Ministry of Higher Education

and Scientific Research

University of Diyala

College of Medicine

Cations levels in patients with kidney dysfunction

A study submitted to the Council of the College of Medicine, Diyala University, In Partial Fulfillment of Requirements for the Bachelor Degree in medicine and general surgery.

Submitted by

Batool Saad

Supervised by

Dr. Salih Mahdi Salman

2022-2023

Supervisor Recommendation

I certify that the research article entitle (**Cations levels in patients with kidney dysfunction**) was performed by the 6th class student (**Batool Saad**) at the faculty of Medicine, University of Diyala, and Baqubah Teaching Hospital under my supervision.. The student has finished required revisions and the article is ready for debate.

Prof. Dr. Salih Mahdi Salman

26th March 2023

Abstract

Objectives: To discuss the cations levels in kidney impairment among the patients attending Baquba teaching hospital.

Methods: This is a cross sectional study. It was conducted in the period from October 2022 to January 2023. We collected a sample of 40 patients who attend Baquba teaching hospital. Every patient with kidney impairment was eligible for study and we excluded the patients who were older. We collected information about age, gender, the electrolyte levels (Na, K and Ca).

Results: 40 patients were enrolled in this study. Their mean age was 40-47 years, 50% of them were males and 50% were female, the sodium level was abnormal in 47.5% of the patients, 37.5% of them had abnormal potassium levels and 77.5% of them had abnormal calcium levels.

Conclusion: Cation levels are affected by renal impairment and could have catastrophic effect on the heart, bone and blood pressure.

Keyword: Cations, kidney dysfunction, hypernatremia, hyponatremia, hyperkalemia, hypokalemia, hypercalcemia, hypocalcemia.

Introduction

The primary function of kidneys is to regulate fluid and electrolyte balance by adjusting urine volume and the excretion of most electrolytes. In the average adult, healthy kidneys reabsorb 99% of the plasma filtrate, producing around 1.5 to 1.8 L of urine per day and filters the total volume many times each day. In addition, under the influence of antidiuretic hormone, aldosterone, and other hormones, selective reabsorption and secretion of water and electrolytes in the renal tubules result in urine that is greatly different in composition and concentration from plasma. This process assists in the maintenance of normal plasma osmolality, electrolyte balance, blood volume, and acid–base balance [1,2].

The nephron is the functional unit of the kidney and each kidney contains approximately 1.2 million nephrons. The kidneys have three different types of nephrons1: superficial cortical nephrons (85% of all nephrons), which extend partially into the medulla; midcortical nephrons with short or long loops; and juxtamedullary nephrons3 (about 12% of nephrons). Each nephron is composed of the glomerulus, Bowman's capsule, and a tubular system [3].

Disturbances in sodium and water balance are frequently encountered abnormalities. The body contains approximately 60% water content that is divided into extracellular fluid and intracellular fluid. Sodium is the major cation in the extracellular fluid, and potassium is the major cation in the intracellular fluid. Free movement of water ensures the sodium concentration in the extracellular compartment is equal to the potassium in the intracellular compartment. The difference in cationic solute composition between extracellular and intracellular fluid is maintained by a pump– leak mechanism maintaining osmotic equilibrium [4].

Sodium imbalance occurs when there is expansion of the extracellular fluid volume. Sodium intake is greater than water intake with sodium retention. Urinary sodium retention is a cardinal manifestation of primary glomerular disease. Causative factors include a high intake of ingested or administered sodium, patients with impaired renal function such as a patient with oliguric renal failure with retention of sodium, and a dialysis error. Hypervolemia occurs with a serum sodium level of more than 145 mmol/L is established. Clinical manifestations can range from thirst to serious complications such as pulmonary edema [5].

Potassium is the most abundant intracellular fluid electrolyte. The hormone aldosterone is released from the adrenal gland when potassium levels are high. The exchange process is done with hydrogen ions instead of sodium. If hydrogen ions are retained, metabolic acidosis can occur. Another method to lower high potassium levels is increased insulin, moving potassium into and out of cells. Potassium is critical for the function of nerve and muscle cells, and the cardiac muscles are extremely sensitive to hyperkalemia, resulting in dysrhythmias [6].

Hyperkalemia happens via retention of potassium by the kidneys, disturbance to aldosterone secretion and distal flow, oliguric patient, a high-potassium diet, and taking an angiotensin converting enzyme inhibitor with a high-normal range of serum potassium [7].

Plasma calcium concentration is maintained within a narrow range (8.5-10.5 mg/dL) by the coordinated action of parathyroid hormone (PTH), $1,25(OH)_2D_3$, calcitonin, and ionized calcium (iCa^{2+}) itself. The kidney plays a key role in this process by the fine regulation of calcium excretion. More than 95% of filtered calcium is reabsorbed along the renal tubules. In the proximal tubules, 60% of filtered calcium is reabsorbed by passive mechanisms. In the thick ascending limb,

15% of calcium is reabsorbed by paracellular diffusion through paracellin-1 (claudin-16) [8].

