Available online at www.scholarsresearchlibrary.com

Scholars Research Library

Scholars Research Library

Annals of Biological Research, 2011, 2 (2) : 65-71
(http://scholarsresearchlibrary.com/archive.html)

ISSN 0976-1233
CODEN (USA): ABRNBW

Correlation between Packed Cell Volume and Body Mass Index in Hypertensive and Normotensive Subjects

[1*]Akinjide M. Akinnuga, [2]Olubayode Bamidele and [3]Ijeoma C. Chukwuebuka

[1]Lead City University, Ibadan, Oyo State, Nigeria
[2]Bowen University, Iwo, Osun State, Nigeria
[3]Madonna University, Okija, Anambra State, Nigeria

ABSTRACTS

In several reports, packed cell volume (PCV) and body mass index (BMI) have been identified as risk factors that significantly contribute to blood pressure increase. Therefore, there is necessity to investigate the correlation between PCV and BMI in hypertensive and normotensive subjects. In this study, a total of 192 subjects of age between 19 and 70 years were examined, 79 subjects (40 males and 39 females) were hypertensive and 113 subjects (61 males and 52 females) were normotensive. In all subjects, blood pressure, body weight and height were measured via comfort automatic blood pressure monitor, Hasson emperor weighing scale and meter rule respectively. PCV also was measured through an automated haematology analyzer. There was a positive correlation between BMI and PCV in hypertensive subjects (male and female) but only significant in male subjects ($r = 0.306$, $P<0.05$) thus PCV has a significant positive linear correlation with BMI in hypertensive male subjects. Also, the linear positive correlation between PCV and BMI was not significant in both male and female normotensive subjects ($r = 0.088$ and $r = 0.0288$ respectively, $P<0.05$) unlike in hypertensive subjects where it was only significant in male but not significant in female ($r = 0.0265$, $P<0.05$). It can be suggested that there was close correlation between PCV and BMI (especially male subjects); this correlation increases the risk of hypertension, therefore primary preventions of hypertension by monitoring PCV and BMI in hypertension-prone subjects are considered important.

Keywords: packed cell volume, body mass index, hypertension, normotension, hematocrit.

INTRODUCTION

Packed cell volume (Hematocrit) and body mass index estimations are essential risk factors that have been traceable to the etiology of cardiovascular diseases especially hypertension [1, 2, 3 4, 5]. Also, packed cell volume and body mass index have consistently been associated with an

increased risk for type 2 diabetes mellitus [6, 7, 8, 9, 10]. In addition, it has been reported that there was a significant relationship in approximately 7,000 men between packed cell volume and the development of non-insulin-dependent diabetes mellitus, independent of age, body-mass index (BMI), smoking, physical activity, high-density lipoprotein (HDL)-cholesterol, and systolic blood-pressure [11].

However, packed cell volume and body mass index are risk factors for concomitant diseases such as diabetes mellitus and hypertension, therefore more attention has to be given to the two factors in hypertensive and non-hypertensive individuals. Packed cell volume (PCV) is the single most important determinant of whole blood viscosity and body mass index is an indicator for total body fat determination, thus increased blood viscosity and increased body fat storage have been reported in several studies to contribute immensely to blood pressure increase [2, 4, 12, 13, 14, 15].

Interestingly, according to the above previous research works, works have been done on correlation between BMI and blood pressure or PCV and blood pressure but there are scarcity of information on correlation between PCV and BMI.

This present study was aimed at investigation of the correlation between the determinants of blood viscosity (PCV) and total body fat distribution (BMI) in hypertensive and normotensive subjects.

MATERIALS AND METHODS

This study was carried out at Imo State University Teaching Hospital (IMSUT) and St Domain's Hospital, orlu, Imo state, Nigeria. A total of 192 subjects examined in this study were all Igbo ethnic group of Imo state origin, not known to come from any definite work of life thus no consideration was given to variations based on occupation.

Out of 192 subjects, 79 subjects (40 males and 39 females) were clinically diagnosed to have solely hypertension with no other disease conditions, and 113 subjects (61 males and 52 females) were not clinically diagnosed to have any disease condition including hypertension.

