Original Article

Correlation of abdominal adiposity with components of metabolic syndrome, anthropometric parameters and Insulin resistance, in obese and non obese, diabetics and non diabetics: A cross sectional observational study. (Mysore Visceral Adiposity in Diabetes Study)

M.Premanath, H.Basavanagowdappa[1], M.Mahesh[1], M.Suresh[1]

Senior Consultant in Internal Medicine and Diabetes, Mysore, [1]Department of Medicine, JSS Medical College, JSS University, Mysore, Karnataka, India

ABSTRACT

Objectives: To measure Visceral Fat (VF) and Subcutaneous Fat (SCF) by ultrasound, in obese and non-obese diabetics and obese and non-obese non diabetics, in a South Indian (Asian Indian) Population and correlate them with Body Mass Index (BMI), Waist Circumference (WC), components of metabolic syndrome and Insulin Resistance (IR) **Research Design and Methods:** This was a prospective observational study, 80 diabetics (40 obese and 40 non obese) and 80 non diabetics (40 obese and 40 non obese) a total of 160 subjects were enrolled, out of whom 153 completed the study. The subjects were evaluated with respect to BMI, WC, Blood Pressure (BP); Fasting Blood Sugar (FBS) Fasting Insulin levels (FIL), HbA1C and Lipid profile.The SCF and VF were measured by Ultrasonography.The results were statistically analyzed. **Results:** WC correlated significantly with VF in all the groups. Diabetics had more VF compared to non-diabetics. Insulin resistance was significant in all the groups; however diabetics had greater levels of IR, BMI, WC, VF and SCF had no correlation with IR and had no significant correlation with metabolic parameters. **Conclusions:** In this study population, WC was found to be a useful surrogate measure of VF conforming to its well established applicability in other populations. Contrary to other studies elsewhere, SCF and VF were found to be poor indicators of Insulin Resistance. BMI, WC, VF and SCF were not useful in the prediction of metabolic syndrome. Ultrasound was found to be an easier and economic method of measuring abdominal adiposity and actual measurement of abdominal fat was more informative than anthropometric measurements.

Key words: Insulin resistance, ultrasound, visceral adiposity, waist circumference

Access this article online	
Quick Response Code:	Website: www.ijem.in
	DOI: 10.4103/2230-8210.139231

Introduction

Abdominal obesity and the consequent Insulin resistance are said be important contributing factors for diabetes, dyslipidemia and cardiovascular disease (CVD).[1-3] Several studies have shown higher risk of diabetes in association with abdominal obesity.[4] There is sparse data regarding these issues from India although 30 to 65% of adults in

Corresponding Author: Dr. M. Premanath, Prem Health Care, 671, Nrupatunga Road, M-Block, Kuvempunagar, Mysore - 570 004, Karnataka, India. E-mail: premanath.manjunath@gmail.com

India can be categorized as overweight or obese, which is an alarming figure for a developing country.[5]

Waist Circumference (WC) is a simple measurement for Visceral fat (VF), but may not represent only VF, as subcutaneous fat (SCF) also contributes to it.[4] WC has been shown to correlate with visceral fat and with hyperglycemia, hypertension and dyslipidemia.[6]Asian Indians, have a higher risk for obesity related complications at a lower level of BMI *vis-à-vis* their Caucasian counterparts owing to higher visceral fat.[7] The cut off points for Asian Indians are different when compared to western population as modified and recommended by World health organization (WHO). The Body Mass Index (BMI) of 23 to 24.9kg/mt^2 for overweight and >25kg/mt^2 for obesity.[5]

Measurement of visceral fat may have more significance than measuring WC. Computerized Tomography (CT) is the gold standard for the measurement of visceral fat volume, but is expensive, involves radiation and may not be universally available. Magnetic Resonance Imaging (MRI) is also a good method, but is much more expensive, over estimates fat deposits and may not be again universally available, both these methods cannot be used routinely.[8-10] Ultrasonography is relatively inexpensive, readily available equally reliable and involves no radiation and is a method with established validity.[11-19]

Studies have shown that the Visceral fat volume measured by CT, is very well correlated with the visceral fat measured by ultrasound (r -0.710, $P < 0.001$),[2] (r -0.860, $P < 0.001$), with a sensitivity of 69.2%, specificity of 82.8% and a diagnostic concordance of 74%.[18]

This study was carried out at Mysore, a city in South India. We have measured the SCF and VF in diabetics (obese and non-obese) as well as non-diabetics (obese and non-obese) using Ultrasonography. We correlated WC and BMI with SCF and VF and each with BP, Triglycerides (TG) high density lipoprotein (HDL), total cholesterol (TC), LDL (components of metabolic syndrome) and Insulin resistance (IR).

