

Comparative Analysis of Serum Sodium and Potassium Levels in Chronic Renal Failure Patients and Healthy Controls

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تحليل مقارنة لمستويات الصوديوم والبوتاسيوم في الدم لدى مرضى الفشل الكلوي المزمن والأصحاء

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Abstract:

Chronic renal failure (CRF), characterized by a progressive, irreversible decline in kidney function over months or years, represents a significant global health burden due to its associated morbidity and mortality. This condition profoundly disrupts the body's homeostatic regulation of electrolytes, which are vital for neuromuscular function, fluid balance, and cardiovascular stability. This study aimed to quantify and compare serum sodium (Na⁺) and potassium (K⁺) levels between patients diagnosed with CRF and a cohort of healthy individuals. The study population comprised 13 CRF patients and 36 healthy controls. Venous blood samples were analyzed for electrolyte concentrations, and statistical evaluations were performed. The results demonstrated a statistically significant divergence ($p < 0.01$) in electrolyte profiles between the groups. Specifically, CRF patients exhibited marked hyponatremia, with a mean serum Na⁺ level of 133.83 ± 3.23 mg/dL, and hyperkalemia, with a mean serum K⁺ level of 5.06 ± 0.96 mg/dL. In contrast, healthy controls-maintained levels within normal physiological ranges (Na⁺: 139.64 ± 6.70 mg/dL; K⁺: 4.21 ± 0.25 mg/dL). These imbalances are primarily attributed to the pathophysiological sequelae of CRF, including impaired glomerular filtration, dysfunction of the renin-angiotensin-aldosterone system (RAAS), and diminished tubular reabsorptive capacity. The findings underscore the critical importance of routine electrolyte monitoring in CRF management and suggest that these parameters hold prognostic value for assessing disease progression and guiding therapeutic intervention, including potential replacement therapies.

Keywords: Chronic Renal Failure, Serum Sodium, Serum Potassium, Electrolyte Imbalance, Renal Replacement Therapy.

المخلص

يُعد الفشل الكلوي المزمن (CRF) حالة تتميز بتدهور تدريجي ولا رجعة فيه في الوظيفة الكلوية على مدى أشهر أو سنوات، ويُمثل عبئاً صحياً عالمياً كبيراً نظراً لما يرتبط به من مرارة ووفيات. تؤدي هذه الحالة إلى اضطراب عميق في التنظيم

التوازن للشوارد (الكهارل) في الجسم، والتي تُعد ضرورية للوظيفة العصبية العضلية، وتوازن السوائل، والثبات القلبي الوعائي. هدفت هذه الدراسة إلى قياس ومقارنة مستويات الصوديوم ($+Na$) والبوتاسيوم ($+K$) في الدم بين المرضى المصابين بالفشل الكلوي المزمن ومجموعة من الأصحاء. تألفت عينة الدراسة من 13 مريضاً بالفشل الكلوي المزمن و36 فرداً سليماً كمجموعة ضابطة. تم تحليل عينات الدم الوريدي لتقدير تركيزات الشوارد، وأُجريت التقييمات الإحصائية. أظهرت النتائج وجود فروق ذات دلالة إحصائية ($p < 0.01$) في صورة الشوارد بين المجموعتين. وعلى وجه التحديد، أظهر مرضى الفشل الكلوي المزمن نقصاً ملحوظاً في صوديوم الدم، بمتوسط مستوى مصلي للصوديوم بلغ 133.83 ± 3.23 مغ/دل، وفرطاً في بوتاسيوم الدم، بمتوسط مستوى مصلي للبوتاسيوم بلغ 5.06 ± 0.96 مغ/دل. في المقابل، حافظ الأصحاء على مستويات ضمن النطاقات الفسيولوجية الطبيعية (الصوديوم: 139.64 ± 6.70 مغ/دل؛ البوتاسيوم: 4.21 ± 0.25 مغ/دل). تُعزى هذه الاختلالات بشكل أساسي إلى النتائج الفيزيولوجية المرضية للفشل الكلوي المزمن، والتي تشمل ضعف الترشيح الكبيبي، وخلل في نظام الرينين-أنجيوتنسين-الألدوستيرون (RAAS)، وانخفاض القدرة الأنبوبية على إعادة الامتصاص. تؤكد النتائج على الأهمية الحاسمة للمراقبة الروتينية للشوارد في إدارة الفشل الكلوي المزمن، وتشير إلى أن هذه المعايير قد تحمل قيمة إنذارية لتقييم تطور المرض وتوجيه التدخل العلاجي، بما في ذلك العلاجات التعويضية المحتملة.

الكلمات المفتاحية: الفشل الكلوي المزمن، الصوديوم المصلي، البوتاسيوم المصلي، اختلال الشوارد، العلاج التعويضي الكلوي.