The age of the selected population of the subjects ranges between 19 and 70 years [1].

Blood pressure measurement

Blood pressure (diastolic blood pressure, DBP and systolic blood pressure, SBP) of the subjects was measured using comfort automatic blood pressure monitor. Blood pressure was measured three times at least ten minutes after arrival to the hospital. The data obtained were used to classify the subjects as hypertensive and normotensive subjects.

Also, the hypertensive subjects were confirmed to have hypertension based on more than one visit to the hospital for treatment of hypertension via medical records.

Body mass index (BMI) measurement
The body weight and height of the subjects were measured by Hasson emperor weighing scale and meter rule respectively, with light clothing and without shoes.

BMI was calculated thus:

$$BMI = \text{weight (kg)}/\text{height (m}^2\text{)}.$$

Packed cell volume (PCV) measurement
Blood samples were collected by cubital venepuncture from all of the subjects using 5ml disposable syringe and the blood samples were dispensed into EDTA anticoagulant bottle and refrigerated at -20^0c before analysis. PCV was estimated using an automated haematology ANALYZER KX-21N, made by sysmex Japan. The sysmex KX-21 is an automatic multi-pair blood cell counter for in vitro diagnostic use in clinical laboratory. It performs speedy and accurate analysis of blood parameters and detects the abnormal samples. The automated haematology analyzer reading correlated well with readings by the standard manual methods [16].

Statistical Analysis
All data obtained were presented as mean±SEM and analyzed by Pearson correlation via SPSS software. Student's t test was also used to determine the significance of the correlation coefficient.

RESULTS

Normotensive Subjects
Table 1 showed the distribution of BMI, PCV, DBP and SBP in normotensive subjects. As shown in the table, BMI was 25.7±0.4kg/m^2 and 25.9±0.5kg/m^2 in female and male respectively. The PCV was 39.2±0.9% and 41.7±0.8% in females and males respectively. The SBP and DBP in female and male subjects did not change with BMI and PCV.

As in hypertensive subjects, there was positive correlation between BMI and PCV in normotensive subjects. The linear positive correlation was not significant in both male and female normotensive subjects as shown in figure 1 and figure 2.

Table 1: Distribution of BMI, PCV, DBP and SBP in male and female normotensive subjects

Parameters	Females	Males
BMI (kg/m^2)	[a]25.7±0.4	[a]25.9±0.5
PCV (%)	[a]39.2±0.9	[a]41.7±0.8
DBP (mmHg)	75.6±1.2	78.8±1.2
SBP (mmHg)	122.7±2.6	125±1.5
Sample Size (N)	52	61
Correlation Coefficient (r)	[a]0.0288	[a]0.088
Calculated t value (tcal)	[a]0.207	[a]0.6896

[a]*Insignificant linear positive correlation between PCV and BMI, P<0.05*

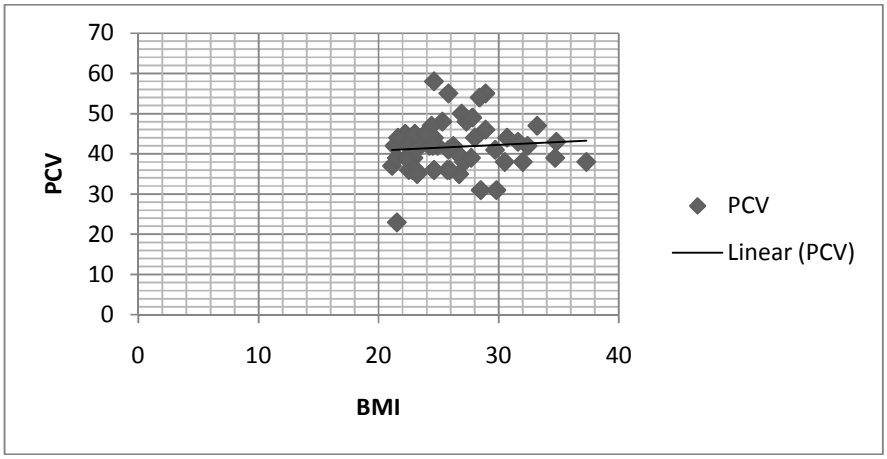

Figure 1: Scatter diagram showing correlation between BMI and PCV in normotensive males (n=61, r=0.088)