Objectives

To correlate sonographically measured SCF and VF with BMI and WC, Blood pressure, Total Cholesterol, Triglycerides, HDL, LDL,(components of metabolic syndrome) and Insulin resistance, in diabetics (obese and non-obese) and non-diabetics (obese and non-obese).

Materials and Methods

This was a prospective, cross sectional, comparative, observational study carried out from March 2010 to Feb 2011. Ethical clearance was obtained from the Institutional ethical committee.One hundred and sixtysubjects were recruited in four groups of 40 each,

- Group A – Obese Diabetics
- Group B – Non obese Diabetics
- Group C – Obese non diabetics
- Group D – Non obese non diabetics.

Subjects of both sexes aged 18 years and above were recruited.

Exclusion criteria

1.Type$_1$diabetes. 2. Type $_2$ Diabetics on glitazones and/or Insulin. 3. Presence of any acute illness. 4. Pregnancy. 5. Subjects on anti-obesity medication. 6. Co morbid conditions like Chronic obstructive pulmonary disease (COPD), HIV and TB. 7. Subjects coming under the category of Overweight (a BMI of 23-24.9kg/mt^2).

Written Informed consent was obtained from all the participants. Height (cm) and weight (kgs), were recorded. Waist circumference was measured (cm) midway between the lower border of the ribs and the iliac crest with subject in standing position, Blood pressure was recorded in the sitting position, in the right arm, with a standard mercury sphygmomanometer after five minute rest. Average of three readings was taken. Asian Indian BMI criteria for categorizing as obese and non-obese were followed. Fasting Blood Sugar (GOD-PAP method), HbA1C (HPLC method), serum cholesterol (CHOD-PAP method), serum triglycerides (Enzymatic method), HDL (3rd generation direct assay) LDL (3rd generation direct assay), Serum Insulin fasting assay (CLIA method) were done for each subject at a NABL accredited standard laboratory. Insulin Resistance was calculated by the HOMA-IR assessment formula of FBS (m moles) multiplied by Fasting Insulin (m IU) divided by 22.5.

Sonographic measurements

The measurement of subcutaneous fat (SCF), pre-peritoneal fat (PPF) and Visceral fat (VF) were done by the same Ultrasonologist for all subjects using a GE P5 Logic system with multiple frequency (2-5 Mg HZ) convex probe for measuring VF and linear probe (8-12 Mg HZ) for measuring abdominal wall fat. Criteria, as defined by Stolk *et al.,*[13] was used for the measurement, the details are as given below.

Visceral fat thickness (defined as the distance between the anterior border of lumbar vertebra and posterior surface of Rectus abdominus muscle) was measured midway between xiphisternum and umbilicus, approximately 5 cm

from umbilicus at three positions along the horizontal line [Figure 1]. All measurements were done at the end of quiet expiration, applying minimal pressure, not displacing or deforming the abdominal contents.[13]

Longitudinal scans were obtained using a linear probe along the mid line (linea alba) and fat skin barrier. The thickness of the subcutaneous fat was defined as the distance between the anterior surface of the linea alba and the fat skin barrier. Pre peritoneal fat was measured as extending from the anterior surface of the left lobe of the liver to the posterior surface of the linea Alba [Figure 2].

Statistical analysis

The group comparisons for various parameters like VF, SCF, etc., were done through one way ANOVA, where as Pearson's product moment correlations was employed to find out the relationship between physical and ultrasound parameters across all the groups for blood pressure, TC, TGL, HDL etc., Confidence limits at 95% interval were calculated for mean intra-abdominal fat values for all the 4 groups. The significance levels fixed for 0.05 levels for all the statistical tests applied. The statistical calculations were done using PASW (version 18.0, previously named SPSS).

