

Original Research Article

A comparative study of sagittal abdominal diameter and other anthropometric indices of obesity in adults with family history of cardiovascular risk factors

Nishmita R.*, Ravi K.

Department of Medicine, BMCRI, Bangalore, Karnataka, India

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***Correspondence:**

Dr. Nishmita R.,

E-mail: nishmitar@gmail.com

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ABSTRACT

Background: Obesity is evolving into a modern day epidemic in India. Visceral Adipose Tissue is the main culprit, being the metabolically active component that is responsible for the cardiovascular risk factors. The day-to-day anthropometric indices fail to take body composition and distribution into account and hence miss a chance at early detection. Family history plays an important role by serving as a genetic link in the transmission of risk factors. There is a need for vigorous screening techniques which are clinically plausible for an early diagnosis of visceral adiposity in individuals predisposed to the numerous risks caused by its accumulation. Sagittal abdominal diameter is a useful tool in measuring the visceral adipose tissue clinically and in the current study; a comparison was done between SAD and already existing anthropometric indices.

Methods: The study was done on 223 subjects in Bangalore during the period of November 2015 to May 2017. Among the subjects studied, 54.7% were Male and 45.3% were female. When compared to BMI, WC and WHR, SAD correlated better with total cholesterol, fasting blood sugar, post prandial blood sugar and HbA1c.

Results: The mean SAD was more in people with a combined history of DM and HTN than in individuals with history of either DM or HTN alone.

Conclusions: We could conclude that Sagittal Abdominal Diameter is comparable to Body Mass Index, but it correlates better with metabolic risk profile of an individual than BMI, WC and WHR.

Keywords: Sagittal abdominal diameter, Visceral adiposity, Family history, Cardiovascular disease

INTRODUCTION

The Indian subcontinent is caught at the crossroads of an epidemiological transition, as non-communicable diseases like coronary heart disease and type 2 Diabetes mellitus are fast replacing infections as the leading cause of morbidity and mortality.¹ Obesity is associated with a major prevalence of cardiovascular risk factors by affecting atherosclerosis through its impact on known risk factors for CAD such as dyslipidaemia, hypertension,

glucose intolerance, inflammatory markers, and the pro-thrombotic state as well as yet unrecognized features of the metabolic syndrome.²⁻⁴ With change in lifestyle and westernization of Indian Diet, Obesity is snowballing into an escalating epidemic.⁵ There is a need to explore newer, inexpensive and easily obtainable anthropometric measures to clinically evaluate the relationship between visceral adiposity and cardiovascular risk factors.^{6,7} A novel anthropometric index, sagittal abdominal diameter (SAD) is a useful tool in measuring the visceral adipose

tissue clinically. It reflects visceral adipose tissue (VAT) based on the fact that subcutaneous fat is displaced inferiorly by gravity in supine position. This study was done to compare sagittal abdominal diameter with other anthropometric indices of obesity in apparently healthy individuals who were not previously diagnosed or treated for DM, hypertension, ischemic heart disease or any other cardiovascular risk factors and assess its utility in predicting these condition by correlating the metabolic parameters like fasting blood sugar, post prandial blood sugar, lipid profile HbA1c and ECG with the anthropometric measurements. The individuals were included in the study based on the presence of a family history of a first degree relative suffering from either of these. Family History serves as a bridge from genetics to genomics in clinical practice.⁹ Because Family History is an independent risk factor for CVD, it has the potential to become a screening tool to identify people, especially asymptomatic young adults, who are at increased CVD risk. The intention of this study is twofold. Firstly, to assess utility of SAD as a screening test for dysglycemia, dyslipidemia or pre-hypertension in an apparently healthy population with only a family history of cardiovascular risk factors. Secondly to look for any linear correlation between sagittal abdominal diameter and dyslipidemia, pre-hypertension and dysglycemia, such that SAD can be used as a stand-alone robust predictor of these derangements in future studies.

