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Status of Renal Parameters in Prostatic Enlargement.

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ABSTRACT

Benign prostatic hyperplasia (BPH) is a well known condition characterized by prostate growth accompanied by lower urinary tract symptoms (LUTS). Both are highly prevalent and major conditions among older men. The aim of our study is to find out renal status in prostate enlargement patients by investigating renal parameters. To assess levels of kidney function tests, acid phosphatase and serum amylase in prostate enlarged patients and compare with control subjects. Fifty six outpatient with enlargement of prostate, attending the department of internal medicine (Urology) Krishna hospital, karad, Maharashtra were selected to participate in this study compared with 66age matched healthy males as control. The mean values of biochemical parameters were observed and found significant in BUL, BUN, creatinine, acid phosphatase and amylase (p<0.01, p<0.001, p<0.0001) as compared to control. Decreased uric acid significantly as compared to conrol is also observed. On the basis of our study we could come to the conclusion that, in enlargement of prostate, levels of BUL, creatinine, serum amylase, acid phosphatase changes significantly which could lead to kidney damage in severe cases.

Keywords: Benign prostatic, Blood urea, Creatinine, Uric acid, Acid phosphatase, Amylase

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INTRODUCTION

Benign prostatic hyperplasia (BPH) is a well known condition characterized by prostate growth accompanied by lower urinary tract symptoms (LUTS). Both are highly prevalent and major conditions among older men [1].

Pathologically, BPH is due to cellular proliferation of the epithelial and stromal elements.within prostate gland. These changes begin histologically in the third decade of life and clinically fifth decade of life. When prostate enlargement occurs increased reistance in the proximal urethra may limit urinary flow during micturition often resulting in pathophysiologic changes in the bladder wall.

Consequently, LUTS due to prostatic obstructuion are inseparable from symptoms due to bladder detrusor dysfunction [2, 3]. Clinically BPH is distinguished by progressive development of LUTS and symptoms are variable that ranged from nocturia,incomplete emptying, urinary hesitancy, weak stream frequency and urgency to the development of acute urinary retention.[4-6].

For detection of renal function, it is well accepted today that bladder outlet obstruction due to BPH might cause hydronephrosis and renal failure. Most studies have found that the incidence of azotemia in men with BPH varies from 15-30%[7,8].

The aim of our study is to find out renal status in prostate enlargement patients by investigating renal parameters.

MATERIALS AND METHODS

Study design

Fifty six outpatient with enlargement of prostate, attending the department of internal medicine(Urology) Krishna hospital, karad, Maharashtra were selected to participate in this study compared with sixty six age matched healthy males as control.

These patients were selected with some clinical and biological information Patients who suffered from cardiac diseases, diabetes, hypertension, dyslipidemia, smokers, alcoholism, active acute infections and metabolic disorders were excluded.

All patients were diagnosed according to international prostate symptoms scores(IPSS). Diseased individuals were undergoing per rectal examination and ultrasound of the prostate[9]. Blood samples were collected from all subjects (diseased and healthy). 10 ml of venous blood were drawn in plain centrifuge in order to get the serum and kept all samples in deep freezing -20°C till time of analysis.

Biochemical parameters

For estimation of creatinine, uric acid, urea, amylase and acid phosphatase, enzymatic kits (Erba, Mannheim,XL system packs, spine react) were used and measured on fully autoanalyser.

Jaffe's reaction is a colorimetric method used in clinical chemistry to determine creatinine in blood and urine. Creatinine reacts with picric acid in alkaline media such as sodium hydroxide solution and formed reddish orange color called creatinine picrate[10] . Uric acid was assessed by enzymatic method, depending upon conversion of uric acid to allantoin by uricase[11]. Urease liberates ammonia which reacts with compound[12]. In presence of glutamate dehydrogenase and reduced NADH, ammonia combines with α -keto glutarate to produce L- glutamate.

Acid phosphatase

Acid phosphatase determined by kinetic method using α -napthyl phosphate (Hill mann method). α -napthol reacts with diazotized compound forming a colour with a maximum absorbance at 405 nm [13].

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Amylase

 α -Amylase activity measured by CNPG method. 2-chloro-4nitrophenol β -1-4 galactopyranosylmaltotrioside is a direct substrate for determination of α -Amylase activity,which does not require the presence of ancillary enzymes. The rate of 2-chloro-4nitrophenol formation can be monitored at 415nm and is proportional to the α -Amylase activity [14].

Statistical analysis: Analysis is done by using two tailed unpaired 't' test and set to p<0.05.

RESULTS

Table 1: Biochemical parameters in patients of prostatic enlargement and controls.

Parameters	Controls	Subjects
	n=66	n=56
Blood urea level	24.23±6.51	31.64±16.92**
Blood urea nitrogen	11.32±3.04	14.78±7.90*
BUN/CREAT Ratio	11.0± 3.3	10.9±4.3
Uric Acid	5.99±1.54	5.34± 1.55*
Serum creatinine	1.06±0.17	1.41±0.82***
Acid phosphatase	2.68±1.53	3.36±1.58*
Amylase	59.19±14.66	77.63±35.86***

*p<0.05, **p<0.01, ***p<0.001

DISCUSSION

The mean values of biochemical parameters were observed and found significant in BUL, BUN, creatinine, acid phosphatase and amylase (p<0.01, p<0.05, p<0.001, p<0.05, p<0.001) as compared to control subjects. Decrease uric acid significantly (p<0.05) as compared to control is also observed. (Table 1)

Benign prostatic hyperplasia is non malignant condition, is mostly prevalent in older man and is reported to be a major cause of lower urinary symptoms (LUTS)[15].

