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Research Article

Pattern of Serum Creatinine and Urea in Patients Seen with Symptoms of Bladder Outlet Obstruction

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Abstract: Bladder outlet obstruction (BOO) is a major urological condition characterized by impedance of urine outflow from the bladder into the urethra. The causes are numerous and commoner in aging males. The result is renal impairment if not addressed promptly. The aim of this study was to highlight the pattern of serum creatinine and urea in our patients population during the period under study and to define any association between the causes of BOO, age of patients and their renal status. One hundred and three (103) case notes were retrieved, twenty (20) did not contain complete investigation results needed for the study and were excluded. Eighty-three (83) patients entered the study with requisite results. Age ranged from 19-93 years with a mean of 61.70 $\pm$ 11.139 years. Data on clinical history, physical examination, diagnostic and treatment modalities were collected and analysed. Serum creatinine was abnormal in 42.2% and urea in 24.1% of patients. There was neither statistical significant association between the renal status of respondents and causes of BOO nor their renal status and age at diagnosis.
Keywords: Bladder outlet obstruction, causes, renal status, age

INTRODUCTION

Bladder outlet obstruction (BOO) is defined as impedance of urine outflow from the bladder into the urethra[1]. It can be caused by a number of conditions notably Cancer of the Prostate (Cap), Benign Prostatic Hyperplasia(BPH) and Urethral Stricture which has been noted to be the commonest causes of bladder outlet obstruction [2]. Others causes are meatal stenosis, urethral diverticulum, urethral stones and malignances. BOO can lead to a constellation of complications in the urinary tract and eventually to renal failure and death if not addressed promptly.

Creatinine is produced in the muscles by non-enzymatic changes of creatine and phosphocreatinine. The liver has a momentous role in the assembly of creatinine through methylation of guanidine aminoacetic acid. It is produced at a relatively constant level and the plasma level therefore depends on the rate of excretion by the kidneys. Levels, however, are affected by age, gender, ethnic group, muscle bulk, ingestion of cooked meat, malnutrition and the use of some drugs e. g Trimethoprim. The plasma concentration alone is only a rough guide to renal function [3]. In human, some craetinine is secreted by the tubules and some may be reabsorbed so that when precise measurement of renal function is needed Glomerular filtration rate (GFR) is measured [4].

Urea is an organic compound playing a vital role in the metabolism of nitrogen-containing compounds [5]. It is excreted in the kidneys and serves to contribute to the establishment of the osmotic gradient in the medullary pyramids and to the ability to form a concentrated urine in the collecting ducts. The amount of urea in the medullary interstitium and consequently in the urine varies with the amount of urea filtered and also with dietary intake of protein.

Both serum creatinine and urea can be affected if the normal drainage of the urinary tract is impaired from any cause. BOO frequently lead to renal impairment. The pathophysiology of renal impairment starts at the level of the bladder causing increased bladder wall thickness and intravesical pressure. With increased wall thickness, there is kinking of the intramural portion of the ureters (Hockey stick deformity) and weakness of the vesico-ureteric valve, the result is bilateral vesico-ureteric reflux with attendant hydroureters and hydronephrosis. Persistent hydronephrosis will cause sustained increases in intra-pelvic pressure leading to destruction of the renal papillae, the nephrons and renal parenchyma ultimately resulting in impaired renal function [6]. Although both serum craetinine and urea are widely used as an index of renal function, serum creatinine exceeds GFR because of tubular secretion whereas, urea is usually lower due to tubular re-absorption. The mean of

creatinine and the urea clearance is a more accurate estimate of GFR and so of renal function than either is separately [7].

AIM AND OBJECTIVES

The aim of this study was to highlight the pattern of renal function vis-à-vis the serum levels of creatinine and urea in patients who presented with BOO and to associate these serum parameters with the causes of BOO and age at presentation.

PATIENTS AND METHODS

This is a retrospective study of all patients who presented with symptoms of bladder outlet obstruction from January 2014 to December 2014. Information on patient's biomedical data, symptoms, signs and their duration including the causes of bladder outlet obstruction were obtained from the case notes.