METHODS

Source of data, study design and study duration

The study was conducted on 223 subjects attending outpatient departments or admitted to Victoria hospital and Bowring and Lady Curzon hospitals affiliated to Bangalore medical college and research institute, Bangalore. Current study was a cross sectional study conducted from from November 2015 to May 2017

Inclusion criteria

Inclusion criteria for current study were; patients willing to give written informed consent, age group: >18yrs and Patients who have a family history of a first degree relative suffering from either hypertension, diabetes, dyslipidemia or ischemic heart disease.

Exclusion criteria

Exclusion criteria for current study were; patients who are a known case (patients who were diagnosed previously and were initiated on treatment) of diabetes, hypertension or ischemic heart disease, spinal deformity, pregnancy, abdominal tumours, lump, ascites, pathological diseases (cancer, insufficient renal and hepatic performance, and chronic inflammatory pathologies) and subjects in whom anthropometric measurements were not feasible. Patients who were incidentally found to be diabetic, hypertensive or

dyslipidaemia during the course of the study were included. Later on they were initiated on treatment.

Investigations done on patients

Fasting blood sugar, post prandial blood sugar, HbA1C, fasting lipid profile, ECG.

Procedure

Adults with a family history of cardiovascular risk factors satisfying the inclusion criteria and exclusion criteria, attending outpatient departments and/or getting admitted to hospitals affiliated to Bangalore medical college & research institute, during the period of November 2015 to May 2017 were taken up for the study after obtaining written informed consent. Anthropometric measurements were performed on subjects. Body weight was measured, and height was measured to the nearest 0.1 cm by using a wall-mounted stadiometer. BMI was calculated by dividing weight (kg) by the square of height (m²). With the subject in a standing position, the Waist circumference was taken at the midline between the lower border of the rib cage and iliac crest to the nearest 0.1 cm after a normal expiration. Hip circumference was measured at the maximum buttock girth. Waist to Hip ratio was calculated. Sagittal abdominal diameter or “supine abdominal height” was measured after a normal expiration to nearest 0.1 cm in supine position with straight legs on a firm examination table, without clothes in the measurement area, at the level of iliac crest (L4-5) level with the help of a sliding beam caliper. Out of the two limbs of the caliper, the lower limb was slid underneath the back of the subject and the upper limb was brought down to slightly touch the abdomen at end expiration. Sagittal Abdominal Diameter was measured at the vertical distance between the two horizontal limbs of the caliper. Fasting blood sugars, Lipid Profile, HbA1C and Post Prandial Blood sugars were investigated. ECG was done.

Statistical analysis

Descriptive and inferential statistical analysis has been carried out in the present study. Results on continuous measurements are presented on Mean±SD (Min-Max) and results on categorical measurements are presented in number (%). Significance is assessed at 5 % level of significance. In this study, descriptive statistics such as mean and standard deviation (SD) for continuous variables, frequencies and percentages were calculated for categorical variables were determined. Comparison between males and females were analysed using Chi-Square test and unpaired t test respectively for categorical and continuous variables. SAD was divided into 4 quartiles and comparison in between quartiles was done using Kruskal-Wallis Test. Pearsons correlation coefficient was calculated between various quantitative Variables in the study. Bar charts and Pie charts were

used for visual representation of the analysed data. Level of significance was set at 0.05.

RESULTS

Age group/gender distribution

Among the subjects studied, 122 (54.7%) were Male and 101 (45.3%) were female. Among the subjects included

in the study, maximum number of males i.e. 32.8% were in the age group of 21-30 and maximum number i.e. 28.7 % of females were in the age group of 31-40 years. Among the various metabolic parameters studied, there was no statistically significant difference among male and female in terms of age, FBS, PPBS, HbA1C or lipid profile. The mean SAD was more in people with a combined history of DM and HTN than in individuals with history of either DM or HTN alone. However, the values did not attain statistical significance.

