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THE ROLE OF OBESITY IN ALTERATION OF PROSTAT SPECIFIC ANTIGEN AND ACID PHOSPHATASE IN ADULT YOUNG SUDANESE MALES

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ABSTRACT

Background: serum prostat specific antigen(pSA) and acid phosphatase(ACP)adiagnostic biomarker of prostate problem and is possibly associated with obesity. The purpose of this study to detect the role of obesity in alteration of (PSA) and(ACP) In adult young obese male and other with normal weight as control with different body mass indx. **Methodology:** Acase control study involving 40adult young sudanese obese males(BMI>30kg\m), the mean age between 18-40years, control group was included 40 healthy volunteer males with normal weight(BMI20-25kg\m) were analyze of ACP by spectrophotometr and PSA by using ELISA. **Result**: showed signficant decrease in PSA In study group comparint to the control group,that is due to heamodilution and metabolic syndrome. negative correlation between age and ACP (p value0.00) and insignificant change in PSA(P value 0.07). highly negative **Correlation(t 0 .0) PSA and ACP with increase of BMI. **Conclusion**: The level of prostate specific antigen and acid phosphatase decrease with obesity that may casuse mask to aerly detecation of prostate cancer.

KEYWORDS: prostate specific antigen, body mass index, Acid phosphatase,obesity, prostate cancer.

INTRODUCTION

Obesity is chronic disease and is second leading cause of preventable death, is major risk factor for hypertension, cardiovascular disease, type 2diabetes mellitus and some cancer in both men and women, sleep apnea, osteoarthritis, infertility.[1]

One-third of all cases of high blood pressure are associated with obesity and obese individuals are 50% more likely to have elevated blood cholesterol levels. type2 diabetes mellitus account for nearly 90%of all cases of diabetes. About 88-97% of type 2diabetes cases diagnosed in overweight people are a direct result of obesity.

over weight and obesity also increase s risk of coronary heart disease thus, excess weight is an established risk factor for high blood pressure and high cholesterol level and gallbladder disease.[1]

Susceptible to develop obesity partly due to environmental factors, family tendency and hypothalamic dysfunction(2). one of the environmental factors that could render an individual more susceptible to develop obesity could be prenatal nutritional environment(2)

The prostate cancer is leading of death among men and is a second most common cancer worldwide.(3)

The relationship between obesity and prostate cancer (PC) derives from the possibility that there could be modifiable risk factor this common and sometimes fatal disease. Several studies report that obesity increases the risk of advanced PC,PSA recurrence following treatment and prostate cancer mortality, weight gain since age18 years has similarly been associated with PC Mortality. however, the duration of PC Survivorship has increase since broad utilization of PSA test and most patients are diagnosed with localized or low –grade prostate cancer. the relationship between obesity and PC risk in the PSA are remains in question, with several studies suggesting that obesity is associated with lower risk of localized or low-grade PC. similarly, type 2 diabetes is strongly associated with obesity and study report either higher or lower PC risk with continued follow-up aftera T2DMdignosis[4] Despite the apparently abundant BMI literature, few study addressed the direct relationship between BMI and prostate cancer detection, as will as between BMI and PSA levels prior to prostate cancer is dignosis. the latter relationship is important, as same investigation indicated that elevated BMI may be associated with lower circulating serum PSA. thus men with high BMI may be excluded from screen or early detection based on spuriously low perceived risk of PC[5]

There have been several large studies that have reported obesity at risk factor for PC[6] though other have no association[7] or even a protective affect at all.[8]

The main casus of increase ACP is prostate disease with development of PSA as the major serum test for prostate, ACP has become less popular for use in PC, although, with the availability of newer immunochemical method, its use in diagnosis of PC is being reevaluate.[9]

The recent studier which concerned the relationship between the obesity, PSA and ACP are not enough to prove that what is the affect of obesity on them? because of the important of using of PSA and ACP enzyme as marker for many problems in prostate, our study looking for the affect of obesity on them to detect if its lowering or increasing them, or have no effect at all Several hypotheses have been proposed to explain the apparent association between obesity and PC. As a protective factor in incidence, obese individuals have been reported to have lower PSA level.[10]

