International Journal of Research in Medical Sciences
Ongong'a RO et al. Int J Res Med Sci. 2024 Jan;12(1):37-43
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012

DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20233974

Original Research Article

Assessment of electrolyte imbalance among benign prostate hyperplasia patients in Western Kenya

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Received: 18 October 2023
Revised: 14 November 2023
Accepted: 06 December 2023

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ABSTRACT

Background: Benign prostate hyperplasia (BPH) results in the enlargement of the gland and ultimately obstructs the bladder and the kidney. The effect on the kidney results in the dysregulation of the electrolyte causing electrolyte imbalance.
Methods: An analytical cross-sectional study conducted at a tertiary teaching hospital aimed at assessing the levels and severity of electrolyte imbalance among BPH patients. The blood samples were analyzed for electrolytes and PSA levels for both patients and control group. An Independent t-test was used to compare the means of the BPH patients and healthy control subjects. Chi-square was used to determine the association between the electrolyte imbalance and the PSA levels of the BPH patients.
Results: The mean age of the BPH patients and the healthy individuals was 65.47±12.55 and 64.52±12.19 years respectively. Hyponatremia, and hypernatremia were observed in 26.08% (n=104) and 4.22% (n=8) of the BPH patients respectively. There was a statistical significance positive correlation between potassium (K) and sodium (Na) concentrations ($r=0.350$, $p<0.01$), as well as a notable positive association between chloride (Cl) and magnesium (Mg) levels ($r=0.288$, $p<0.01$). PSA biomarker levels varied among the patients. There was a statistical significance (<0.0001*) difference in PSA levels between the BPH patients and the control group.
Conclusions: With high prevalence of electrolyte imbalance among BPH patients there is a need to monitor the electrolytes and PSA levels in the management of BPH aiming at restoration of kidney function.

Keywords: Benign prostate hyperplasia, Imbalance, Kidneys, PSA, Serum electrolyte

INTRODUCTION

Benign prostate hyperplasia (BPH) is one of the urological diseases affecting aged men above the fourth decade globally. The condition is non-malignant and results in the enlargement of the prostate glands leading to the manifestation of the lower urinary tract symptoms which include, incomplete emptying of the bladder, intermittent urination, stopping urination while beginning. In addition, this symptom further affects the quality of life of an individual.[1,2] Globally it is estimated that around 90 million people have symptoms related to BPH making it one of the major public health problems among the elderly thus increasing the healthcare cost.[3,4] In Africa, 50% of male aged 65 years and above have also demonstrated symptoms related to BPH and at some point, in time experienced lower urinary tract symptoms (LUTS). The prevalence of BPH in men aged 60 years and above have been recorded to be about 56% in Kenyan population.[5,6] The aetiopathogenesis of BPH is surmised to be regulated by several biological processes such as hormonal imbalance tissue degeneration due to

aging and metabolic syndromes.[7] Histology has been outlined as the perfect means of diagnosing BPH. However several diagnostic methods such as questionnaire scores, imaging reports, physical examinations, and laboratory reports must be evaluated before concluding that a patient is suffering from BPH.[8,9]

Whilst BPH exhibits itself as a localized prostatic disorder some studies have suggested that it may have an effect on the body system, and if not managed properly can cause several complications such as urinary retention, renal insufficiency, and renal failure.[10] Electrolytes are essential for supporting the numerous physiological processes in the body. They are electrically charged particles that promote the healthy operations of the organs and tissues, control cellular activity, and maintain fluid equilibrium.[11,12] Irregular expression of electrolytes within the body has been known to aggravate the progression of certain conditions such as metabolic syndrome and BPH. These conditions may arise due to impairment of the steady state of the body electrolytes, patients may tend to display sodium, potassium, chloride, magnesium, and bicarbonate abnormalities, which may have a connotation in the management of the patient.[13,14] The impairment of electrolyte concentration within the body can be measured in the serum using biochemical techniques this can be used to determine the clinical manifestation in patients.[15] In recent years, more attention has been drawn to the possibility that BPH can affect the homeostasis of electrolytes. BPH has been linked to changes in electrolyte levels in several studies, raising the possibility of an interaction between the disease and electrolyte balance. This electrolyte imbalance could be detrimental to the pathophysiology of BPH exacerbating the side effects and also interfering with the treatment results.[16]

The study aimed to assess the prevalence and severity of electrolyte imbalance among BPH patients in Kisumu County. We also examined the association between the PSA level and the electrolyte profile.

