 (1.7%) was present in alkalosis range of PH. Electrolyte disorders are common in CKD. Hyperkalemia among the most common electrolyte disorders. in our study hyperkalemia was found in 21 patient(37.5%) while 3 patient(5.3%) were hypokalemic. in hyperkalemia group maximum patient(14=25%)was in stage G5 followed by G4(5.3%), G3B (5.3)in equal then in stage G3a(1.7%).in hypokalemia 2 patient belongs to stage G3b(3.5%) and one patient (1.7%) in stage G3a.there is strong relationship of high serum potassium level as ckd progressed to advanced stage diabetes and hypertension were leading etiology responsible for hyperkalemia .Aysun Aybal Kutlugun *et al*l in their study prevalence of hyperkalemia was 33.9% in study population.[8]Hypokalemia (serum [K +] <3.5 mEq/L) is less common in CKD than hyperkalemia. It can, however, occur due to a multitude of reasons including non-K + -sparing diuretic use, alkalosis, hypomagnesemia, vomiting, and diarrhea. In a study of patients (*n* = 2,500) with CKD stages 1–4 (mean eGFR of 40.6 mL/min/1.73m 2), those with hypokalemia (serum K + <3.5 mEq/L) had a significantly higher risk of developing ESRD than the risk in patients with serum K + of 4.5–5 mEq/L,{11} Serum [Na +] represents water balance and is the primary determinant of serum osmolality. Changes in serum osmolality drive fluid in and out of cells and affect cell volume and function.In our study found we found hyponatremia in 10 (19.4%) patient out of study population and hypernatremia in 3 patient (5.3%)while rest patient were in normal sodium ranges.in hyponatremia group 5 patient(8.9%) were in stage G5,4 patient(7.1%) were in stage G3B,2 patient(3.5%) in stage G4 and one patient belongs to stage G1(1.7%) In a retrospective study involving a cohort of veterans (*n* = 655,000) with non-dialysis-dependent et CKD, Kovesdy al. [35] found a U-shaped association between serum [Na +] and mortality with both hypernatremia (Na + >145 mEq/L) and hyponatremia (Na + <136 mEq/L) associated with increased mortality. In the same study noted above, veterans with CKD (mean eGFR of 52 mL/min/1.73 m 2) were followed for a median period of 5.5 years, and 26% of the subjects developed at least 1 episode of hyponatremia. In CKD, as cited above, there is a reported 2% (*n* = 13,289) prevalence of hypernatremia and 7% (*n* = 45,666) occurrence of at least 1 episode of hypernatremia.[9]Magnesium (Mg 2+) is the second most abundant intracellular cation with more than 99% located intracellularly (53% in bones, 46.5% in soft tissues) and less than 0.5% located extracellularly. About 20–30% of circulating Mg 2+ is protein bound (mainly to albumin), while 70–80% is freely filtered by kidneys. In our study we found 14 patient(25%) in hypermagnesemia group,3 patient(5.3%) in hypomagnesaemia group .In hypermagnesium group maximum patient were in G5 stage(12=21.4%) follower by G4 and G3a stage one patient (1.7%)each. In hypomagnesemia group early ckd stages were involved as stage G3b,G3a,and G2 one patient(1.7) in each. Both hypermagnesemia (>2.3 mg/dL) and hypomagnesemia (<1.7 mg/dL) are relatively common with reported prevalence of 31 and 20%, respectively, in hospitalized patients.

Table 3. Sodium Imbalance In Different Stages Of Ckd

Stage	SODIUM									
	Hyponatremia (<136)			Normal(136-146)			Hypernatremia(>146)			Total
	M	F	Total	M	F	Total	M	F	Total	
G1	00	00	00	00	00	00	00	00	00	00
G2	00	00	00	00	00	00	00	00	00	00
G3a	01	00	01	01	00	01	00	00	00	01
G3b	03	01	04	02	02	04	00	01	01	1
G4	02	00	02	05	00	05	00	00	00	08
G5	04	01	05	09	04	13	02	00	02	33
TOTAL	10	02	12	17	06	23	02	01	03	56

Table 4. potassium Imbalance In Different Stages Of Ckd

Stage	POTASSIUM									
	Hypokalemia (<3.5)			Normal(3.5-5.0)			Hyperkalemia(>5.0)			TOTAL
	M	F	Total	M	F	Total	M	F	Total	
G1	00	00	00	00	00	00	00	00	00	00
G2	01	00	01	00	00	00	00	00	00	01
G3a	00	00	00	02	00	02	01	00	01	03
G3b	01	01	02	04	02	06	02	01	03	11
G4	00	00	00	05	00	05	03	00	03	08
G5	00	00	00	14	05	19	10	04	14	33
TOTAL	02	01	03	26	06	32	16	05	21	56

Table 5. Magnesium imbalance in different stages of ckd

stage	MAGNISIUM									
	Hypomagnesaemia (<1.5mg/dl)			Normal (1.5-2.3mg/dl)			Hypermagnesemia >2.3mg/dl)			TOTAL
	M	F	Total	M	F	Total	M	F	Total	
G1	00	00	00	00	00	00	00	00	00	00
G2	01	00	01	00	00	00	00	00	00	01
G3a	01	00	01	02	00	00	00	00	00	03
G3b	00	01	01	09	02	06	00	01	01	11
G4	00	00	00	07	00	04	01	00	01	08
G5	00	00	00	15	04	23	06	06	12	33
TOTAL	02	01	03	33	06	39	07	07	14	56