Obese men also have higher plasma volume compared with normal-weight men, which has been suggested to dilute the amount of PSA, ACP in plasma, reducing its concentration in plasma. In addition obese individuals have been shown larger prostates. but some study is showed obesity does not make pressure or enlargement on prostate gland.[11,12]

Recent studies across Asian populations have, however, demonstrated that PSA is age-dependent and increases significantly for every decade of life.[13] It has also been reported that PSA levels are negatively associated with body mass index (BMI=weight by kg\highest by meters.[2] Over25 is called over weight by international stander and people with BMI30 and above are considered obese[14], primarily as result of a hemodilution effect caused by increased plasma volume.[15, 16]

MATERIALS AND METHODS
These study was carried in Khartoum state A case control cross sectional study was included 40 adult young Sudanese obese male(BMI>30), mean age of them between18-20 years, control group was included 40 healthy volunteer males with normal weight (BMI20-25) whose mean age should be matched, the subject had nonsmoker, has no metabolic disease, no recent history of surgery or trauma were been included. Furthermore the subject with previous history of malignancy, infection, hypertension and DM were been excluded. Prepared questionnaire was used to collect the subject data including their past medical history, smoking status, family & clinical histories. All the patients were asked to sign an informed consent prior inclusion in the study. The laboratory tests were all performed under controlled condition. Acid phosphates was estimated by spectrometry (Humbler 200) and PSA estimated by using ELISA technique (plate reader washer).

RESULTS
Table 1: Comparison between the control group and the study group

	PSA Mean	SD	T	P value
Control	6.41	5.30396	4.018	0.034
Case	2.92	1.093451		

BMI and PSA: significant changes

Significant changes
The mean level of PSA were found significant decrease in case compared to control group
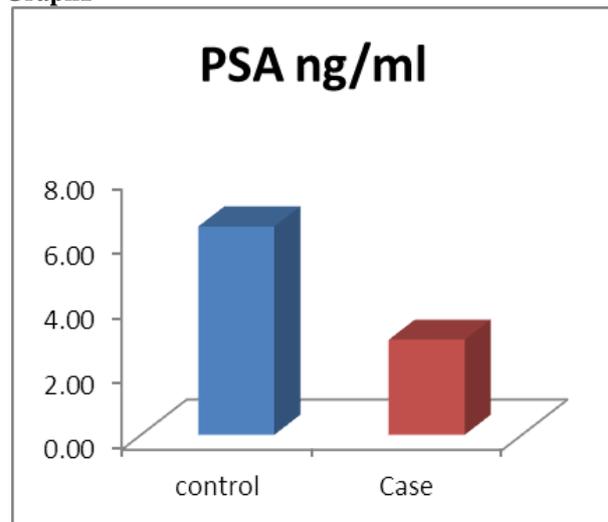
Graph1

Table 2: Comparison between study group and control group according to age:

	ASP	PCA
Age	-0.017[ns]	-0.055[**]
P value	0.07	0.00

The mean level of ACP: significant correlation at p value 0.00.

PSA: no significant change (p value 0.07).

Table 3: comparison of BMI, PSA and ACP On study group according to type of obesity

	Number	BMI	PSA ng/ml	ACP U/L
Obesity	34	30-35	2.89	2.08
Fatal obesity	26	>35	2.67	1.95
P value	-	0.00	0.08	0.03

The mean level of the BMI was found significant increased (p value: 0.00) was increase of obesity, while the mean value of ACP was found significantly decreased (p value: 0.03) and the mean level of PSA insignificant decreased (p value: 0.08).

Table 4: Comparison OF BMI and ACP between the control group and the study group:

	ACP			
	Mean	**SD**	**T**	**P value**
Control	11.55	1.986493	19.59	0.00
Case	4.00	1.358732		

BMI: significant change

ACP: significant **chan**ge

DISCUSSION

The study involved 40 young health obease sudanese male in addtion to40 health normal weigh males. The result of table 1 and fig 1 showed signficantdecrease in level of PSA and ACP in study comparint to control group, that me be due to heamodultion and metabolic syndroum these reasult agreed with astudy of Choi et al.[15]

The result of table 2 showed insignificant change (p value >0.05) in PSA reasult between age and significant change in ACP.