METHODS

The analytical cross-sectional study was conducted at Jaramogi Oginga Odinga Teaching and Referral Hospital, in Western Kenya. The hospital is located approximately 450km west of the capital city of Kenya. During the two years of study (2020-2022), a total of 388 subjects were recruited, 194 being cases (confirmed BHP patients) and the other 194 being the control group (healthy population).

Ethical clearance was sought from the ethics review committee of Jaramogi Oginga Odinga teaching and referral Hospital. In addition, all participants consented to participate in the study. The declaration of Helsinki was followed while handling all patient records, and confidentiality was upheld by eliminating all patient identification information. Data were kept on a password-protected computer that only the investigator could access.

Inclusion criteria

All the BPH patients were newly confirmed through histological diagnosis before recruited into the study. The recruitment only included male patients aged 35 and above.

Exclusion criteria

Patients with chronic illnesses such as diabetes and hypertension, metabolic syndrome, any other disease due to malnutrition, and conditions known to interfere with electrolyte measurement such as dehydration, patients on medications such as diuretics, men on dietary supplements were excluded from the study. The patients with renal dysfunction were also excluded from the study.

The control group aged 35 years and above was recruited from healthy male individuals with no history of BPH or no signs and symptoms related to urological ailment. The age of the participants was collected through verbal questioning and from the patients' files and confirmed by checking of the ID cards.

Laboratory analysis

The venous blood sample was collected from the brachial vein for both the patients and the control group in a yellow top vacutainer and serum was prepared for the estimation of the PSA biomarker and the electrolyte. The electrolytes were estimated by the 9180-electrolyte analyzer Roche@™ under the principle of ion-selective electrodes while the PSA biomarker levels were analyzed using the Cobas e411 machine Roche @™ under the principle of elecsys sandwich method. The reference range for sodium, potassium, chloride, magnesium, bicarbonate, and PSA levels was 135-155mmol/l, 3.5-5.5mmol/l, 97-107mmol/l, 0.66-1.07mmol/l, 21-28mmol/l and 0-4ng/ml respectively, according to the recommendations of the international federation of clinical chemistry. The sodium levels below the reference range were termed hyponatremia while the levels above the upper limit of the reference range were termed hypernatremia, the potassium levels below the reference lower limit of the reference range were termed hypokalemia while the potassium levels above the upper limit of the reference range were termed hyperkalemia, chloride levels below the reference range was termed hypochloremia while the chloride levels above the upper reference range were termed hyperchloremia, magnesium levels below the lower limit of the reference range was termed hypo magnesium while the levels above the upper reference range were termed hyper magnesium, low bicarbonates levels below the lower reference range was termed acidosis while the bicarbonate levels above the reference range limit were termed alkalosis.

Data analysis

The clinical data and sociodemographic data were obtained from the patients through verbal questions and access to the patient's files. The statistical analysis was carried out using the IBM® SPSS® Version 25. The distribution and expression of the PSA marker were presented as numbers and percentages. Independent t-test was used to compare the means of the patients with BPH and the healthy control subjects. Chi-square was used to determine the association between the electrolyte imbalance and the PSA levels of the BPH patients while the Pearson correlation was used to determine the correlation between the electrolyte levels and the age of the patients. The statistical levels were recorded with 95% confidence interval and statistically significant was considered at $p<0.05$.

RESULTS

A total of 194 patients met the inclusion criteria for cases, and 194 individuals for the control group. The mean age ±SD was 65.47±12.55 (38-92 years) for cases and the control group was 64.52±12.19 (39-91 years). Most of the BPH patients and the control group were realized in the age group 70-79 years (n=53, n=52 respectively) years. The least number was 90-99 years (n=2) age group for the cases while the least number in 30-39 years (n=1) age group for the control group, based on the educational status, 59.01% (n=115) of the BPH patients had no formal education. In contrast, in the control group, 53.09% (n=100) had no formal education. Additionally, 67.1% (n=130) of the patients resided in a rural setup, while 55.1% (n=107) of the control participants lived in a rural setup. The majority of the patients (63.9%, n=124) had experienced BPH symptoms for a period of 6-12 months, while none of the control participants had these symptoms. Moreover, the severity of lower urinary tract symptoms (LUTS) was classified as severe in the majority of the patients (63.1%, n=123) none of the control participants presented with LUTS (Table 1).