Table 1: Comparison of various metabolic parameters among the study subjects (n=223).

Parameter	Males mean (SD)	Females mean (SD)	Total mean (SD)	P value
Age	35.82 (14.05)	39.59 (14.61)	37.53 (14.40)	0.051
FBS	110.16 (44.47)	111.56 (46.36)	110.79 (45.23)	0.818
PPBS	134.05 (34.07)	138.09 (58.10)	135.88 (55.84)	0.592
HbA1c	6.05 (1.59)	5.94 (1.14)	6.00 (1.40)	0.557
TC	155.93 (41.05)	156.71 (45.63)	156.28 (43.09)	0.894
LDL	86.82 (37.34)	86.77 (35.68)	86.80 (36.52)	0.992
HDL	44.33 (16.33)	44.17 (12.11)	44.26 (14.54)	0.936
VLDL	24.82 (13.99)	25.74 (14.78)	25.23 (14.33)	0.634
TG	124.41 (69.93)	128.66 (74.00)	126.34 (71.67)	0.660
SBP	120.55	121.41	121.0	0.542
DBP	75.30	76.11	75.7	0.612

Table 2: Correlation between family history and SAD (n=223).

Family history	Father N (%)	Mother N (%)	Siblings N (%)
DM	19.21 (3.65)	19.35 (3.79)	20.24 (4.00)
HTN	19.16 (3.93)	18.93 (3.95)	19.65 (4.23)
Both DM & HTN	20.05 (3.77)	20.32 (4.03)	18.00 (-)
P value	0.479	0.357	0.798

Table 3: Distribution of study subjects according to their BMI (n=223).

BMI (Asian criteria)	Father (N=122) frequency (%)	Mother (N=101) frequency (%)	Siblings frequency (%)
<18.5 (Underweight)	19 (15.6)	7 (6.9)	26 (11.7)
18.5-22.99 (Normal)	58 (47.5)	34 (33.7)	92 (41.3)
23-24.99 (Overweight)	17 (13.9)	23 (22.8)	40 (17.9)
25.0-29.99 (Pre-obese)	27 (22.1)	29 (28.7)	56 (25.1)
≥30 (Obese)	1 (0.8)	8 (7.9)	9 (4.0)
<18.5 (Underweight)	19 (15.6)	7 (6.9)	26 (11.7)
18.5-22.99 (Normal)	58 (47.5)	34 (33.7)	92 (41.3)

*p value=0.003

Table 4: Correlation between BMI and SAD (n=223).

BMI	Quartiles of SAD, frequency (%)				P value
	1 st (N=73)	2 nd (N=60)	3 rd (N=50)	4 th (N=40)	
<18.5	24 (32.9)	1 (1.7)	1 (2.0)	0 (0.0)	<0.001
18.5-22.99	41 (56.2)	30 (50.0)	14 (28.0)	7 (17.5)	
23.24.99	4 (5.5)	15 (25.0)	15 (30.0)	6 (15.0)	
25.0-29.99	3 (4.1)	14 (23.3)	18 (36.0)	21 (52.5)	
≥30	1 (1.4)	0 (0.0)	2 (4.0)	6 (15.0)	

In our study, BMI had strong positive correlation to Weight (r=0.853), SAD (r=0.668) and WC(r=0.586). It indicates that BMI correlates the same with Visceral Adipose Tissue measured by SAD and Subcutaneous

adipose tissue measured by WC and is not useful in differentiating the two of them. BMI correlates poorly with WHR in women but moderately with men because

of the difference in fat distribution, which WHR appreciates better in women than in men.

Table 5: Gender wise correlation between SAD and other variables in the study (n=223).

