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Original Research Article

# Pattern of Electrolyte Derangements in the Setting of Bladder Outlet Obstruction (BOO): A Hospital Based Study

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**Abstract:** Bladder Outlet Obstruction is a major Urological problem especially in the aging male population. Effect of which may lead to Chronic kidney failure with attendant high mortality, decrease quality of life, increase health care expenditure and end-stage renal disease if untreated. This most often happens when presentation for care is late. Chronic kidney failure is characterized, among other things, by failure to maintain electrolyte homeostasis and inadequate excretion of metabolic wastes. A retrospective study of case notes of one hundred and three (103) patients who were seen in our facility between April 2014 and March 2015; with symptoms of bladder outlet obstruction. Eighty four (84) patients completed the required tests which were used for this study. Data from 84 patients with a mean age of 61.70 years (SD±11.139) were analyzed. The cause of BOO and their relative frequency were: Cancer of the prostate (Cap) in 32.0%, Benign prostatic hyperplasia in 50.2% and urethral stricture in 17.5%. Electrolyte derangements were seen in 18.3% for potassium, 6.1% for sodium, 10.8% for chroride and 35.7% for bicarbonate. There was no significant statistical association between electrolyte status and causes of BOO or even with the age of the patients. Electrolyte derangement is one of the complications of BOO resulting from renal compromise. Late presentation especially in high pressure chronic urinary retention most commonly lead to chronic renal failure. Preventive measures involving proactive health awareness to all men in this age group or even younger men should be done to forestall this morbid condition.
**Keywords:** Bladder outlet obstruction, electrolyte, pattern, renal failure.

## INTRODUCTION:

Bladder outlet obstruction is defined as impedance or blockage of urine flow from the bladder into the urethra [1]. The causes are numerous but majorly due to cancer of the prostate (Cap) benign prostate hyperplasia (BPH) and urethral stricture [2]. Complications arise mostly when presentation is late and include acute, acute or chronic or chronic urinary retention. This process disrupts normal renal homeostasis of fluid and electrolytes among other serum parameters handled by the kidneys. In this study, the principal electrolytes looked at were serum potassium, sodium, chloride and bicarbonate. The age of the patients and causes of BOO were also important in this study which did not however show any significant statistical association with the electrolyte status.

Normal renal function is central to electrolyte homeostasis. Filtration of blood occurs at the renal corpuscles, each of which is composed of a glomerulus and a Bowman's capsule. These ultra-filtrate flows along the length of the nephron, which is a tubular structure lined by a single layer of cells and surrounded by capillaries whose primary function is re-absorption of water and small molecules and secretion of wastes from blood into the urine. In the ultra-filtrate load, 65% of sodium is reabsorb in the proximal tubule, 25% in the thick ascending loop of Henle (through $Na^+$-$K^+$-2Cl symporter), 5% is reabsorbed in the distal tubule by $Na^+$-$Cl^-$ symporter and 5% in the principal cells of the collecting duct stimulated by aldosterone via ENaC[3]. Chlroide reabsorption usually follows sodium by active (transcellular) and passive (paracellular) process [3]. Potassium absorption varies upon dietary needs. Majority (~65%) is reabsorbed in the proximal tubule, 20% occurs in the thick ascending limb of the loop of Henle by $Na^+$-$K^+$-Cl symporter and secreted in the collecting duct via $Na^+$-$K^+$ ATPase increased by aldosterone or rarely reabsorbed via hydrogen potassium ATPase. Bicarbonate helps maintain the acid base balance and 80-90% reabsorption occurs in the proximal tubule and the rest in the thick ascending limb

of loop of Henle and intercalated cells of the collecting ducts [4].

In BOO, there is high detrusor pressure and low flow rate causing retrograde pressure and hydronephrosis. The result is progressive atrophy of the tubular cells and loss of tubular reabsorption of electrolytes leading to their accumulation in the blood. This study was meant to highlight the pattern of electrolyte derangements in the setting of bladder outlet obstruction. The patterns are as follows: 18.3%, 6.2%, 10.8%, 35.7% respectively for potassium, sodium, chloride and bicarbonate. The study did not show any significant statistical association between the causes of BOO, the age of the patients and their electrolyte status. However, the study was able to demonstrate a significant level of electrolyte derangements that may ultimately lead to high mortality in the setting of BOO if not addressed early. Preventive measures hinge on better health awareness to the population at risk of prostate disease which is said to account for more than 50% of lower urinary tract obstruction and retention in males [5].