Results

Out of a total of 160 recruited, 153 completed the study. Number of males were 93 (60.78%) and 60 (39.22%) were females. The mean age of males was 43.40 ± 12.23 years and females were 44.29 ± 11.61 years [Table 1]. There were 42 subjects in Group-A, 36 in Group B, 38 in Group C and 37 in Group D, [Table 1].

Table 2, depicts anthropological, clinical and Biochemical measurements in all the four groups. As it can be seen, the highest VF was seen in Group-A followed by Group-C, B and D. Comparison of VF between the groups were statistically significant(*P*-0.000). The highest IR was also in Group-A followed by Groups B, C and D in that order. Diabetic group as expected had higher IR compared to non diabetics. Even here also, comparisons between the four groups were highly significant (*P*-0.006) SCF was highest in Group C followed by Group A, B and D. showing that obese non diabetics had both higher SCF and VF. Both BMI and WC were highest in Group A followed by Group C, B and D. In so far as metabolic parameters were concerned, not much significance could be given to the values in diabetics either obese or non obese considering that all of them were on anti hypertensive and lipid lowering medications. The values in non diabetics for Systolic blood pressure (SBP), Diastolic blood pressure (DBP), TC, HDL and TG revealed no significant difference in comparison with diabetics. LDL was the lone parameter not significantly higher in these groups.

BMI and WC are the two anthropometric measurements routinely utilized to grade obesity. We aimed to know whether these anthropometric measurements would correlate with SCF or VF. BMI correlated significantly with SCF and VF in groups A, C and D. WC correlated with SCF in groups A and D, whereas it correlated significantly with VF in all the four groups (*P*-0.003, *P*- 0.000) [Table 3].

Table 1: Age-sex cross tabulation

Age	Sex				Total	%
	Male	%	Female	%		
<30	20	21.5	09	15.0	29	18.9
31-40	18	19.4	14	23.4	32	20.9
41-50	23	24.7	20	33.3	43	28.2
51-60	23	24.7	13	21.6	36	23.5
>60	09	9.7	04	6.7	13	8.5
Total	93		60		153	100
Mean	43.4±12.23		44.29±11.61			