Our study indicates that blood levels of urea, creatinine, BUN higher in prostate enlargement suggesting involvement of kidney in this condition [16].

Significant differences in the mean values of blood urea, marked in prostate disorders in general compared to controls, BUN and BUN/ creatinine ratio was highly significant in cancer of prostate and BPH in particular, amongst the various disorders of prostate, when compared to controls. In support to this observation, it has been stated earlier that many patients with kidney disease responded to surgical treatment of BPH [17].

On the basis of our findings we conclude that patients with prostate disorders are likely to progress into renal dysfunction.

Increased acid phosphatase observed in prostatic enlarged patients as compare to controls. High acid phosphatase observed in preoperative patients relative to the control value may arise due to enlargement of the prostate gland, leading to increased synthesis or expression of this enzyme. Acid phosphatase activity in carcinoma of the prostate is three times above normal. When the magnitude of the increase is taken into consideration, changes in serum acid phosphatase activity can be used potentially to differentiate between BPH and carcinoma of the prostate. Acid phosphatase therefore has the dual potential of being used to differentiate prostate enlargement due to BPH or prostate carcinoma and as a prognostic marker of benign prostate hypertrophy [18].

In this study uric acid level decreases (Table 1) Sampatkumar et.al. observed that uric acid level raised in prostate carcinoma patients it may be due to enhanced turnover rate of nucleic acids may be due to



rapidly growing malignant tissue, increased tissue breakdown after treatment of malignant tumors, increased tissue damage due to trauma and raised rate of catabolism. They also observed that uric acid levels are raised in carcinoma of prostate where it is normal in other disorders.[19]

Although the exact etiology of BPH is not known it seems that the natural history and evolution of benign prostatic enlargement ends up in urinary obstruction causing degradation of renal function over time.

Increase amylase activity in prostate enlargement patient is observed (Table 1). Hanafy et al observed that increased serum amylase levels in prostatic cancer or in benign prostatic hypertrophy has not been previously reported. This association could explain by the increased amylase in prostatic tissue due to the increased cell replication rate, prostatic amylase could be increased to split more polysaccharides into simple sugar, which is necessary for cellular energy and nutrition[20].

CONCLUSION

On the basis of our findings, we conclude that patients with prostate enlargement are likely to progress into renal dysfunction

REFERENCES

- [1] Eman alsaadi . Kerbala journal of Pharamaceutical Sciences. 2013; 5:24-33.
- [2] Bostwick DG, Cooner WH, Denis L, Jones GW, Scardino PT, Murphy G. Cancer 1992;70:291-301.
- [3] Gat Y, Gornish M, Heiblum M and Joshua S. Andrologia 2008;40:273-281,
- [4] de la Rosette JJ, Alivizatos G, Madersbacher S, Perachino M, Thomas D, Desgrandchamps F, de Wildt M. Eur Urololgy. 2001; 40(3):256-264.
- [5] Irani J, Brown CT, van der Meulen J, Emberton M, BJU Int. 2003; 92(9):937-42.
- [6] Untergasser G, Madersbacher S, Berger P. Exp Gerontol. 2005;40:121–8.
- [7] Sacks SH, Aparicio SA, Bevan A, Oliver DO, Will EJ, Davison AM. BMJ. 1989; 298:156-9.
- [8] Roehrborn CG: (3rd IC BPH).Geneva, 1996; 167-254.
- [9] Perry J. Clinical Practice development, Continence uk, 2007; 1(4):21-29.
- [10] Myers, GL, Greg Miller, W. Coresh, J, Fleming, J, Greenberg, N. et al. Clinical Chemistry 2006; 52: 5-18.
- [11] Bezkorovainy A, Searcy, RL. Diagnostic Biochemistry. Mc Graw Hill, New York, NY, 1969;328.
- [12] Textbook of clinical Chemistry.BurtisCA and Ashwood ER(EDs) Tietz Second Edition, WBSaunders Company,1994.
- [13] Kaplan A etal. Clinical chemistry. The C.V. Mosby Co. St Louis. Toronto. Princeton 1984; 1079-83.
- [14] Ziva, JF and Pannall, PR Clinical chemistry in diagnosis and treatment. Lloyd London 1979;, Chapter XV:341-2.
- [15] Yeon won Park, Seung ki Min, Jun Ho Lee, World J. Mens health 2012; 30(3):183-88.
- [16] Reshma K,Sudha K, Poornima A Manjrekar, Madan Gopal, Souparnika, Yigesh K and Ravikiran AK, International journal of Biomedical and Advance research, 2014; 05(09), 415-17.
- [17] Andrew D. Rule Derba J. Jacobson, Rosebud O Roberts, Cynthia J. Girman Michala E McGree, Michael M Lieber and Steven J. Jacobsen. Kidney international, 2005;.67, 2376-82.
- [18] Hassan G, Gregory U, Donatien G, Mahmoud A. The Internet Journal of surgery. 2004; ISPUB. COM .Volume 6 number 1.
- [19] Bhagylakshmi A, Sampatkumar V, Ramadevi, Rama rao J, Harini . International Journal of Pharmacy and Biological Sciences, 2012; 2, 117-22.
- [20] Hanafy MH, Gursel, EO Veenema, RJ Urology April 1973 Volume 1, Issue 4,; 372-73.

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