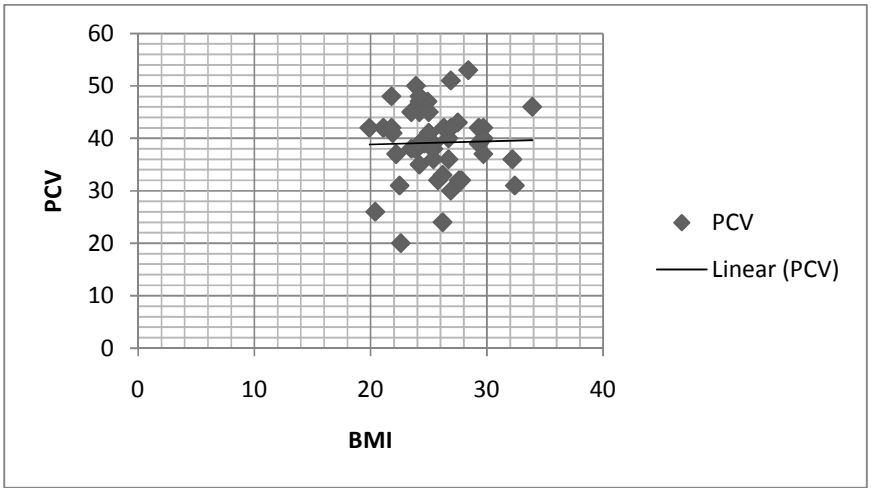

Figure 2: Scatter diagram showing correlation between BMI and PCV in normotensive females (n=52, r=0.0288)

Table 2: Distribution of BMI, PCV, DBP and SBP in male and female hypertensive subjects

Parameters	Females	Males
BMI (kg/m^2)	[a]28.7±0.8	[b]28.9±0.7
PCV (%)	[a]44.4±0.9	[b]48.7±0.9
DBP (mmHg)	90.6±2.3	95.6±2.8
SBP (mmHg)	156.2±2.2	163.9±3.6
Sample Size (N)	39	40
Correlation Coefficient (r)	[a]0.0265	[b]0.306
Calculated t value (tcal)	[a]0.163	[b]2.264

[a]*Insignificant linear positive correlation between PCV and BMI, P<0.05*
[b]*Significant linear positive correlation between PCV and BMI, P<0.05*

Hypertensive Subjects

In the hypertensive subjects as shown in Table 2, BMI was 28.7±0.8kg/m^2 and 28.9±0.7kg/m^2 in females and males respectively. PCV was 44.4±0.9% and 48.7±0.9% in females and males respectively. Also, the blood pressures increased as BMI and PCV increased.

There was a positive correlation between BMI and PCV in hypertensive subjects (male and female) but only significant in male subjects as observed in figure 3 and figure 4, thus PCV increased with increased BMI.