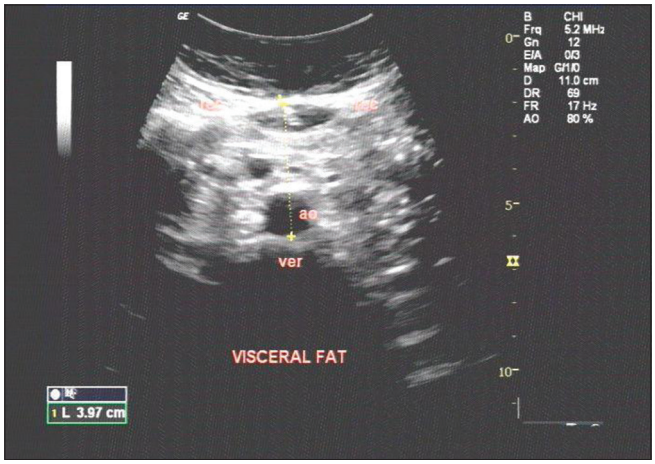

Figure 1: Measurement of Visceral Fat (VF) by Ultrasound

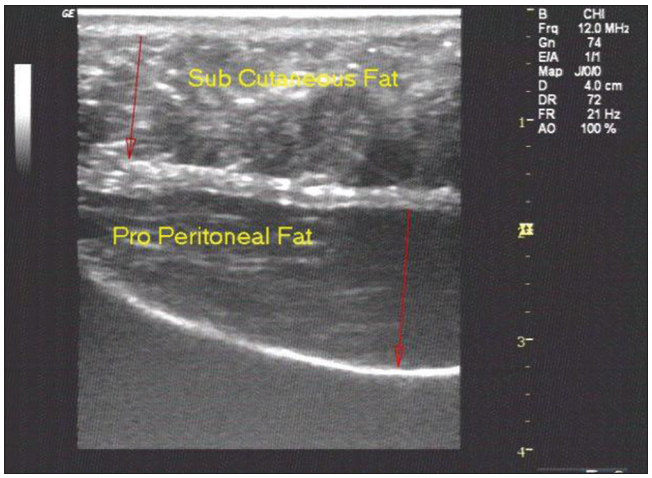

Figure 2: Measurement of Subcutaneous Fat (SCF) by Ultrasound

Table 2: Clinical, biochemical and fat measurements in the study groups

Parameters	Group - A	Group-B	Group-C	Group-D	*P* value
Number	42	36	38	37	
BMI (kg/m^2)	30.18±5.18	21.39±1.52	30.62±4.01	20.92±5.83	
WC (cm)	101.77±10.90	84.838.05	96.71±9.34	79.61±6.88	
SCF (cm)	2.79±0.72^b	2.21±0.68^a	3.15±0.56^b	2.18±0.82^a	0.000
VF (cm)	9.16±1.93^c	7.03±1.88^{ab}	8.08±2.08^{bc}	5.86±1.65^a	0.000
SBP (mmHg)	128.21±23.22	128.07±16.28	125.11±11.30	120.44±11.54	
DBP (mm/Hg)	82.37±7.08	79.62±8.88	80.85±7.33	78.74±7.10	
TC (mg%)	185.09±33.36	175.56±19.91	185.85±28.07	177.69±21.20	
TG (mg%)	161.68±83.22	146.17±62.80	152.27±56.93	126.44±53.86	
HDL (mg%)	44.24±6.65	42.82±8.34	42.92±5.16	43.22±4.76	
LDL (mg%)	97.27±50.98	105.73±19.66	123.00±32.77	117.29±32.99	
IR	9.47±17.59	4.81±4.95^{ab}	3.88±5.49^a	1.60±2.25^a	0.006

BMI: Body mass index, WC: Waist circumference, SCF: Subcutaneous fat, VF: Visceral fat, TC: Total cholesterol, TG: Triglycerides, HDL: High density lipoprotein, LDL: Low density lipoprotein, IR: Insulin resistance, SBP: Systolic blood pressure, DBP: Diastolic blood pressure

The VF/SCF ratio considered to be a significant parameter for visceral adiposity was compared between diabetics (both obese and non obese) with non diabetics (both obese and non obese) Diabetics had a significantly higher ratio signifying higher VF in them (*P*-0.000) as shown in Table 4.

Blood pressure, HDL, TG and WC are part of metabolic syndrome. We tried to correlate BMI, WC, SCF and VF with the metabolic parameters to know whether any consistent relationship existed. BMI correlated only with DBP in group D, WC correlated with DBP in groups B and D and with TG in groups B and C. SCF correlated with SBP in group A, with TC in group D and with LDL in group D, whereas VF correlated only with TG in group D. None of these correlated with IR in any of the groups. There was no consistency in any of these correlations [Table 5].

Insulin Resistance (IR) was the most important parameter of the investigations done and we wanted to know whether increase in VF would increase IR or not. IR was significant when compared between the groups, (*P*-0.006) as shown in Table 2, but surprisingly did not correlate with either VF or SCF or with BMI or WC [Table 5].

Table 3: Pearson's correlation of BMI and WC with SCF and VF

Group	BMI				WC			
	SCF		VF		SCF		VF	
	r	*P*	*r*	*P*	*r*	*P*	*r*	*P*
A	0.550	0.000	0.390	0.011	0.616	0.000	0.454	0.003
B	0.100	0.562	0.195	0.255	0.281	0.097	0.567	0.000
C	0.543	0.001	0.419	0.010	0.120	0.519	0.681	0.000
D	0.380	0.020	0.552	0.000	0.495	0.004	0.603	0.000

BMI: Body mass index, WC: Waist circumference, SCF: Subcutaneous fat, VF: Visceral fat

Table 4: Comparison of VF/SCF ratio amongst diabetics and non diabetics

	Diab	*N*	Mean	Std. deviation	Std. error mean
VF/SCF Ratio	Diab	78	3.4581	1.18091	0.13371
	Non-diab	74	2.7754	0.91713	0.10661

		t-test for equality of means			
		t	df	Sig. (2-tailed)	Mean difference
VF/SCF ratio	Equal variances assumed	3.966	150	0.000	0.6827