Aim of study

To discuss the cations levels in kidney impairment among the routine patients attending Baquba teaching hospital.

Patients and methods

Patients: This is a cross sectional study. It was conducted in the period from October 2022 to January 2023. We collected a sample of 40 patients who attend Baquba teaching hospital. Every patient with kidney impairment was eligible for study and we excluded the patients who were older. We collected informations about age, gender, the serum electrolyte levels (Na, K and Ca). We used the normal ranges below as control group to determine the increase and decrease in electrolytes.

- Na+ 140 (135-145) (mEq/L)
- K+ 4.5 (3.5-5.5) (mEq/L)
- Ca+ 9.5 (8-10.5) (mg/dl)

We preserved the privacy and we coded the patients for the reasons of confidentiality and risk of bias.

Statistical analysis

SPSS Version 25 was used for the description of the data. We expressed the quantitative data by arithmetic mean, standard deviation and mode and the qualitative data by frequencies.

Methods:- Monarch Biorex® 240 was used in our study. The ion-selective electrode method of electrolyte measurement is used. The membrane is made of a material that is selectively permeable to the ion being measured. The inside of the electrode is filled with a fluid containing sodium ions, and the outside of the glass membrane is immersed in the sample. The membrane acts as an ion exchanger, thereby causing a change in the membrane potential. The potential difference

developed across the glass membrane is dependent upon the difference in cations concentration or activity on the inside and outside of the glass membrane. This potential is measured by comparing it to the potential of a reference electrode. The potential of the reference electrode is kept constant, the difference in voltage between the other two electrodes is thus due to the concentration of cations in the sample.

Results

40 patients were enrolled in this study. Their mean age was 40.47 years and their age groups are demonstrated in table 1.

Table 1. Age groups

Age groups	Frequency	Percent
Less than 10 years	1	2.5
10-30 years	9	22.5
30-50 years	19	47.5
More than 50 years	11	27.5
Total	40	100.0

Half of them were males and half were female as demonstrated in table 2.

Table 2. Gender and age groups

	Gender		
Age groups	Male	Female	**Total**
Less than 10 years	0	1	1
10-30 years	5	4	9
30-50 years	8	11	19
More than 50 years	7	4	11
Total	20	20	40

The cations levels are demonstrated in table 3.

Table 3. Cations levels

Cations levels		Frequency	Percent
Na^+	Hypernatremia	12	30.0
	Normal	21	52.5
	Hyponatremia	7	17.5
	Total	40	100.0
K^+	Hyperkalemia	11	27.5
	Normal	25	62.5
	Hypokalemia	4	10.0
	Total	40	100.0
Ca^+	Hypercalcemia	1	2.5
	Normal	9	22.5
	Hypocalcemia	30	75.0
	Total	40	100.0

The distribution among age groups is demonstrated in table 4.

Table 4. Age groups distribution

Cations		Age groups				
		Less than 10 years	11-30 years	30-60 years	More than 60 years	Total
Na^+	Hypernatremia	0	3 (25%)	5 (41%)	4 (33%)	12
	Normal	1 (4.7%)	5 (23.8%)	13 (62%)	2 (9.5%)	21
	Hyponatremia	0	1 (14.2%)	4 (57%)	2 (28.5%)	7
Total		1	9	22	8	40
K^+	Hyperkalemia	0	2 (18%)	7 (64%)	2 (18)	11
	Normal	1 (4%)	7 (28%)	13 (52%)	4 (16%)	25
	Hypokalemia	0	0	2 (50%)	2 (50%)	4
Total		1	9	22	8	40
Ca^+	Hypercalcemia	0	1 (100%)	0	0	1
	Normal	0	2 (22.2%)	5 (55.5%)	2 (22.2%)	9
	Hypocalcemia	1 (3.3%)	6 (20%)	17 (56.6%)	6 (20%)	30
Total		1	9	22	8	40

The distribution among the gender is demonstrated in table 5.

Table 5. Gender distribution

		Gender		
		Male	Female	Total
Na^+	Hypernatremia	6 (50%)	6 (50%)	12
	Normal	9 (42.8%)	12 (58.2%)	21
	Hyponatremia	5 (71.5%)	2 (28.5%)	7
Total		20	20	40
K^+	Hyperkalemia	6 (54.5%)	5 (45.5%)	11
	Normal	12 (48%)	13 (52%)	25
	Hypokalemia	2 (50%)	2 (50%)	4
Total		20	20	40
Ca^+	Hypercalcemia	1 (100)	0	1
	Normal	4 (44.4%)	5 (55.6%)	9
	Hypocalcemia	15 (50%)	15 (50%)	30
Total		20	20	40

Discussion

In the current study, we demonstrate the association between the cation electrolytes levels in kidney impairment. Up to our knowledge this the first study about this topic in Diyala governorate.