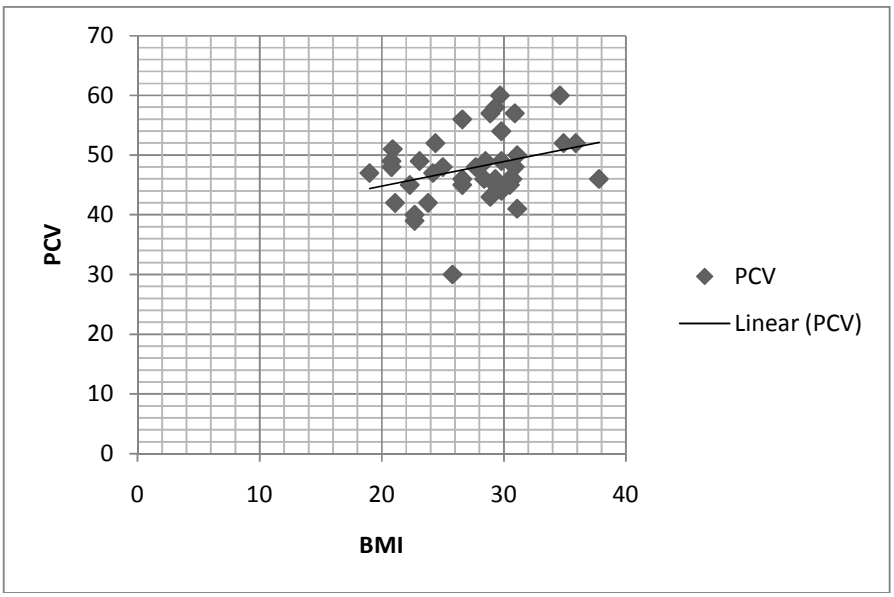

Figure 3: Scatter diagram showing the correlation between BMI and PCV in hypertensive males (n=40, r=0.306)

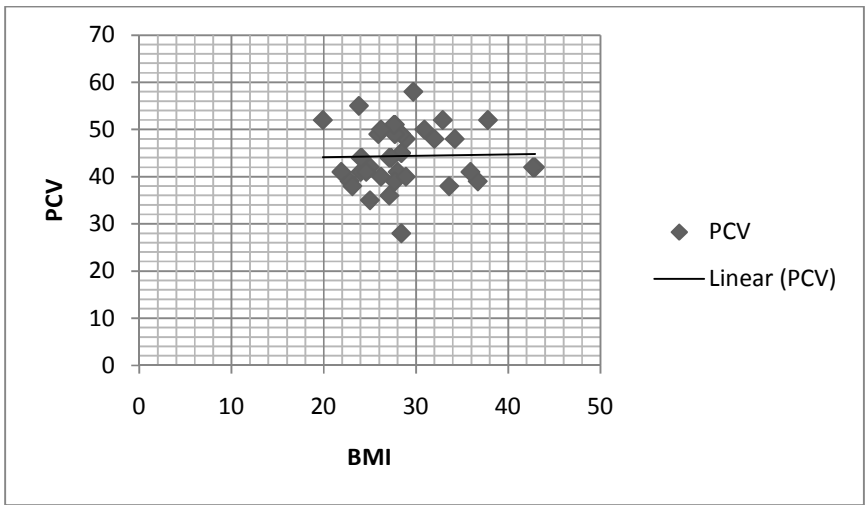

Figure 4: Scatter diagram showing correlation between BMI and PCV in hypertensive females (n=39, r=0.0265)

DISCUSSION

The increased PCV and BMI being the cause of some cardiovascular diseases such as hypertension as reported in previous or several studies [1, 2, 3, 4, 5] may be more confirmed through this present study via the knowledge of the correlation between them. Although, the relationship between blood pressure and BMI [5, 17] or blood pressure and PCV [2, 4] had been reported earlier either in the absence or presence of hypertension but no work has been reported on relationship between BMI and PCV. Also, a number of metabolic consequences of high BMI have been proposed as the blood pressure elevating mechanism [18].

In the normotensive subjects, a positive relationship was demonstrated between PCV and BMI but not significant. The insignificant correlation may be due to normal weight, high physical activities, less fat storage and normal PCV of the normotensive subjects.

Moreover, significant correlation was demonstrated between PCV and BMI, especially in male hypertensive subjects. In other words, more significant correlation between PCV and BMI existed in male than female hypertensive subjects probably as a result of established fact of higher PCV in male than female. In addition, it has been reported that haemoglobin, PCV and ferritin among obese women were higher than non-obese, and it was suggested that obese women appear to have greater iron stores than non-obese women, in terms of haemoglobin, PCV and ferritin concentrations [19, 20].

Interestingly, since there is significant correlation between PCV and fat storage in obese individuals according to the previous work [19], therefore it is not unreasonable to suggest that such significant correlation exist between PCV and BMI in hypertensive individuals. The significant correlation may be due to high PCV, high body weight, less physical activities, high fat storage of the hypertensive subjects.