SCF: Subcutaneous fat, VF: Visceral fat

Discussion

This study is to our knowledge, one of the very few, that has undertaken a comprehensive comparison across four groups: Obese and non obese non diabetics and obese and non obesediabetics, in an Asian Indian population, the age of the participants in both the gender were well matched [Table 1]. Waist circumference (WC) is considered as the best predictor of VF than BMI in normal subjects,[1] whereas BMI correlated better with SCF than VF.[20] In Diabetic subjects, WC predicted VF better than BMI and SCF.[7] Asian Indians have a higher truncal fat with a lower BMI compared to other ethnic groups.[1,21] Ultrasound measurement of VF correlated better with components of metabolic syndrome (Met-S) than measured WC.[22]

Increased VF would play a major role in the development of T2 DM, CVD and Met-S[23] There is no consensus regarding the cut off points of VF above which the risk of these would increase. Studies have postulated VF 6.9cm in women,[24] 7 to 9 cm in men and 7 to 8 cm in non diabetics and 4.67 cm in men an 3.55 cm in women diabetics,[25,26] >5.8cm in men and >4.7cm in women diabetics,[20] A VF/SCF ratio of 2.7 ± 1.i[24] and >2.5 would likely to increase the risk.[27]

This study showed a uniformly high VF (9.16 ± 1.93, 7.03 ± 1.88, 8.08 ± 2.08 and 5.86 ± 1.65cm) in all the

Table 5: Pearson's correlation of BMI, WC, SCF and VF with components metabolic syndrome

Parameters	SBP	DBP	HDL	TC	TG	LDL	IR
BMI	NS	0.047 (D)	NS	NS	NS	NS	NS
WC	NS	0.002 (B) 0.032 (D)	NS	NS	0.047 (B) 0.042 (C)	NS	NS
SCF	0.008 (A)	NS	NS	0.046 (D)	NS	0.048 (D)	NS
VF	NS	NS	NS	NS	0.004 (D)	NS	NS

NS: Non significant, BMI: Body mass index, WC: Waist circumference, SCF: Subcutaneous fat, VF: Visceral fat, TC: Total cholesterol, TG: Triglycerides, HDL: High density lipoprotein, LDL: Low density lipoprotein, IR: Insulin resistance, SBP: Systolic blood pressure, DBP: Diastolic blood pressure

groups, despite lower BMI in non obese diabetics and non diabetics. SCF was high along with a high VF in obese non diabetics. WC consistently correlated with VF in all the groups where as BMI did not [Tables 2 and 3]. Hence WC can be considered as a surrogate marker of VF. The high VF would probably show increased risk among these subjects to CVD, T2DM and Met S.

VF has been a significant part of the definition of metabolic syndrome which also includes BP, Tg, HDL and blood sugar. Strong association of VF was seen with T2DM,[4,7,27] with Tg, TC and decreased HDL.[1,27] Mesenteric fat had a significant correlation with SBP, Tg and HDL.[14]

In this study, we have not defined Met-S in our subjects and those in group 4 did not have all the components. Since, we measured BP and components of Met-S in all the subjects, correlation was done between WC, BMI, SCF and VF and components of Met-S. None of them had any consistent correlation with the components of Met S. The inconsistent correlation in obese and non obese diabetics is probably due to the fact that they were on treatment at the time of recruitment. Obese and non obese non diabetics were not on any treatment and still there was no consistent correlation which probably would mean that VF alone may not be responsible for the changes in the components of Met Syndrome.

Obesity in general and visceral obesity in particular, is considered as the most important factor for the causation of Insulin Resistance (IR). Asian Indians have more IR independent of generalized or truncal obesity.[28] VF predicted IR[21,29] and it was the conduit by which obesity lead to IR.[30,31] VF has been implicated in hepatic IR by producing more free fatty acids and lipolysis. Secretion of several inflammatory adipocytokines by VF has also been said to lead to IR.[27] On the other side, IR was also associated with SCF mass.[32-34] Hence clear proof of the association of VF with IR is lacking.[32] It could be that VF and SCF and their joint interaction may lead to IR.[31] Asian Indians probably have a metabolic defect which causes IR, independent of generalized or truncal obesity[28] and there could be contribution of genes and environmental factors.[35,36]