Results of serum creatinine and urea, prostate specific antigen (PSA), prostate biopsy where applicable, abdominopelvic and trans-rectal ultrasound scan, retrograde urethrocystogram and micturating cystourethrogram were extracted from case notes. Data was analysed using SPSS version 20.0.

RESULTS

Eighty three (83) case notes were reviewed. All were males and of the Christian faith. The ages of respondents ranged from 19-93 with a mean of 61.70 ± 11.14 years. The creatinine values ranged from 35.0 - 942.0mmol/l with a mean of 162.45 ± 155.44. The urea levels ranged from 1-26.7mmol/l with a mean of 6.08 ± 4.78. FBS ranged from 3.1+ 11.2 with a mean of 4.99 ± 1.52.

Table 1: Causes of Bladder Outlet Obstruction

Characteristics	Frequency	Percent
Cap	26	32.0
BPH	42	50.5
Urethral Stricture	15	17.5

Table 2 : Mean Age of Respondents Per Diagnosis

Characteristics	Type of Diagnosis		
	Cap	BPH	Urethral stricture
Age(Mean ± Standard Deviation)	66.18 ± 7.05	62.85+ 7.02	50.17 + 17.87
Age (Minimum-Maximum)	55-84	48-76	19-93

Table 3 : Renal status of respondents

Characteristics	Frequency	Percent
Creatinine level		
Normal	47	56.6
Low	1	1.2
High	35	42.2
Urea Level		
Normal	58	69.9
Low	5	6.0
High	20	24.1

Table 4 : Treatments Given to Respondents

Treatment Type	Frequency	Percent
Catheterization		
Yes	53	64.1
No	30	35.9
Dialysis		
Yes	2	1.9
No	81	98.1

Table 5: Association between renal parameters and causes of Bladder outlet obstruction

Characteristics	Causes of Bladder Outlet Obstruction			Test Statistics and values
	Cap n (%)	BPH n (%)	Urethral Stricture n (%)	
Urea				
Normal	20 (71.4)	29 (70.7)	9 (64.3)	X^2 7.03
Low	1 (3.6)	3 (7.3)	1 (7.1)	Df =4
High	7 (25.0)	9 (22.0)	4 (28.6)	P= 0.950*
Creatinine				
Normal	13 (46.4)	26 (61.9)	8 (61.5)	X^2 7.59
Low	0 (0.0)	0 (0.0)	1 (7.7)	Df =4
High	15 (53.6)	16 (38.1)	4 (30.8)	P= 0.165*

(*Fishers exact p-value). There is no statistically significant association between renal parameters and the causes of bladder outlet obstruction(p>0.05)

Table 6: Association between Age of respondents and renal status

Age	Levels of Renal parameters			Test Statistics and values
	Normal n (%)	Low n (%)	High n (%)	
		Levels of Urea		
				X^2 =1.074
				DF=2
Less than 50 years	5 (8.6)	0 (0.0)	1 (5.0)	P= 0.585
50 years and above	53 (91.4)	5 (100.0)	19 (95.0)	
		Levels of Creatinine		
				X2 =1.890
Less than 50 years	5 (10.6)	0 (0.0)	1.(2.9	DF=2
50 years and above	42 (89.4)	1 (100.0)	34 (97.`	P=0.288 *

(*Fishers exact p-value). There is no statistically significant association between renal parameters and the ages of respondents (p> 0.05)

DISCUSSION

Bladder Outlet Obstruction (BOO) results from increased resistance created by pathologies at the bladder neck and beyond. Causes of BOO are quite numerous and incidence vary from one region to another [1].

The commonest cause was BPH with about half the respondents diagnosed with the condition.

A similar finding was reported by another study by Amu et al [8], Ricardo et al [9] noted BPH as the most common cause of obstructive uropathy. In another study, Amugo et al [2] found out that the majority of patients in their study who presented with renal impairment had a diagnosis of BPH..

The mean age at diagnosis showed a clinical relevance being highest in Cap patients (66.18 $\pm$ 7.05 years) with a median age of 68.5 years slightly higher than 66 years in National Cancer Institute Guidelines [10]. Patients with cancer of the prostate had been known and noted in many studies to be relatively older than patients with BPH and Urethral Stricture at diagnosis stated respectively as over 70, 66 and 50 years [11]. This age differences was also seen in our study.