We demonstrated the cations levels in kidney impairment and we used the hospital records as a reference for our findings.

We found that the sodium level was abnormal in 47.5% of the patients, 37.5% of them had abnormal potassium levels and 77.5% of them had abnormal calcium levels. Hypocalcemia was the commonest finding due to the decrease synthesis of vitamin D in renal failure patients.

The high percentage of calcium ion (77.5%) compered to sodium (47.5%) and potassium (37.5%) is due to lowest relative atomic radii of Ca^{++} (114 pm) compared to sodium (116 pm) and potassium (152 pm) which make the movements and excretion of both sodium and potassium ions easier than calcium.

Figure : Relative radii of cations ions.

Almost there was no difference in electrolyte changes in males and females which is consistent with findings of Yi et al [9].

Hyponatraemia has many causes and it is prevalent in hospitalised elderly patients which is associated with increased mortality. In the study by Sunderam et al., an iatrogenic origin was thought to explain the low sodium levels in 73% of inpatients and was attributed mainly to intravenous fluids and diuretic use [10].

Even though urinary excretion of K^+ decreases with a reduction in number of nephrons due to decreased renal function, K^+ secretion per nephron and K^+ excretion in the intestine are increased in a compensatory manner [11].

Patients with chronic kidney disease (CKD) have marked disruption in bone and mineral metabolism resulting in a complex disorder that has been termed CKD-mineral bone disorder (CKD-MBD). Perturbations begin in the earliest stages of the CKD and worsen with progressive kidney disease [12]. The biochemical alterations of CKD-MBD include elevated fibroblast growth factor-23 (FGF23) and parathyroid hormone (PTH), decreased 1,25-dihydroxyvitamin D (1,25D), elevated serum phosphate, and decreased serum calcium. Additionally, decreased calcium absorption and decreased urinary calcium excretion are observed, as well as heterogeneous bone disease and excessive vascular and soft tissue calcification. CKD-MBD is associated with an increased fracture risk and higher rate of cardiovascular events and cardiovascular-related deaths [13].

Conclusion

Cation levels are affected by renal impairment and could have catastrophic effect on the heart, bone and blood pressure. It should be managed carefully.

References

1. Inker LA, Perrone RD, Sterns RH, et al. Assessment of kidney function. UpToDate. Waltham (MA): UpToDate Inc; 2017
2. Kear TM. Fluid and Electrolyte Management Across the Age Continuum. Nephrology Nursing Journal. 2017 Nov 1;44(6).
3. Ellison D, Farrar FC. Kidney influence on fluid and electrolyte balance. Nursing Clinics. 2018 Dec 1;53(4):469-80.
4. Skorecki K, Ausiello D. Disorders of sodium and water homeostasis. InGoldman's Cecil Medicine 2012 Jan 1 (pp. 720-734). WB Saunders.
5. Casey G. Kidney care and renal disease. Nurs N Z 2012;18(4):20–4.
6. Rosenberg M. Overview of the management of chronic kidney disease in adults. UpToDate[Internet]. 2018.
7. Hammer GD, McPhee SJ. Pathophysiology of Disease: An Introduction to Clinical Medicine 7/E. McGraw-Hill Education; 2014.
8. Jeon US. Kidney and calcium homeostasis. Electrolytes & Blood Pressure: E & BP. 2008 Dec 1;6(2):68-76.
9. Yi TW, Levin A. Sex, gender, and cardiovascular disease in chronic kidney disease. InSeminars in Nephrology 2022 Mar 1 (Vol. 42, No. 2, pp. 197-207). WB Saunders.
10. Panese S, Mártin RS, Virginillo M, Litardo M, Siga E, Arrizurieta E, Hayslett JP. Mechanism of enhanced transcellular potassium–secretion in man with chronic renal failure. Kidney international. 1987 Jun 1;31(6):1377-82.
11. Isakova T, Wahl P, Vargas GS, Gutiérrez OM, Scialla J, Xie H, Appleby D, Nessel L, Bellovich K, Chen J, Hamm L. Fibroblast growth factor 23

is elevated before parathyroid hormone and phosphate in chronic kidney disease. Kidney international. 2011 Jun 2;79(12):1370-8.

12. Moorthi RN, Moe SM. CKD–mineral and bone disorder: core curriculum 2011. American journal of kidney diseases. 2011 Dec 1;58(6):1022-36.

13. Hill Gallant KM, Spiegel DM. Calcium balance in chronic kidney disease. Current osteoporosis reports. 2017 Jun;15:214-21.