SAD	Males		Females	
	Correlation coefficient	P value	Correlation coefficient	P value
Age	0.435	<0.001	0.320	0.001
SBP	0.344	<0.001	0.342	0.001
DBP	0.214	0.018	0.200	0.016
Weight	0.684	<0.001	0.577	<0.001
Height	-0.151	0.097	0.165	0.100
WC	0.496	<0.001	0.450	<0.001
HC	0.419	<0.001	0.375	<0.001
WHR	0.307	0.001	0.108	0.283
BMI	0.779	<0.001	0.560	<0.001
HDL	-0.104	0.253	-0.050	0.617
LDL	0.075	0.410	0.144	0.152
VLDL	0.402	<0.001	0.368	<0.001
TC	0.161	0.077	0.218	0.028
TG	0.402	<0.001	0.369	<0.001
FBS	0.577	<0.001	0.279	0.005
PPBS	0.568	<0.001	0.345	<0.001
HbA1C	0.482	<0.001	0.395	<0.001

Table 6: Quartiles of SAD and its correlation to other variables in the study (n=223).

SAD	Quartiles of SAD				P value
	1 st (13.5-19)	2 nd (19.5-21)	3 rd (21.5-23)	4 th (>23.5)	
Age	31.03 (14.05)	36.45 (14.30)	40.12 (10.30)	47.78 (13.25)	<0.001
Weight	51.10 (9.19)	61.38 (5.82)	65.32 (8.19)	67.63 (8.81)	<0.001
Height	161.48 (8.52)	162.52 (6.77)	162.12 (6.77)	159.73 (7.44)	0.305
WC	75.14 (14.60)	84.47 (8.22)	89.72 (7.71)	91.48 (9.85)	<0.001
HC	86.79 (14.50)	94.10 (8.52)	98.60 (9.73)	101.95 (13.04)	<0.001
WHR	0.85 (0.09)	0.90 (0.07)	0.91 (0.10)	0.89 (0.11)	0.004
BMI	19.57 (3.07)	23.29 (2.41)	24.87 (2.89)	26.57 (3.56)	<0.001
HDL	45.71 (15.39)	43.42 (14.08)	45.64 (14.61)	41.15 (13.45)	0.362
LDL	83.21 (36.88)	86.85 (33.17)	89.92 (38.64)	89.38 (38.67)	0.739
VLDL	18.93 (10.16)	24.95 (14.02)	27.20 (15.38)	34.72 (14.54)	<0.001
TC	147.86 (40.77)	155.23 (35.93)	162.66 (47.69)	165.27 (49.19)	0.129
TG	94.67 (50.75)	124.88 (70.06)	136.38 (77.16)	173.75 (72.55)	<0.001
FBS	87.40 (23.01)	108.67 (43.24)	126.06 (53.76)	137.60 (46.31)	<0.001
PPBS	107.49 (35.44)	127.38 (45.28)	156.46 (67.35)	174.70 (54.07)	<0.001
HbA1C	5.42 (0.59)	5.65 (1.02)	6.41 (1.77)	7.06 (1.68)	<0.001

Even in spite of BMI being normal, 23% had SAD in the higher quartiles and even with overweight and obese individuals, 35% had SAD in the lower quartiles. Men showed high correlation to Weight, BMI, FBS and PPBS, moderate correlation to age, SBP, WC, HC, HbA1C, VLDL, TG. Women showed high correlation to Weight and BMI. Moderate correlation to WC, HC, VLDL, TG, PPBS and HbA1C and low correlation to FBS. All metabolic and anthropometric parameters showed positive correlation with SAD except Height and HDL (statistically significant at $p<0.001$) It was found in this

study that the Pearson correlation coefficients (r values) of various metabolic parameters like FBS, PPBS, HbA1c, VLDL and TGC in men were greater than that found in women. However women had increased r values in LDL and total cholesterol. With progressing age, SAD goes on increasing. 1st Quartile (31.03) to 4th Quartile (47.78). This was statistically significant ($p<0.001$). The mean SAD in people with dysglycemia in the form of HbA1c 5.7-6.4 was more 22.57cm vs. 18.36cm in individuals with no dysglycemia ($p<0.001$). The mean SAD in people with

impaired fasting glucose was more 20.56cm vs. 17.55cm in individuals with normal fasting glucose ($p < 0.001$).