In patients, the mean PSA levels were 135.76±578.03, whereas the control participants had a mean of 2.01±1.09. For sodium levels, the mean was 137.17±9.84 among patients, and control participants had a mean of 143.64±5.95. The mean for potassium ions was 4.15±0.89 in patients and 4.4±0.66 in the control group. As for hydrogen carbonate, patients had a mean of 24.86±3.10, while the control participants had a mean of 1.99±0.14 (Table 2).

Table 1: Characteristics of the study participants.

Characteristics	Categories	BPH patients (n=194) (%)	Control group (n=194) (%)	P value
Ages (years)	30-39	3 (1.5)	1 (0.5)	0.73
	40-59	25 (12.9)	21 (10.8)	
	50-59	41 (21.1)	45 (23.2)	
	60-69	46 (23.7)	51 (26.3)	
	70-79	5.3 (27.3)	52 (26.8)	
	80-89	24 (12.4)	22 (11.3)	
	90-99	2 (1.0)	2 (1.0)	
Education	Formal	79 (40.72)	91 (46.90)	0.34
	Informal	115 (59.27)	103 (53.09)	
Occupation	Salaried	38 (14.43)	44 (22.68)	0.07
	Farmer	51 (26.29)	59 (30.41)	
	Business	56 (28.87)	63 (32.47)	
	Unemployed	49 (25.26)	28 (14.43)	
Residence	Urban	64 (32.9)	87 (44.9)	0.02
	Rural	130 (67.1)	107 (55.1)	
Duration of BPH symptoms	0-6 months	21 (10.82)	0 (0.00)	
	6-12months	124 (63.9)	0 (0.00)	
	13-18months	46 (23.7)	0 (0.00)	
	>18months	03 (1.5)	0 (0.00)	
LUTS severity	Mild IPSS 0-7	27 (13.9)	0 (0.00)	
	Moderate IPSS (8-19)	44 (22.7)	0 (0.00)	
	Severe IPSS (20-35)	123 (63.4)	0 (0.00)	

After performing an independent t-test there was a statistical difference at a p value <0.05 in the sodium (0.000), potassium (0.000), and hydrogen carbonate (0.000), (Table 3).

In the case group, hyponatremia was observed in 26.08% (n=104), while hypernatremia was only seen in 3.10% (n=12). Hypokalemia affected 27.4% (n=53) of the patients, while 7.8% (n=15) had hyperkalemia. Hypochloremia was found in 19.1% (n=37), and 9.3% (n=18) presented with hyperchloremia. Hypomagnesemia was observed in 66.4% (n=129) of the patients, and 3.0% (n=06) had hypermagnesemia. Acidosis was noted in 14.4% (n=28) of the patients, and 14.9% (n=29) exhibited alkalosis. In the control group, general electrolyte imbalance affected 4.22% (n=26). Among them, 1.5% (n=6) had hyponatremia, while no individuals presented with hypernatremia. Potassium ion imbalances were found in 0.6% (n=1) with hypokalemia and 1.0% (n=2) with hyperkalemia. Hypomagnesemia was observed in 3% (n=6), while hypermagnesemia was revealed in 2% (n=4), Acidosis affected 2.6% (n=5), and 1.0% (n=2) experienced alkalosis (Table 4).

The measurement of the PSA biomarker revealed that 1.5% (n=3) of the patients secreted the PSA normally, 9.8% (n=19) mildly, 21.3% (n=41) moderately, and 67.5% (n=131) severely. In contrast, none of the control group participants had either moderate or severe secretion of the PSA biomarker. However, 1.5% (n=3) of the control participants had mild secretion (Table 5).

In the cases group, 95.88% (n=186) had electrolyte imbalance, while 4.22% (n=8) did not experience electrolyte imbalance. In the control group, it was observed that 3.1% (n=12) had electrolyte imbalance (Table 6).

Table 2: Comparison of the lab data of BPH patients and the control participants.