It is to be noted here that presently, there is no consensus regarding the cut off values for IR. The values suggested are 1.35 to 1.96 for normal individuals and 2.42 for diabetics[37] and 1.78 for normal and 3.88 diabetic individuals[38] by homeostasis model assessment of insulin resistance (HOMA IR) method. The present study showed significantly higher values than the values mentioned above in diabetics and obese non diabetics. Between group comparisons of IR was significant (0.006) [Table 2]. Contrary to the expectations, WC, BMI, SCF and VF did not correlate with IR at all [Table 5] there by opening new avenues for research regarding the causation of IR.

This study has brought out the following (a) Presence of increased visceral adiposity even in those with lower BMI. (b) A higher VF in diabetics (c) Usefulness of WC as a possible marker for VF (d) lack of usefulness of abdominal adiposity as a predictor of Met S, (e) The lack of interrelationship of VF and SCF with IR. Further research is needed to confirm or refute the interesting and unexpected results of the present study.

We agree that the study has a few limitations. (i) the study was slightly underpowered.The numbers studied could have been larger for more meaningful results. (ii) It would have been ideal if we had diabetics not on ANY form of treatment. Though we excluded those diabetics prior and on Insulin and glitazone therapy, it was difficult to get diabetics not on any treatment. All the recruits in the Diabetes group were already on other medications.

Acknowledgements

We thank Dr.M.Raghunath DNB (Radiology) Consultant Sonologist for the measurement of abdominal fat by Ultrasonography, Dr.Lancy D'Souza PhD, Professor of Psychology, Maharaja's College, University of Mysore, for the statistical analysis. Dr.OsamaHamdy.MD, PhD, FACC, Medical Director, Obesity Clinical Program, Director of Inpatient Diabetes Management, Joslin Diabetic Center, Harvard Medical School, USAfor his invaluable suggestions and guidance from the inception of this study. The major research grant for this study was provided by Mysore Physicians Medical and Research

Trust (MPMRT) Mysore and minor grant was provided by Medical Education and Research Trust (MERT) Bangalore. We are grateful to both the organizations. We thank all our subjects for their participation and cooperation.

Conclusions

In this study of South Indian Population, WC was found to be a useful surrogate measure of VF. SCF and VF were found to be poor indicators of Insulin Resistance. BMI, WC, VF and SCF did not correlate consistently with the components of metabolic syndrome, suggesting a need for reassessment of their role. Further research, looking at other pathogenic mechanisms, for IR in Asian Indians is needed. Actual measurement of abdominal fat was found to be superior in comparison with anthropometric measurements for evaluation of IR.