## PATIENTS AND METHOD

One hundred and three (103) patients who were seen between April 2014 and March 2015 in our facility with symptoms of BOO were used for this study. Of this number eighty four (84) patients completed the required investigations. Information from their case notes included biodata, signs and symptoms, requisite investigations including urinalysis, urine microscopy culture and sensitivity, electrolyte urea and creatinine, prostate specific antigen (PSA), abdominopelvic ultrasound scan, retrograde urethrocystogram/micturating cystourethrogram and a prostate biopsy where indicated. Data collected were analysed using the statistical package for social sciences (SPSS) Version 20.0.

## RESULTs

Eighty four (84) out of one hundred and three (103) patients seen within the study period representing 81.5% of the population who were all males and of Christian faith were studied. The mean age was 61.70s years (SD±11.139). Eighty-two(82) patients had complete results for potassium with values between 1.7-6.3mmol/l with a mean of 4.458 SD±0.683. Eighty Two (82) patients also had complete results for sodium with values between 107-150mmol/l and a mean of 140.70 SD±4.971. Eighty three (83) patients had complete results for chloride with values ranging from 84-116mmol/l with a mean of 104.10 SD±4.833, while 84 patients had complete results for bicarbonate with a range of 13-28 and a mean of 22.29 SD±2.669. Potassium was deranged in 18.3% comprising of hyperkalaemia in 17.1% and hypokalaemia in 1.2%. Sodium was deranged in 6.2%; hypernatraemia in 4.9% and hyponatraemia in 1.2%. Chloride level was high in 10.8% and metabolic acidosis was encountered in 35.7% (Table 3). Prostate specific antigen (PSA) ranged from 0.5 to 121.8 with a mean of 32.236 SD±37.93. Patients seen were typically diagnosed with BOO secondary to cancer of the prostate, benign prostatic hyperplasia and urethral stricture respectively in the following relative frequency; 32.0% 50.5% and 17.5%. Mean age per diagnosis was equally noted as follows; 66.18±7.05, 62.85±7.02 and 50.17±17.87 respectively for Cap, BPH and Urethral Stricture (Table 2).

The majority of the respondents were of the Ibibio tribe (78.6%) and pensioners (33.0%) respectively.

**Table-1: Socio-demographic characteristics of respondents (n=103)**

| Characteristics | Frequency | Percent |
|---|---|---|
| **Tribe** | | |
| Ibibio | 81 | 78.6 |
| Annang | 19 | 18.4 |
| Igbo | 2 | 1.9 |
| Others | 1 | 1.0 |
| **Occupation** | | |
| Pensioners | 34 | 33.0 |
| Trading | 23 | 22.3 |
| Civil/Public servants | 22 | 21.4 |
| Farmers | 11 | 10.7 |
| Artisans | 7 | 6.8 |
| Drivers | 3 | 2.9 |
| Applicants | 3 | 2.9 |

**Table-2: Clinical Characteristics and mean age of respondents**

| Characteristics | Diagnosis | | |
|---|---|---|---|
| | Cap | BPH | Urethral Stricture |
| Frequency of diagnosis n (%) | 33 (32.0%) | 52 (50.5%) | 18 (17.5%) |
| Mean age ± SD (Years) | 66.18 ± 7.05 | 62.85 ± 7.02 | 50.17 ± 17.87 |
| Age range (in years) | 55 – 84 | 48 - 76 | 19 – 93 |

**Table-3: Electrolytes levels of respondents**

| **Characteristics** | **Frequency (n)** | **Percent (%)** |
|---|---|---|
| **Potassium level (n=82)** | | |
| Normal | 67 | 81.7 |
| High | 14 | 17.1 |
| Low | 1 | 1.2 |
| **Sodium level (n=82)** | | |
| Normal | 77 | 93.9 |
| High | 4 | 4.9 |
| Low | 1 | 1.2 |
| | | |
| **Chloride level (n=83)** | 74 | 89.2 |
| Normal | 9 | 10.8 |
| High | 0 | 0.0 |
| Low | | |
| **Bicarbonate (n=84)** | | |
| Normal | 54 | 64.3 |
| Low | 30 | 35.7 |