Table 7: Correlation between SAD and metabolic derangement (n=223).

SAD	Cases mean (SD)	Reference mean (SD)	P value
HDL <30	19.00 (3.27)	19.58 (3.89)	0.450
LDL >150	20.66 (4.34)	19.35 (3.73)	0.094
TC >200	20.38 (4.31)	19.37 (3.73)	0.170
HbA1c >6.4	22.57 (3.38)	18.36 (3.43)	<0.001
HbA1c 5.7-6.4	22.12 (2.70)		
FBS 100-125	20.56 (2.96)	17.55 (3.46)	<0.001

The mean SAD in people with hypercholesterolemia (TC>200mg/dl) was more 20.38cm vs. 19.37cm in individuals with TC<200mg/dl ($p > 0.001$). When compared to BMI, SAD correlated better with TC ($r=0.189$ vs. $r=0.148$), FBS ($r=0.435$ vs. $r=0.296$), PPBS ($r=0.461$ vs. $r=0.287$) and HbA1c ($r=0.435$ vs. $r=0.207$). This was statistically significant ($p < 0.001$). To summarise, all metabolic and anthropometric parameters showed positive correlation with SAD except Height and HDL (statistically significant at $p < 0.001$). In our study SAD and BMI had more positive correlations with FBS, PPBS, HbA1c, TC and BP when compared to WC and WHR. . SAD had higher r values than BMI and hence correlated better.

DISCUSSION

Many studies have been conducted to assess the utility of sagittal abdominal diameter as an anthropometric tool. However these studies have been mostly on Caucasian and mongoloid population.^{7,8} The current study is a cross sectional study which assessed sagittal abdominal diameter in the South Indian metropolitan city of Bangalore which has a different demography than the earlier studies and is first of its kind. This study had a predominantly younger population between 20-40 years, comparable to Vasques et al.¹⁰

This study comprised of 54.7% males and 45.3% of females. This was similar to study conducted by Riserius et al and Sharda et al.^{11,12} This study was also unique in that the prime inclusion criteria was the presence of a family history of cardiovascular risk factors in apparently healthy individuals. All the study subjects had atleast one first degree relative suffering from cardiovascular risk factors like diabetes, hypertension or ischemic heart disease. 30% of the subjects had more than one relative suffering from either of the cardiovascular risk factors. In males, 16.5% had a paternal family history and 14.7% had a maternal family history of suffering from more than one CVD risk factor. In females, 12.8 % had paternal and 9% had maternal family history of more than one risk factor. 3% of them were diabetics, 32.5% were hypertensives, 28.5% were both hypertensive and diabetic. 2.2% had ischemic heart disease and 1.8% had stroke. The history of father suffering from diabetes was the most elicited i.e. 42.6%.

Age

Abdominal adipose tissue is known to increase with age in both sexes with women developing a more “android” or centralized body fat distribution as they age, particularly after menopause. In this study, it was found that, with increasing age SAD also increased. With increasing age, due to sedentary life style and accumulation of metabolic risk factors, there will be an increase in the visceral adipose tissue which is reflected in our study.

Sex

Riserius et al proved that the greater correlation of SAD in men may be explained by the well-established gender dimorphism in regional adipose tissue distribution. They said that at a given BMI, men present a higher visceral adipose tissue content compared with women.¹¹ These findings can be explained by the fact that catecholamine mediated free fatty acid release is lower in women than in men, whereas free fatty acid release from the upper body depots is comparable basal fat oxidation is lower in females as compared to males, thereby contributing to a higher fat storage in women. This was demonstrated by Vasques et al which was done in an exclusive female population.¹⁰

Family history

In our study, the mean SAD was more in people with a combined history of diabetes and hypertension than in individuals with history of either diabetes or hypertension alone ($p > 0.001$).