References

1. Hamdy O, Porramatikul S, Al-Ozairi E. Metabolic Obesity: Between visceral and sub cutaneous fat.Curr Diab 2006;2:1-7.
2. Kim SK, Kim HJ, Hur KY, Choi SH, Ahn CW, Lim SK, *et al*. Visceral fat thickness measured by ultra sonography, can estimate not only visceral obesity but also risks of cardio vascular and metabolic diseases. Am J Clin Nutr 2004;79:593-9.
3. Abate N, Garg A, Peshock RM, Stray-Gunderson J, Grundy SM. Relationships of generalized and regional adiposity to insulin sensitivity in men. J Clin Invest 1995;96:88-98.
4. Boyko EJ, Fujimoto WY, Leonetti DL, Newell-Morris L. Visceral adiposity and risk of type 2 diabetes – A prospective study among Japanese Americans. Diabetes Care 2000;23:465-71.
5. Mishra A, Chowbey P, Makkar BM, Vikram NK, Wasir JS, Chadha D, *et al*. Consensus statement for diagnosis of obesity, abdominal obesity and metabolic syndrome for Asian Indians and Recommendations for physical activity, medical and surgical management, Review Article. J Assoc Physicians India 2009;57:163-70.
6. Lele RD, Marta AR. Insulin Resistance. In: Tripathy, Chandalia, Das, Rao, Madhu, Mohan, editors. RSSDI Text book of Diabetes. 2nd ed., vol. 1.RSSDI; 2008. p. 291-313.
7. Anjana M, Sandeep S, Deepa R, Vimaleswaran KS, Farooq S, Mohan V. Visceral and central abdominal fat and anthropometry in relation to diabetes in Asian Indians. Diabetes Care 2004;27:2948-53.
8. Basu A, Jenson MD. Fat metabolism in Diabetes In: Kahn, Weir, King, Jacobson, Moses, Smith, editors. Joslin's Diabetes Mellitus. 14th ed., chapter 16. Lippincot Williams and Wilkins; 2007. p. 265-73.
9. Kim SK, Park SW, Kim SH, Cha BS, Lee HC, Cho YW. Visceral fat amount is associated with Carotid atherosclerosis, even in type 2 diabetic men with normal waist circumference. Int J Obes 2009;33:131-5.
10. Valchos IS, Hatziioannou A, Perelas A, Perrea DN. Sonographic Assessment of Regional Adiposity. Am J Radiol 2007;189:1545-53.
11. Hirooka M, Kumagi T, Kurose K, Nakanishi S, Michitaka K, Matsuura B, *et al*. A Technique for the measurement of visceral fat by ultrasonography: Comparison of measurement by Ultrasonography and Computed Tomography. Intern Med 2005;44:794-9.
12. Gong W, Ren H, Tong H, Shen X, Luo J, Chen S, *et al*. A comparison of ultrasound and MRI, to assess visceral fat in metabolic syndrome. Asia Pac J Clin Nutr 2007;16 Suppl 1:S339-45.
13. Stolk RP, Wink O, Zelisson PM, Meijer R, Van Gils AP, Grobbee DE. Validity and reproducibility of ultrasonography for the measurement of intra-abdominal adipose tissue. Int J Obes 2001;25:1346-51.
14. Liu KH, Chan YL, Chan WB, Kong WL, Kong MO, Chan JC. Sonographic measurement of mesenteric fat thickness is a good correlate with cardiovascular risk factors: Comparison with subcutaneous and preperitonial fat thickness, magnetic resonance imaging and anthropometric indexes. Int J Obes Relat Metab Disord 2003;27:1267-73.
15. Tornaghi G, Raiteri R, Pozzato C, Rispoli A, Bramani M, Ciplot M, *et al*. Anthropometric or ultrasonic measurements in assessment of visceral fat? A comparative study. Int J Obes Relat Metab Disord 1994;18:771-5.
16. Armellini F, Zamboni M, Rigo L, Todesco T, Bergamo-Andreis IA, Procacci C, *et al*. The contribution of sonography, to the measurement of intra abdominal fat. J Clin Ultrasound 1990;18:563-7.
17. Armellini F, Zamboni M, Robbi R, Todesco T, Rigo L, Bergamo-Andreis IA, *et al*. Total and intra abdominal fat measurements by ultrasound and computerized tomography. Int J Obes Relat Metab Disord 1993;17:209-14.
18. Hirooka M, Kumagi T, Kurose K, Nakanishi S, Michitaka K, Matsuura B, *et al*. A Technique for the measurement of visceral fat by ultrasonography: Comparison of measurements by ultrasonography and computed tomography. Intern Med 2005;44:794-9.
19. Wajchenberg BL. Subcutaneous and visceral adipose tissue: Their relation to metabolic syndrome. Endocr Rev 2000;21:697-738.
20. Roopakala MS, Anagha S, Ashtalakshmi, Srinath, Ashok, Giridhar, *et al*. Anthropometric Measurements, as predictors of Intra abdominal Fat thickness. Indian J Physiol Pharmacol 2009;53:259-64.
21. Banerji MA, Faridi N, Atluri R, Chaiken RL, Lebovitz HE. Body composition, visceral fat, leptin and insulin resistance in Asian Indian men. J Clin Endocrinol Metab 1999;84:137-44.
22. Stolk RP, Meijer R, Mali WP, Grobbee DE, van der Graaf Y; Secondary Manifestations of Arterial Disease Study Group. Ultrasound measurements of intra abdominal fat, estimate the metabolic syndrome better than do measurements of waist circumference. Am J Clin Nutr 2003;77:857-60.
23. Goodpaster BH, Shanthi K, Resnick H, Harris TB, Schwartz AV, Kritchevsky S, *et al*. Association between regional adipose tissue distribution and both type 2 diabetes and impaired glucose tolerance in elderly men and women. Diabetes Care 2003;26:372-9.
24. Rebeiro-Filho FF, Faria AN, Azjen S, Maria-Teresa Z, Sandra RG. Methods of estimation of visceral fat: Advantages of ultrasonography. Obes Res 2003;11:1488-94.
25. Vlachos IS, Hatziioannou A, Perelas A, Perrea DN. Sonographic assessment of regional adiposity. AJR Am J Roentgenol 2007;189:1545-53.
26. Riberio-Filho FF, Faria AN, Kohlmann O Jr, Ajzen S, Ribeiro AB, Zanella MT, *et al*. Ultrasonography for the evaluation of visceral fat and cardiovascular risk. Hypertension 2001;38:713-7.
27. Kabir M, Catalano J, Suchitra A, Kim SP, Van Citters GW, Dea MK, *et al*. Molecular evidence supporting the portal theory: A causative link between visceral adiposity and hepatic insulin resistance. Am J Physiol Endocrinol Metab 2005;288:E454-61.
28. Chandalia M, Abate N, Garg A, Stray-Gundersen J, Grundy SM. Relationship between generalized and upper body obesity to insulin resistance in Asian Indian men. J Clin Endocrinol Metab 1999;84:2329-35.
29. Hayashi T, Boyko EJ, McNeely MJ, Leonetti DL, Kahn SE, Fujimoto WY. Visceral Adiposity, Not Abdominal Subcutaneous Fat Area, Is Associated With an Increase in Future Insulin Resistance in Japanese Americans. Diabetes. Published Ahead of Print at http://diabetes.diabetesjournals.org on 25 February 2008.