	Group	N	Mean	STD	SEM
Ages	Cases	194	65.41	12.55	0.90
	Control	194	64.57	12.06	0.87
Psa	Cases	194	135.76	578.03	41.50
	Control	194	2.01	1.09	0.079
Na+	Cases	194	137.17	9.84	0.71
	Control	194	143.64	5.95	0.43
K+	Cases	194	4.15	0.89	0.06
	Control	194	4.47	0.66	0.04
Cl-	Cases	194	101.06	4.63	0.33
	Control	194	101.77	6.48	0.47
HCO_3	Cases	194	24.86	3.10	0.22
	Control	194	24.49	1.99	0.14
Mg2+	Cases	194	0.61	0.17	0.01
	Control	194	0.86	0.19	0.01

Table 3: Unpaired T-test values of the BPH patients and the controls.

	f	P.value	t	df	MD	SED	CI Difference	
Ages	0.127	0.722	0.673	386.000	0.840	1.249	-1.615	3.296
PSA	19.088	0.000*	3.223	193.001	133.743	41.500	51.8912	215.597
Na+	29.004	0.000*	-7.841	317.378	-6.4742	0.825	-8.0988	-4.849
K+	31.685	0.000*	-4.150	339.785	-0.3191	0.077	-0.4703	-0.168
CL-	3.269	0.071	-1.240	386.000	-0.7093	0.572	-1.8343	0.416
HCO3	42.989	0.000*	1.394	329.130	0.3691	0.265	-0.1517	0.889
Mg2+	0.597	0.440	-13.512	386.000	-0.2512	0.018	-0.2877	-0.214

*Statistically significant at a p value <0.05

Table 4: Levels of electrolyte imbalance among BPH patients and controls.

Electrolytes	Variable	BPH patients		Controls		Total		P value
		N	%	N	%	N	%	
Sodium (Na^+)	Hyponatremia	104	26.80	6	1.50	110	28.3	0.00
	Normonatremia	78	20.10	188	48.50	266	68.60	0.00
	Hypernatremia	12	3.10	0	0.00	12	3.10	0.00
Potassium (K^+)	Hypokalemia	53	27.4	1	0.6	54	13.9	0.42
	Normokalaemia	126	65	191	98.4	317	81.7	0.93
	Hyperkalemia	15	7.8	2	1.0	17	4.4	0.24
Chloride (Cl-)	Hypochloremia	37	19.1	4	2.1	41	10.6	0.99
	Normochloremia	139	71.6	178	91.8	317	81.7	0.99
	Hyperchloremia	18	9.3	12	6.2	30	7.7	0.88
Magnesium (Mg+)	Hypo-magnesemia	129	66.4	06	3.0	135	34.8	0.00
	Normo-magnesemia	59	30.4	184	94.8	243	62.6	0.53
	Hyper-magnesemia	06	3.0	4	2.0	10	2.6	0.02
Bicarbonate (HCO_3)	Acidosis	28	14.4	5	2.6	33	8.5	0.36
	Normal	137	70.6	187	96.4	324	83.5	0.24
	Alkalosis	29	14.9	2	1.0	31	8.0	0.01

Table 5: Severity of PSA levels among the patients and control groups.

Variables (PSA levels)	Normal (0-4mmol/l)	Mild (4-9mmol/l)	Moderate (10-20mmol/l)	Severe (>20mmol/l)	Total
BPH patients	3	19	41	131	194
Control group	191	3	0	0	194
Total	194	22	41	131	388

Table 6: Frequency of electrolyte imbalance.

Group	Imbalance (%)	No imbalance (%)	Total	P value
Sample	186 (95.88)	08 (4.12)	194	
Control	26 (13.4)	168 (86.59)	194	0.242
	212	176	388	

Association between the two study groups and PSA level

The distribution of PSA levels among the BPH patients were as follows; 1.6% (3) normal, 9.8% (19) mild, 21.1% (41) moderate and 67.5% (131) presented with severe levels. The control group had PSA levels distributed as follows; 98.5% (191) normal and 1.5% (3) presented with mild PSA levels. The study observed that there was a statistically significant (<0.0001*) difference in PSA levels between the BPH patients and the control group at a chi-square value of 365.822 with a df of 3.

Association between age distribution and PSA level

Out of the 388 patients recruited into the study, the PSA levels were distributed as follows; 50% (194) had normal PSA levels, 5.7% (22) mild, 10.6% (41) moderate and 33.8% (131) presented with severe levels. There was a statistically significant (<0.0001*) association between the age distribution and PSA levels at a chi-square value of 267.826 with a df of 168.