However, the information from these previous studies [17, 18, 19, 20] confirmed the significant positive linear correlation between PCV and BMI, and there is a need for further studies to confirm these findings and to evaluate whether the correlation between PCV and BMI is due to cause or effect relationship.

Conclusively, primary prevention of hypertension by monitoring PCV and BMI in hypertension-prone subjects should be considered important.

REFERENCES

[1] W.C. Smith, G.D. Lowe, A.J. Lee, H. Tunstall-Pedoe, *J. Hypertens*., **1992**, 10, 467–472
[2] F.G. Fowkes, G.D. Lowe, A. Rumley, S.E. Lennie, F.B. Smith, P.T. Donnan, *Eur Heart J.*, **1993,** 14, 597– 601
[3] G.T.C. Ko, C.S. Cockram, C.C. Chow, W.B. Chan, W.Y. So, R. Ma, R. Osaki, J.C.N. Chan, *International Journal of Cardiology*., **2005**, 101, 429– 433
[4] A. Strand, H. Gudmundsdottir, A. Høieggen, E. Fossum, R. Bjørnerheim, I. Os, S.E. Kjeldsen, *Journal of the American Society of Hypertension*., **2007**, 1, 6, 400–406

[5] R.P. Gelber, J.M. Gaziano, J.E. Manson, J.E. Buring, H.D. Sesso, *American Journal of Hypertension*., **2007**, 20, 370–377
[6] F.S. Facchini, M. Carantoni, J. Jeppesen, G.M. Reaven, *Metabolism*, **1998**, 47, 7, 831-835
[7] G. Vazquez, S. Duval, D.R. Jacobs, K. Silventoinen, *Epidemiol. Rev*., **2007**, 29, 115-128
[8] R. Nyamdorj, Q. Qiao, T.H. Lam, J. Tuomilehto, S.Y. Ho, J. Pitkaniemi, T. Nakagami, V. Mohan, E.D. Janus, S.R. Ferreira, *Obesity (Silver Spring*)., **2008**, 16, 1622-1635
[9] L.J. Tamariz, J.H.Young, J.S. Pankow, H.C. Yeh, M.I. Schmidt, B. Astor, F.L. Brancati, *Am J Epidemiol.*, **2008**, 168, 1153–1160
[10] J. Ärnlöv, J. Sundström, E. Ingelsson, L. Lind, Diabetes Care published ahead of print September 17, **2010**, doi:10.2337/dc10-0955
[11] G.S. Wannamethee, I.J. Perry, A.G. Shaper, *Diabetes*., **1996**, 45, 576-579
[12] R.B. Devereux, J.I. Drayer, S. Chien, T.G. Pickering, R.L. Letcher, J.L. DeYoung, et al. *Am. J. Cardiol.*, **1984**, 54, 592–595
[13] R.J. Garrison, W.B. Kannel, J. Stokes 3rd, W.P. Castelli, *Prev. Med*. **1987**, 16, 235–251
[14] P.A. Cassano, M.R. Segal, P.S. Vokonas, S.T. Weiss, *Ann. Epidemiol*., **1990**, 1, 33– 48
[15] A.E. Field, N.R. Cook, M.W. Gillman, *Obes. Res*., **2005**, 13, 163–169
[16] O.I. Samuel, N. Thomas, O.U. Ernest, N. Imelda, N.S. Elvis, E. Ifeyinwa, *BMC Clin. Pathol*., **2010**, 10, 3.
[17] F. Tesfaye, N.G. Nawi, H. VanMinh, P. Byass, Y. Berhane, R. Bonita et al, *J. Human Hypertens.*, **2007**, 21, 1, 28-37
[18] J.S. Kaufman, M.C. Asuzu, J. Mufunda, T. Forrester, R. Wilks, A. Luke et al, *Hypertens.*, **1997**, 30, 1511-1516
[19] J. Fricker, G.L. Moel, M. Apfelbau, *Am. J. Clin. Nutr*., **1990**, 52, 863-866
[20] Z. Paknahad, S. Mahboob, N. Omidvar, M. Ebrahimi, A. Ostadrahimi, S.H. Afiatmilani, *Pakistan J. Nutr*., **2008**, 7, 2, 377-380