Table 6. Calcium imbalance in different stages of ckd

stage	CALCIUM									
	Hypocalcemia (<8.7)			Normal (8.7-10.2)			Hypercalcemia (>10.2)			TOTAL
	M	F	Total	M	F	Total	M	F	Total	
G1	00	00	00	00	00	00	00	00	00	00
G2	01	00	01	00	00	00	00	00	00	01
G3a	00	00	00	02	00	02	01	00	01	03
G3b	01	02	03	04	03	07	01	00	01	11
G4	04	00	04	03	00	03	01	00	01	08
G5	16	07	23	08	02	10	00	00	00	33
TOTAL	22	09	31	17	05	22	03	00	03	56

Table 7. Chloride level in different stages of ckd

stage	CHLORIDE									
	Hypochloremia (<102)			Normal(102-109)			Hyperchloremia (>109)			TOTAL
	M	F	Total	M	F	Total	M	F	Total	
G1	00	00	00	00	00	00	00	00	00	00
G2	00	00	00	01	00	01	00	00	00	01
G3a	00	00	00	01	00	01	02	00	02	03
G3b	03	01	04	01	00	01	02	00	02	11
G4	00	00	00	05	00	05	03	04	07	08

Serum magnesium concentrations are dependent on appropriate renal function. Hence, in CKD patients, both hypo- and hypermagnesemia are fairly common. Hypermagnesemia is commonly encountered in end-stage (Stage 4 and 5) renal disease because of extremely low GFR. On the other hand, hypomagnesemia is also often observed because of medications (diuretics, PO_4 binders, antacids, and proton-pump inhibitors) and volume expansion.

Kotha NB, Serum magnesium levels in chronic kidney disease patients found that Serum magnesium was 2.02 ± 0.36 mg/dL in cases and 2.01 ± 0.17 mg/dL in controls. The overall distribution showed a trend of hypomagnesemia in CKD patients but it was not statistically significant (**p**= 0.877). The hypomagnesemia was more on the lower side of normal rather than frank hypomagnesemia. In our data, there was 21.6% of frank hypomagnesemia and 10.8% of frank

Table 8. Phosphorus level in different stages of CKD

stage	PHOSPHORUS									
	Hypophosphatemia (<2.5)			Normal(2.5-4.3)			Hyperphosphatemia (>4.3)			TOTAL
	M	F	Total	M	F	Total	M	F	Total	
G1	00	00	00	00	00	00	00	00	00	00
G2	00	00	00	00	00	00	01	00	01	01
G3a	00	00	00	01	00	01	02	00	02	03
G3b	00	00	00	01	02	03	05	03	08	11
G4	00	00	00	00	00	00	08	00	08	08
G5	00	00	00	00	00	00	24	09	33	33
TOTAL	00	00	00	02	02	04	40	12	52	56

Table 9. Acid base status in different stages of ckd

stage	PH									
	Acidosis (<7.35)			Normal(7.35-7.45)			Alkalosis(>7.45)			TOTAL
	M	F	Total	M	F	Total	M	F	Total	
G1	00	00	00	00	00	00	00	00	00	00
G2	00	00	00	01	00	01	00	00	00	01
G3a	02	00	02	01	00	01	00	00	00	03
G3b	05	02	07	01	03	04	00	00	00	11
G4	07	00	07	01	00	01	00	00	00	08
G5	18	09	27	05	00	05	01	00	01	33
TOTAL	32	11	43	09	03	12	01	00	01	56

hypermagnesemia.[10]In our study serum calcium level in different stages we found that majority of patient (31=55.3%) were hypocalcemic, 3 patient (5.3%)had calcium above normal value and 22 patient had calcium value in normal range . In hypocalcemic group we found that most patient were in advanced stages of CKD. As 23 in G5 (41%), 4 IN G4(7.0%), 3 IN G3B (5.3%)and only one patient (1.7%)was belongs to G2 stage. High calcium were found in one patient(1.7%) each in stage G4, G3b,G3a. In the study of sanjay vikrant *et al*l 462 patients of CKD Stage 3–5D were studied. The frequency of various biochemical abnormalities was hypocalcemia (23.8%), hypercalcemia (5.4%), these results are almost comparable with our study results.[11]Hypocalcemia in chronic renal failure is due to two primary causes - increased serum phosphorus and decreased renal production of 1,25 (OH)2 vitamin D. The former causes hypocalcemia by complexing with serum calcium and depositing it into bone and other tissues. The latter causes hypocalcemia by decreasing the GI absorption of calcium. Serum chloride is a frequently neglected laboratory value typically only considered during states of metabolic acidosis. However, it has become increasingly clear that chloride is an important driver of numerous homeostatic mechanisms including regulation of renin secretion, tubuloglomerular feedback, blood pressure response, and renal sodium handling.In our study about 50 % cases have high chloride value while 10 patient were in low chloride group and remaining 18 patient found with normal serum chloride value In hyperchloremic arm maximum 17 patient belonging to stage g5 followed by 7 patient in g4 and 2 patient each in G3a,and g3B GROUP. Out of 10 hypochloremic patient 6 patients were in stage G5 and 4 patient were in stage 4.almost no study were carried out for accessing direct relationship between serum chloride level and ckd. Minesh khatri *et al* in a cohort study mention men and women with CKD, they demonstrated that higher serum chloride is significantly associated with a modest decrease in eGFR independent of other well-established CKD risk factors.[12]

CONCLUSION

During our study period from July 2022 to July 2023 we enrolled total 56 patient of CKD before hemodialysis. All patient categorized in different stages of creatinine based on eGFR and also categorized according to etiology which was most responsible in development of CKD metabolic acidosis, hyperkalemia, hyponatremia, hypermagnesemia, hyperchloremia, hypocalcemia and hyperphosphatemia were the most common electrolytes imbalance in ckd .and almost all progressed with advancing stages of ckd.

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