Association between sodium levels and PSA level

Out of the 388 patients recruited into the study, the sodium levels were distributed as follows; 50% (194) had normal PSA levels, 5.7% (22) mild, 10.6% (41) moderate and 33.8% (131) presented with severe levels. There was a statistically significant (<0.001*) association between the sodium and PSA levels at a chi-square value of 267.826 with a df of 132.

Association between potassium levels and PSA level

Out of the 388 patients recruited into the study, the sodium levels were distributed as follows; 50% (194) had normal PSA levels, 5.7% (22) mild, 10.6% (41) moderate and 33.8% (131) presented with severe levels. There was a statistically significant (<0.003*) association between the sodium and PSA levels at a chi-square value of 150.113 with a df of 105.

DISCUSSION

The findings of the current study demonstrated a remarkable difference in electrolytes profiles between the BPH patients and the control group. The electrolyte imbalance as observed in Table 2, is known to aggregate the progression of BPH disease.[17] Among the electrolytes analyzed hypo magnesium or low levels of serum magnesium ions was the most prevalent abnormality in BPH patients. These results were consistent with which observed that hypo magnesium was common among BPH patients attending the Ghana police hospital.[18] Magnesium ions are usually involved in a series of physiological, cellular, and molecular processes within the body system that include proliferation and cell signalling.[19] Magnesium deficiency may result due to several conditions including reduced intake, alteration in reabsorption and excretion that may have been propagated by the tissue, and physiological alteration by the BPH. In severe cases of BPH disease, there is usually occurrence of hydronephrosis, hydronephrosis leads to the swelling of kidneys due to obstruction of urinary flow which is a common occurrence in BPH, the obstruction that is occasioned by the enlargement of the prostate glands may cause obstructed urine to back up into the kidneys causing inflammation and if left untreated this may become injurious to the renal tissues thus affecting the electrolyte regulation capacity of the kidneys.[20]

Hyponatremia and hypokalemia were the secondary electrolyte imbalance in our study. Both hyponatremia and hypokalemia might be associated with poor reabsorption which may be attributed to kidney injury or an injury due to urinary tract infection.[21] Chronic kidney injury has also been linked to hypochloremia, which may be caused by excessive chloride loss in urine as a result of tubular failure in the kidneys.[22,23] Hypochloremia is known to cause metabolic alkalosis which is maintained by an increase in Na+ and HCO3 resorption in the kidney at the cost of H^+, K^+, and Cl^-, aldosterone speeds up the retention of Na^+ and HCO_3.[24,25] BPH increases the risk of UTI due to the LUTS that is occasioned by incomplete emptying of the bladder that fosters a bacterial growth-friendly environment.[26]

The study observed that the BPH patients had very high levels of PSA secretions. The prostate surface antigen is produced by the stromal and epithelial cells of the enlarged prostate glands where it cleaves both the semenogelin I and II, most of the PSA that gets into the

circulation is intact and gets complexed with alpha-1 anti-chymotrypsin some get complexed with alpha-2-macroglobulin.[27,28] The elevation of the PSA can be attributed to the excessive growth within the prostate tissues together with the constriction within the prostatic ducts causing inflammation and trauma thus releasing a high amount of PSA.[29] Impaired clearance of PSA from circulation has also been suspected to cause a rise in PSA in patients with BPH attributed to urine obstruction by the inflammation of the prostatic tissues.[30]

This study has limitation. The study design was not able to establish the causality of electrolyte imbalance and BPH.

CONCLUSION

In conclusion, the electrolyte imbalance among BPH patients was high compared to the control group. The severity of the PSA production was significantly associated with electrolyte imbalance among the BPH patients. The study recommends the need for routine monitoring of electrolyte profiles of BPH patients with a view of prompt treatment to correct any deranged levels.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Ethics Review Committee of Jaramogi Oginga Odinga Teaching and Referral Hospital (ref no. IERC/JOOTRH/531/21)

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Cite this article as: Ongong'a RO, Menza N, Demba RN. Assessment of electrolyte imbalance among benign prostate hyperplasia patients in Western Kenya. Int J Res Med Sci 2024;12:37-43.