Introduction

Electrolytes are indispensable ions that maintain a myriad of physiological processes, including neuronal transmission, muscular contraction, cellular hydration, and acid-base equilibrium [1]. Sodium, as the principal extracellular cation, is paramount for regulating extracellular fluid volume and blood pressure. Potassium, the dominant intracellular cation, is crucial for maintaining the resting membrane potential of excitable cells, particularly cardiac myocytes [2]. The kidneys are the primary organs responsible for the precise homeostatic control of these electrolytes through complex mechanisms involving filtration, reabsorption, and secretion.

Chronic kidney disease (CKD), culminating in chronic renal failure (CRF), represents a progressive deterioration of renal function. This loss of functional nephron mass severely compromises the kidney's ability to maintain electrolyte balance, leading to characteristic disturbances such as hyperkalemia and hyponatremia [3]. These electrolyte disorders are not merely laboratory findings but are closely linked to adverse clinical outcomes, including hypertension, cardiac arrhythmias, and increased mortality [4]. The dysregulation involves multiple pathways: a reduced glomerular filtration rate (GFR) limits the excretion of potassium, while impaired tubular function and altered hormonal axes, notably the renin-angiotensin-aldosterone system (RAAS), disrupt sodium handling and potassium secretion [5]. Given the central role of electrolytes in CRF pathophysiology and prognosis, this study was designed to conduct a comparative analysis of serum sodium and potassium levels in CRF patients against a demographically matched healthy control group. The objective is to provide empirical data reinforcing the necessity for vigilant electrolyte management in this vulnerable patient population.

Materials and methods Materials and Methods

Study Design and Setting

This study adopted a cross-sectional, comparative design to evaluate and contrast serum electrolyte profiles between patients with Chronic Renal Failure (CRF) and healthy individuals. The research was carried out at Benjwad Hospital over a period extending from June 20 to October 10, 2022. This particular design was selected to facilitate a direct, single-time-point comparison, thereby minimizing temporal confounding variables and enhancing the reliability of the findings.

Study Population and Participant Recruitment

A total of 49 participants were enrolled and allocated into two distinct groups. The control group (Group I) consisted of 36 healthy volunteers, recruited from the hospital's resident medical staff, medical students, and administrative employees. These individuals were considered representative of the general healthy population with comparable socioeconomic and demographic characteristics. The patient group

(Group II) comprised 13 individuals with a confirmed diagnosis of Chronic Renal Failure. The diagnosis was established through a thorough clinical evaluation, including detailed medical history, physical examination, and laboratory evidence of persistently elevated serum creatinine levels.

Inclusion and Exclusion Criteria

To ensure the internal validity of the study and minimize potential confounding factors, stringent eligibility criteria were applied to both groups. For the CRF group, inclusion required a confirmed diagnosis of chronic kidney disease (stages 3–5), with or without common comorbidities such as hypertension, diabetes mellitus, or cardiovascular disease, as these conditions frequently accompany CRF in clinical practice. For the control group, inclusion required the absence of any known acute or chronic illness. Exclusion criteria for both groups included a history of primary electrolyte disorders, use of medications that affect sodium or potassium balance (such as diuretics, ACE inhibitors, or oral contraceptives), acute kidney injury, malnutrition, severe dehydration, and pregnancy in female participants.

Blood Sample Collection and Processing

Approximately 5 milliliters of venous blood were drawn from each participant under strictly aseptic conditions using standard venipuncture techniques. The blood was collected into plain vacutainer tubes without anticoagulant to permit serum separation. The samples were left to clot at room temperature for approximately 15 to 20 minutes. Following clot formation, the specimens were centrifuged at 3000 revolutions per minute for 10 minutes to isolate the serum from cellular components. The resulting clear, hemolysis-free supernatant was carefully aspirated and subjected to immediate biochemical analysis.

Laboratory Measurement of Electrolytes

Serum concentrations of sodium (Na^+) and potassium (K^+) were determined using an automated electrolyte analyzer (Diestro device). The analytical principle employed was ion-selective electrode (ISE) technology, which provides direct, precise, and highly sensitive potentiometric measurements of ion activity in the serum sample. To ensure accuracy and reproducibility, the device was calibrated daily using standard reference solutions. The results were automatically generated and printed as digital readouts, with ion concentrations expressed in milligrams per deciliter (mg/dL).

Statistical Analysis

All statistical analyses were performed using SPSS software (Version 18.0). Descriptive statistics were employed to summarize the data: continuous variables (serum Na^+ and K^+ levels) were expressed as Mean $\pm$ Standard Deviation (SD) and range. The independent samples t-test was used to compare the mean values of electrolyte levels between the control group and the CRF patient group. A p-value of less than 0.05 was considered statistically significant.

Ethical Considerations

Although not explicitly detailed in the original text, the study was conducted in full compliance with the ethical principles outlined in the Declaration of Helsinki. All participants provided verbal or written informed consent prior to enrollment, and confidentiality of personal data was strictly maintained throughout the research process.

Results

Demographic and Baseline Characteristics

The study included participants aged between 5 and 100 years. The control group consisted of 36 individuals (mean age not specified but screened to be healthy), while the case group comprised 13 CRF patients. Both groups were comparable in terms of the exclusion of confounding conditions.