The result of table 3 showed iinsignificant deascres of ACP and PSA with increase of BMI. That mean negative correlation between them and BMI.these result agree with study of Richard et al.[16]

The result of table4 showed significant change in BMI and ACP.

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CONCLUSION

The level of prostate specific antigen and acid phosphatase decrease with obesity that may casuse mask to aerly detecation of prostate cancer.

There is propotional relationship between ACP and PSA.

REFERENCES

1. Hillier TA, fosse S, Balkau B,simon D,Eschwege E,fagot-Campagna A(2006) weight, the metabolic syndrome and coronary heart disease in type 2 diabetes:assocciations among anational french sample of adult with diabetes-the ENTRED study.J cardiometabSyndr 1: 318-325.
2. spalding KL, Amer E, Westermark PO,bernard S, Buchholz BA, Bergmann O, Blomqvist L hoffstedt J,Naslund E,britton T,concha H,Hassan M,Ryden M,frisen J,Amer P(2008) Dynamics of fat cell turnover in humans.Nature 453: 783-787.
3. Ferlay J, Shin HR, Bray F, Forman D, Mathers C, Parkin DM. Estimates of worldwide burden of cancer in 2008: GLOBOCAN 2008. Int J Cancer.
4. Jemal A.,Siegel R.,Ward E.cancer statistics, 2009. CAcancer J. Clin 2009; 59: 225-249.
5. Herbert Augustin.obesity and prostate cancer –An ambiguous relationsship.European journal of cancer, 2007-05-01, volume 43, issue7, pages 1114-1116.
6. Bushemeyer WC, III, freedland Sj.obesity and prostate cancer:Epidemiology and clinical. Andersson SO, wolk A, Bergstrom R, Adami HO, Engholm G, Englund A,Nyren O.body siz and porstate cancer: A20 year follow up study among 135006swedish construction workers.joournal of national cancer institute 1997; 89: 385-38.
7. NIlsen TI,et al .cancer casuses control. 1999.
8. Harper PS, reasearch samples from famillies with genetic disease :aproposed code of condcuct.Br Med J, 1993; 305: 1391-13993.
9. Kubota Y, Seike K, Maeda S, Shinohara Y, Iwata M, Sugimoto N. Relationship between prostate-specific antigen and obesity in prostate cancer screening: analysis of a large cohort in Japan. Int J Urol. 2011; 18: 72–75. doi: 10.1111/j.1442-2042.2010.02667.x.
10. cynthia C.cherneky,and Barbar J.B.laboratory tests and diagnostic procedures, 6th edition. saunders 2013; 842-951.
11. Rosty C; christa L;Kuzdzal S.identification of hepatocarcinoma-intestine-pancrease\pancreatitis-associted protein 1 as abiomarker for pancreatic ductal adenocarcinoma by proten biochip technology. cancer Res. 2002; 62.
12. Chia SE, Lau WK, Cheng C, Chin CM, Tan J, Ho SH. Prostate-specific antigen levels among Chinese, Malays and Indians in Singapore from a community-based study. Asian Pac J Cancer Prev. 2007; 8: 375–378.
13. Choi HC, Park JH, Cho BL, Son KY, Yoo YJ, Kwon HT. The illusion of prostate-specific antigen decline in patients with metabolic syndrome and insulin resistance. BJU Int. 2011; 108: 1756–1761. doi: 10.1111/j.1464-410X.2011.10225.
14. Choi H;C;Por J;A;Cho B;L.et al. Int. braz j urol.vol.37.No3.Rio de jane iro. May-june 2011; 16.
15. Richard K.D; David L. S;Dreick N.N.Efects of body mass index and age on prostate specific antigen. British journal of medicine and medical research. 2014; 18: 3514-3528.
16. Richard K.D; David L.Dreick N.N.Effect of body mass index and age on prostat specific antigen .British journal of medicin and medical reasearch 2014; 18: 3514-3528.