30. Lebovitz HE, Banerji MA. Point: Visceral adiposity is causally related to insulin resistance. Diabetes Care 2005;28:2322-5.
31. Hanley AJ, Wagenknecht LE, Norris JM, Bryer-Ash M, Chen YIanderson AM, *et al*. Insulin resistance, beta cell dysfunction and visceral adiposity as predictors of incident diabetes: The Insulin Resistance Atherosclerosis Study (IRAS) Family Study. Diabetolgia 2009;52:2079-86.
32. Frayn KN. Visceral fat and insulin resistance – causative or correlative? Br J Nutr 2000;83 Suppl 1:S71-7.
33. Miles JM, Jensen MD. Counterpoint: Visceral adiposity is not causally related to insulin resistance. Diabetes Care 2005;28:2326-7.
34. Garg A. Regional adiposity and insulin resistance. J Clin Endocrinol Metab 2004;89:4206-10.
35. Wagenknecht LE, Langefeld CD, Scherzinger AN, Scherzinger AL, Norris JM, Haffner SM, *et al*. Insulin sensitivity, insulin secretion and abdominal fat. The Insulin Resistance Atherosclerosis Study (IRAS) Family Study. Diabetes 2003;52:2490-6.
36. Henkin L, Bergman RN, Bowden DW, Ellsworth DL, Haffner SM, Langefeld CD, *et al*. Genetic epidemiology of insulin resistance and visceral adiposity: The IRAS family study design and methods. Ann Epidemiol 2003;13:211-7.
37. Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: Insulin resistance and B cell function from fasting plasma glucose and insulin concentrations in man. Diabetolgia 1985;28:412-9.
38. Esteghamati A, Ashraf H, Khalilzadeh O, Zandieh A, Nakhjavani M, Rashidi A, *et al*. Optimal cut-off of homeostasis model assessment of insulin resistance (HOMA – IR) for the diagnosis of metabolic syndrome: Third national surveillance of risk factors of non – communicable diseases in Iran (SuRFNCD -2007). Nutr Metab 2010;7:26.

Cite this article as: Premanath M, Basavanagowdappa H, Mahesh M, Suresh M. Correlation of abdominal adiposity with components of metabolic syndrome, anthropometric parameters and Insulin resistance, in obese and non obese, diabetics and non diabetics: A cross sectional observational study. (Mysore Visceral Adiposity in Diabetes Study). Indian J Endocr Metab 2014;18:676-82.
Source of Support: Nil, **Conflict of Interest:** No.