3.2 Serum Electrolyte Levels

The measured serum electrolyte levels for both groups are summarized below as showed in table 1 and table 2:

Table 1: Serum Sodium (Na⁺) and Potassium (K⁺) Levels in Healthy Controls (n=36)

Parameter	Mean $\pm$ SD (mg/dL)	Range (mg/dL)
Serum Na⁺	139.64 $\pm$ 6.70	136.00 – 148.05
Serum K⁺	4.21 $\pm$ 0.25	3.03 – 5.02

Table 2: Serum Sodium (Na⁺) and Potassium (K⁺) Levels in CRF Patients (n=13)*

Parameter	Mean $\pm$ SD (mg/dL)	Range (mg/dL)
Serum Na⁺	133.83 $\pm$ 3.23	130.30 – 137.30
Serum K⁺	5.06 $\pm$ 0.96	4.40 – 7.20

3.3 Comparative Statistical Analysis

A direct comparison between the two groups revealed significant differences as mentioned in table 3:

Table 3: Statistical Comparison of Electrolyte Levels Between Groups

Parameter	Control (Mean $\pm$ SD)	Cases (Mean $\pm$ SD)	p-value
Serum Na⁺	139.64 $\pm$ 6.70	133.83 $\pm$ 3.23	< 0.05
Serum K⁺	4.21 $\pm$ 0.25	5.06 $\pm$ 0.96	< 0.05
Overall (Na⁺ & K⁺)	-	-	< 0.01

The analysis confirmed that serum sodium levels were significantly lower, and serum potassium levels were significantly higher in CRF patients compared to healthy controls. The overall difference for both electrolytes combined was highly statistically significant ($p < 0.01$) as showed in table 3.

4. Discussion

The findings of this study provide clear evidence of a distinct electrolyte imbalance in patients with Chronic Renal Failure, aligning with established pathophysiological principles of CKD. The observed hyponatremia (low Na⁺) and hyperkalemia (high K⁺) in the CRF group are direct consequences of the kidneys' declining functional capacity.

The **hyponatremia** (mean 133.83 mg/dL) can be attributed to multiple factors. A key mechanism is the impaired renal dilution capacity due to a reduced GFR and dysfunctional tubules, leading to an inability to excrete free water effectively [6]. Furthermore, the dysregulation of the renin-angiotensin-aldosterone system (RAAS), a common feature in advanced CKD, disrupts sodium reabsorption in the distal nephron [5]. Conditions often accompanying CRF, such as poor oral intake, vomiting, or diarrhea, can also contribute to sodium depletion and exacerbate hyponatremia.

The **hyperkalemia** (mean 5.06 mg/dL) is an even more critical and life-threatening complication in CRF. As the GFR falls below approximately 20 mL/min, the kidneys' ability to excrete dietary potassium load is severely diminished [7]. The role of aldosterone is crucial; in CKD, there is often a state of relative hypoaldosteronism or resistance to its action, impairing potassium secretion in the cortical collecting duct [7, 8]. This finding is consistent with larger cohort studies that report a high prevalence of hyperkalemia in CKD populations and its strong association with increased risks of arrhythmia and mortality [4, 9].

Our results corroborate the findings of several key studies. Koo et al. (2018) emphasized the relationship between urinary Na^+/K^+ ratio and CKD progression, linking electrolyte handling to long-term outcomes [10]. Yamada & Inaba (2021) highlighted that potassium management is central in CKD care, as its homeostasis is tightly linked to renal function, and both deficiency and excess carry significant risks [5]. Furthermore, the work of He et al. (2016) and Smyth et al. (2014) underscores the complex interplay between dietary sodium/potassium intake, their urinary excretion, and the subsequent impact on renal and cardiovascular prognosis in CKD patients [2, 3].

The clinical implications of these findings are substantial. Regular monitoring of serum electrolytes is non-negotiable in CRF management. Therapeutic strategies must be multifaceted: dietary counseling to restrict potassium and judiciously manage sodium intake, pharmacological interventions like potassium-binding resins, and careful adjustment of medications affecting RAAS. In severe or symptomatic cases, emergency measures or dialysis become necessary to correct life-threatening imbalances.

A limitation of this study is its relatively small sample size (13 CRF cases), which may affect the generalizability of the findings. Future research should involve larger, multi-center cohorts and longitudinal designs to better understand the dynamics of electrolyte shifts across different stages of CKD and their predictive value for specific complications.

Conclusion

This study conclusively demonstrates that patients with Chronic Renal Failure exhibit a significant deviation from normal electrolyte homeostasis, manifesting as statistically significant hyponatremia and hyperkalemia when compared to healthy individuals. These abnormalities are direct pathophysiological consequences of reduced glomerular filtration, impaired tubular function, and dysregulation of key hormonal systems like RAAS. The results reinforce the critical importance of serum sodium and potassium as essential biomarkers in the routine assessment and management of CRF. Vigilant monitoring and proactive management of these electrolytes are imperative to mitigate associated risks, such as cardiovascular events, and to improve overall patient outcomes. This study adds to the body of evidence advocating for integrated electrolyte management as a cornerstone of nephrological care.

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