**Table-4: Association between electrolyte status of respondents and causes of bladder outlet obstruction**

| **Characteristic** | **Causes of Bladder Outlet Obstruction** | | | **Test Statistics and value** |
|---|---|---|---|---|
| | Cap n(%) | BPH n(% | Urethral Stricture n(%) | |
| **Potassium** | | | | |
| Normal | 25 (89.3) | 33 (80.5) | 9 (68.2) | $X^2$=3.543 |
| Low | 0 (0.0) | 1 (2.4) | 0 (0.0) | DF=4 |
| High | 3 (10.7) | 7 (17.1) | 4 (30.8) | P=0.442* |
| **Sodium** | | | | |
| Normal | 25 (89.3) | 40 (97.6) | 12 (92.3) | $X^2$=3.060 |
| Low | 1 (3.6) | 0 (0.0) | 0 (0.0) | DF=4 |
| High | 2 (7.1) | 1 (2.4) | | P=0.429* |
| **Chloride** | | | | |
| Normal | 26 (92.9) | 38 (90.5) | 10 (76.9) | $X^2$=2.485 |
| High | 2 (7.1) | 4 (9.5) | 3 (23.1) | DF=2 |
| | | | | P=0.352* |
| **Bicarbonate** | | | | |
| Normal | 17 (60.7) | 30 (71.4) | (50.0) | $X^2$=2.333 |
| Low | 11 (39.3) | 12 (28.6) | 7 (50.0) | DF=2 |
| | | | | P=0.281* |

*Fishers exact P value. There is no statistical significant association between electrolyte status and causes of bladders outlet obstruction.

**Table-5: Association between age of respondents and electrolyte status**

| Characteristics | Age of respondents (years) | | Statistical tests and values |
|---|---|---|---|
| | Less than 50 n(%) | 50 and above n(%) | |
| Potassium<br>Normal<br>Low<br>High | <br>5 (83.3)<br>0 (0.0)<br>1 (16.7) | <br>62 (81.6)<br>1 (1.3)<br>13 (17.1) | <br>$X^2$=0.082<br>DF=2<br>P=1.000* |
| Sodium<br>Normal<br>Low<br>High | <br>6 (100.0)<br>0 (0.0)<br>0 (0.0) | <br>71 (93.9)<br>1 (1.3)<br>0 (0.0) | <br>$X^2$ = 0.420<br>DF=2<br>P=1.000* |
| Choride<br>Normal<br>High | <br>5 (83.3)<br>1 (16.7) | <br>69 (89.6)<br>8 (10.4) | <br>$X^2$=0.227<br>DF=1<br>P=0.509* |
| Bicarbonate<br>Normal<br>Low | <br>5 (83.3)<br>1 (16.7) | <br>49 (62.8)<br>29 (37.2) | <br>$X^2$=1.021<br>DF=1<br>P=0.414* |

*There is no statistical significant association between electrolyte status and the age of respondents.

## DISCUSSION

Bladder Outlet Obstruction is a major urological problem especially in the aging male population. It may be defined as impedance or blockage of urine outflow from the bladder into the urethra [1]. This obstruction may be due to functional or mechanical causes or both and can occur in both males and females. In males the presence of the prostate gland which could harbor both malignant and benign diseases is a well recognized culprit of bladder outlet obstruction. Pathologies in this gland account for more than 50% of all the causes of lower urinary tract obstruction and retention in maes [5].

This study and others conducted in this set of patients showed that Cap, BPH and Urethral Stricture are the major causes of BOO [2,6]. This condition usually result in lower urinary tract symptoms which may be obstructive or irritative or both in nature. When patient presentation to health care facility is late, complications may arise for which acute or acute-on-chronic, or chronic urinary retention is the most common [7]. Many other complications of BOO are in part due to complications of chronic urinary retention. These include recurrent urinary tract infection, bladder calculi, haematuria, urethrocutaneous fistula, damage to bladder and kidney failure. Others may be due to disease progression such as low back pain with paraparesis or paraplegia, weight loss and pathological fracture as in advanced cancer of the prostate.

Chronic urinary retention (CUR) as a complication of BOO has been well studied and may result in chronic kidney failure with attendant high mortality, decrease quality of life, increase health care expenditure and if unstreated may lead to end stage renal disease requiring dialysis or kidney transplantation [8].

Normal renal function provides a mechanism for eliminating toxic metabolites and maintaining homeostasis of fluid and electrolytes. Loss of normal function results in alteration in volume and electrolyte homeostasis and inadequate excretion of metabolites [9]. Pathophysiology of renal failure secondary to BOO occur most commonly in the setting of high pressure chronic urinary retention where there is usually high voiding detrusor pressure with low flow rate leading to persistently high pressure within the bladder causing retrograde pressure and bilateral hydronephrosis. This can result in varying degree of renal dysfunction [7].

This study is meant to highlight the pattern of electrolytes in the setting of BOO. Aside from BOO noted as a cause of renal function deterioration, degradation of renal function is extremely common among aging males leading to a suggestion that it is a natural concomitant of aging [10]. Ricardo *et al.* [11] reported that there was no significant statistical relation between these two conditions.