Body mass index (BMI)

In general, body mass index is used to verify if subjects are overweight or obese without considering muscle mass. This apparent paradox may reflect intrinsic limitations of Body Mass Index in differentiating lean and adipose tissues and in accounting for body fat distribution. In this study, even inspite of BMI being normal, 23% had SAD in the higher quartiles. These could be the metabolically obese normal weight individuals who are misclassified into the lower BMI class. 35% of individuals with BMI in the overweight and obese classification had SAD in the lower quartiles.

These could be the metabolically healthy obese individuals.

Waist circumference

Waist circumference (WC) serves as one of the criteria for the diagnosis of metabolic syndrome.¹⁴ However, it does not distinguish visceral from subcutaneous abdominal adipose tissue. In our study, WC correlated the least with SAD because SAD measures VAT and BMI measures SAT. Waist circumference had moderate correlation with blood sugars ($p < 0.001$). WC had weak positive correlation to all parameters except height and hip circumference which showed a negative correlation.

Waist to hip ratio (WHR)

A limitation of waist circumference is that it takes no account of body composition, whereas waist-to-hip ratio (WHR) is a measure of body shape and to some extent of lower trunk adiposity.^{16,17}

Cardiometabolic profile

In our study SAD had a considerable advantage over BMI in predicting dysglycemia as it showed moderate correlation to glycemic parameters as compared to poor correlation by BMI ($p < 0.001$). In a study done by Pajunen et al it was found that the combination of high SAD and high BMI showed a nearly 37-fold increased risk of diabetes incidence compared with the risk of individuals who had normal BMI ($< 25 \text{ kg/m}^2$) and belonged to the lowest SAD quartile (13.5-19 cm). Even in our study, the mean SAD in people with dysglycemia in the form of HbA1c 5.7-6.4 was more 22.57 vs. 18.36 in individuals with no dysglycemia ($p < 0.001$).¹³ When compared to other studies, In the current study, total cholesterol, LDL had a lower mean value. TC showed low positive correlation than other studies. Women correlated better than men (0.21 vs. 0.16). Hypertriglyceridemia may be the major cause of the other lipid abnormalities since it will lead to delayed clearance of the TG-rich lipoproteins and formation of small dense LDL. The mean SAD in people with hypercholesterolemia ($\text{TC} > 200$) was more 20.38 vs. 19.37 in individuals with $\text{TC} < 200$. A positive linear correlation with adiposity is well documented with substantial risks of hypertension being reported among the obese. The current study had 20.6% study subjects were pre hypertensive and 14.8% were found to have newly detected long standing hypertension with ECG changes of LVH. The mean SAD in people with hypertension ($\text{BP} = 140/90$) was more 20.11 vs. 18.21 in individuals with $\text{BP} < 140/90$. In this study, SAD was marginally better than BMI in terms of correlation with SBP (0.279 vs. 0.236)

CONCLUSION

Robust correlation exists between sagittal abdominal diameter and cardiovascular risk factors. Sagittal

abdominal diameter is comparable to body mass index. but it correlates better with metabolic risk profile of an individual than body mass index, waist circumference & waist-to-hip ratio. Sagittal Abdominal Diameter may predict dysglycemia and hypercholesterolemia marginally better than the currently in vogue anthropometric indices. Family history plays a role in ascertaining the genetic risk conferred by a particular body habitus as assessed by anthropometry. The increased risk due to family history was reflected in an increase in sagittal abdominal diameter in individuals with a strong history who also happened to have metabolic derangements. Large scale studies need to be done to validate the hypothesis. Sagittal abdominal diameter may have utility as a novel clinical measurement used in research and screening to identify “metabolically obese” normal weight men who would benefit from lifestyle and pharmacological interventions.

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Ethical approval: The study was approved by the Institutional Ethics Committee

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