Renal function has been largely studied with the use of serum creatinine, urea and creatinine clearance [12]. However some limitations have been noted; serum urea level as well as urea in the medullary interstitium and consequently in the urine vary with the amount of urea filtered and also with the dietary intake of protein. It is also affected by functional state of the liver for its production and rate of protein breakdown, and so it is not a specific measure of renal function [12]. Creatinine on the other hand has a relative accuracy in measurements of renal function but its serum level may be affected by age, gender, ethnic group, muscle bulk, ingestion of cooked meat, malnutrition and the use of some drugs like Trimethroprim. Because of these, its plasma concentration is only a rough guide to renal function[3]. Clinicians now also rely more on the ratio of plasma urea and creatinine in assessing renal status [12]. The normal blood urea nitrogen (BUN) to serum creatinine ratio is 10-20:1 [13]. Although GFR is the best marker for measurement of renal function, serum creatinine concentration is widely interpreted as a measure of renal function in clinical practice [14]. This

is true especially in poor resource centres where sophisticated instruments and expertise are lacking, serum creatinine serves as a marker for renal function.

This study also shows that there is no statistically significant association between age and the renal status of respondents (P value > 0.05). Age – related loss of renal function has been recognized for decades. This is important in many older subjects. Hoang *et al* [15] compared renal function in subjects under the age of forty (40) years and those over the age of fifty-five (55) years and demonstrated a reduction. The pathophysiology of bladder outlet obstruction as it affects renal function has been believed by some clinicians to be due to a natural concomitant of aging [16]. Studies also noted that the acceleration of age-related decline in renal function may be associated with hypertension [17], dyslipidaemia [18], atherosclerotic disease [19], increased levels of advanced glycosylated end-products [20], male gender [21] and all these changes appear in the aging population just like bladder outlet obstruction.

The causes of bladder outlet obstruction in this study are principally Cap, BPH and Urethral Stricture. Amugo *et al* [2] in their survey noted Cap, BPH and Urethral Stricture as the leading causes of BOO. Our study shows no statistically significant association between renal status and causes of BOO. This may also be related to the fact that the aetioliogies of BOO in this study are largely seen in the middle and aging populations likely to have a concomitant age-related decline in renal function. Also, the severity of renal impairment relates more to the degree of obstruction irrespective of the cause. Hill *et al* [22] did not find any relationship between the duration of symptoms of BOO and renal functions.

Renal function was abnormal in about 36.75% of the respondents. 64.1% had urethral catheterization as a temporizing measure to relief the obstruction and aid eliminate the retained metabolic waste from the system. Only 2 patients(1.9%) had haemodalysis whose serum creatinine exceeded 700mmol/l.

Causes of BOO lead to deterioration of renal function and if not arrested early may cause end-stage renal disease (ESRD) in which patients depend on haemodialysis and renal transplant with their attendant complications. Untreated ESRD will ultimately lead to death. The need for prevention, early recognition and prompt treatment of causes of BOO becomes imperative to forestall its complications. Health awareness campaigns directed at susceptible aging male population to detect early prostate diseases can not be over emphasized. Health education on the prevention, early presentation to health facility and appropriate diagnosis and treatment of sexually transmitted diseases can reduce the incidence of Post-inflammatory urethral stricture which is one of the major causes of BOO. Other causes of urethral stricture such as iatrogenic urethral injuries from instrumentation and surgeries, pelvic fracture with urethral injury from road traffic accidents also have their preventive rules which should be obeyed.

These lend support to the fact that renal deterioration from causes of BOO can be prevented and or deferred given the much knowledge and awareness coupled with emerging and teaming population of clinicians training in the area of urological pathology and management.

CONCLUSION

Bladder outlet obstruction is a major health issue of the aging male population especially when it is caused by prostatic diseases and urethral stricture. Renal impairment is a well recognized complication of late presentation, worst still in untreated cases. In our study renal status had neither statistically significant association with age of the patients nor with causes of BOO. This however does not remove the clinical significance of these variables with the renal function of our patients. The way forward is a proactive effort both on the part of the patients and the clinicians especially the first contact physicians in the diagnosis and prompt referral of patients to the urologists.

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