Hyperkalaemia was found in 17.1% and hypokalaemia in 1.2% of patients. Hypernatraemia was found in 6.2% and hyponatraemia in 1.2%. Chloride was high in 10.8% while bicarbonate was high in 35.7%. Association between the electrolyte status of respondents and causes of BOO showed that there is no statistical significance (P value>0.05) [Table 4]. This may be partly due to the fact that pre-existing electrolyte derangement in senile kidneys may not allow explanation of the degree of renal dysfunction vis-à-vis the electrolyte derangement superimposed by effect of BOO. Secondly, BOO may either lead to high pressure chronic retention (HPCR) or low pressure chronic retention (LPCR) where HPCR consistently leads to varying degrees of renal dysfunction as opposed to LPCR in which the bladder is floppy, more compliant with no increased pressure to the kidneys[7].

Thirdly, electrolyte status of an individual is influenced by the amount and type of fluids administered. The retrospective nature of this study could not allow assessment of the amount and type of fluid administered especially in those who were seen in accident and emergency department. Rule *et al*. [12] however reported that there was a cross-sectional association between signs and symptoms of BOO and chronic kidney disease in community dwelling men.

I did not also find any statistically significant association between age of respondents and their electrolyte status (Table 5). This is because the age of the patient is not tied to the severity of BOO which is the determinant of the rapidity and degree of renal impairment. Younger patients by reason of the nature of onset and the severity of the obstruction will present with worse renal function than older subjects with mild degree of outlet obstruction. In the study, majority of the patients presented with normal electrolyte values. This is not a consoling statement however, because a case of a particular electrolyte abnormality is strictly an individual affair which does not affect the status of another person and so this problem should be looked at in that perspective. Prevention is the mainstay of management by way of health awareness campaigns for early presentation to health facility and prompt referral to urologists before these complications set in.

Hyperkalaemia was encountered in 17.1% of the respondents. Renal failure is the most common cause of hyperkalaemia seen in emergency department [13]. The kidney is the main regulator of potassium concentration and hyperkalaemia is not a rare condition in hospitalized patient population especially those with renal disorder [14]. It is a life threatening potentially fatal condition mainly due to severe cardiac arrhythmias and other ECG changes, including asystole which can occur depending on the severity and rapidity of development as well as the presence of other electrolyte disorders and whether appropriate and prompt treatment had been carried out [15]. Low serum bicarbonate level was seen in 35.7% of the patients. Lower serum level of bicarbonate is associated with an increased risk of kidney disease progression. In a single centre retrospective study involving patients with or without kidney disease, the risk of chronic kidney disease progression was 54% higher for patients with bicarbonate levels <22meq/l compared with bicarbonate levels of 25-26meq/l [16]. There is a direct relationship between low serum level of bicarbonate and hyperkalaemia. In metabolic acidosis, more than one-half of the excess hydrogen ions are buffered in the cells. In this setting electro-neutrality is maintained in part by the movement of intracellular potassium into the extracellular fluid. Thus metabolic acidosis results in a plasma potassium concentration that is elevated in relation to total body stores. The net effect in some cases is overt hyperkalaemia, in other patients who are potassium ($k^+$) depleted due to urinary or gastrointestinal losses, the plasma $k^+$ concentration is normal or even reduced[17]. Hypo-and hypernatraemia was seen in 1.2% and 4.9% respectively and chronic kidney disease (CKD) is known to affect the ability of the kidneys to regulate water homeostasis[18] and hence the risk of developing both conditions increase with advancing stages of CKD. However, there is no population based study to show the incidence and prevalence of hypo-and hypernatraemia in patients with CKD.

Elevated serum chloride level was seen in 10.8% of respondents. This could be attributed to excessive saline infusion especially in those who were seen in accident and emergency for which adequate records were not accessed. It is clear that relatively small volumes of saline ($30mlkg^{-1}h^{-1}$) produce a hyperchloraemic acidosis with this acidosis being derived from hyperchloraemia and not from other causes [19-21]. Aside from this, it could also be caused by renal failure with proximal renal tubular acidosis where there is failure of bicarbonate reabsorption and distal renal tubular acidosis with failure of hydrogen ion secretion leading to hyperchloraemic acidosis. Elevated serum chloride has also been noted to have a negative effect on renal function causing vasoconstriction on renal vessels [22] leading to reduction in cortical perfusion and increase in systemic inflammation which is a well known risk factor for acute kidney injury [13].

In the light of this discussion, it is evident that bladder outlet obstruction may result in chronic kidney disease with electrolyte derangements that can further aggravate renal damage and so prompt recognition and treatment of causes of BOO is advocated.

## CONCLUSION

The pattern of electrolyte status in the setting of BOO is usually attended by varying degrees of derangement that impact negatively on the quality of life of the patients. Health awareness campaign targeting on men in this age bracket should be instituted emphasizing early presentation, early referral to urologists to checkmate the consequences of late presentation.

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