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RESEARCH ARTICLE

Assessment of regional body fat and body mass index with glycemic status in the middle-aged offspring of type 2 diabetic parents - A cross-sectional study

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ABSTRACT

Background: Genetic predisposition is a key factor in developing insulin resistance in prone individuals. Sedentary lifestyle, lack of regular physical exercise, excessive intake of junk foods, and stress are proved to be important factor in the development of obesity. Assessing occurrence of insulin resistance earlier, an important component of metabolic syndrome can prevent the morbidity in the subjects. **Aims and Objectives:** This study aims to correlate body mass index (BMI), visceral fat, and subcutaneous fat individually with glycemic status of the subject and assess the better predictor of insulin resistance. **Materials and Methods:** Detailed questionnaire concerned with the study and informed consent was obtained from the subjects. The study included 100 middle-aged non-diabetic offspring of diabetic parents. The mean age of diabetic subjects was 39.4 years. Age - 35–45 years, subjects with BMI in the range between 18.5 and 30 kg/m^2 were included in the study while diabetics, hypertensives, alcoholics, smokers, and subjects with any other recent illness were excluded from the study. Visceral fat and subcutaneous fat were measured using BC-601 TANITA bioimpedance analyzer. Fasting blood sugar was measured using single prick glucometer technique. **Results:** Pearson correlation was applied. Fasting blood sugar showed significant positive correlation with visceral fat and subcutaneous fat with r value of + 0.329 and + 0.464, respectively. Fasting blood sugar did not correlate with BMI and has an r value of + 0.050 only. **Conclusion:** Visceral fat and subcutaneous fat remain a better predictor than BMI in monitoring the preponderance to insulin resistance.

KEY WORDS: Visceral Fat; Subcutaneous Fat; Body Mass Index; Fasting Blood Sugar

INTRODUCTION

Genetic predisposition is a key factor in developing insulin resistance in prone individuals. Sedentary lifestyle, lack of regular physical exercise, excessive intake of junk foods, and stress are proved to be important factor in the development of obesity.[1] Insulin resistance, abdominal obesity, and diabetes are part of metabolic syndrome along with blood lipid disorders and inflammation and are at risk of cardiovascular diseases. The visceral fat is a risk factor for cardiovascular disease, diabetes, impaired glucose metabolism, insulin resistance, endothelial dysfunction, dyslipidemia, hypertension, sleep apnea, and non-alcoholic fatty liver disease.[2-7]

Type 2 diabetes mellitus (T2DM) is one of the most prevalent health problems across the world. Insulin resistance is the key factor for developing T2DM. Obesity and T2DM are well related.[8,9] The International Association for the Study

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of Obesity and International Obesity Taskforce reports showed 475 million adults are obese and overweight globally.[10] Majority of diabetic patients were overweight and had abdominal adiposity and high triglyceride level.[11] Truncal region and peripheral region fat are the adipose tissues in our body. Subcutaneous fat in thoracic and abdominal region is truncal adipose tissues.[12]

Studies show in obese non-diabetic and T2DM subjects; intra-abdominal visceral fat is closely associated with insulin resistance.[13-15] In contrast, studies revealed that in obese individuals compared to visceral fat and subcutaneous fat are the best predictor of insulin resistance.[16-18] Even in people with normal body mass index not subcutaneous adiposity, but visceral fat adiposity has shown high association with type 2 diabetes and mortality.[19,20] Free fatty acids are responsible for the insulin resistance in case of obesity which was studied using isotopic techniques.[21] It showed total adiposity is superior to visceral adiposity in developing insulin resistance. Thus, adipose tissue location is a better predictor of metabolic diseases. The pathogenesis for developing insulin resistance is the expression of adipocytes releasing cytokines including interleukin-6 and tumor necrosis factor-α.[22] Insulin resistance can be contributed by the non-esterified fatty acids and cytokines from visceral fat which enters the portal vein. In our study, offspring of T2DM was included because studies have shown that metabolic defects in this group develop early due to genetic predisposition[23] and for developing type 2 diabetes later. Need has arisen to find the sole adipose tissue depot as a predictor of insulin resistance and metabolic syndrome.

There are many studies using body mass index (BMI), waist–hip ratio, skinfold thickness to assess the obesity, and its complications. Studies have reported that visceral fat percentage and subcutaneous fat percentage are a better predictor than BMI in analyzing the complications due to obesity. There is a paucity of data in our country analyzing body composition among non-diabetic offspring of diabetic parents. This cross-sectional study was aimed to correlate visceral fat, subcutaneous fat, and BMI with glycemic status of the subjects and identify the better predictor of insulin resistance.

MATERIALS AND METHODS

Study Design

This is a cross-sectional study.

Written informed consent was obtained from the subjects to participate in this study. Questionnaire regarding family history (diabetes and hypertension), alcohol consumption, sociodemographic characteristics, drug intake, diet history, cigarette smoking, exercise history, occupational history, and history of recent illness was taken. The institutional ethical committee clearance was obtained.

Subjects

Inclusion criteria

100 male offspring of diabetic parent (fasting blood sugar < 100 mg%, either parent or single parent diabetic), age group - 35–45 years, and BMI between 18.5 and <or equal to 30 are included in the study.

Exclusion criteria

Diabetics, hypertensives, alcoholics, smokers, and subjects with any other illness in past few days are excluded from the study.

BC-601 TANITA segmental body composition analyzer is used for Bioelectric Impedance Analysis (BIA) to measure visceral fat and subcutaneous fat.[24]

Preparations to do bioimpedance analysis

After detailed diet and exercise history data were measured. Measurement is taken 3 times and average is taken. Resistance and impedance offered by water in muscle and fat was measured using BIA. Known amount of current (I), of about 800 μ A at a frequency of 50 kHz, is introduced into the body. According to Ohm's law, voltage difference between source and sink electrodes was measured in the body volume which acts as a volume conductor in living tissues. Results thus obtained are analyzed. BMI was calculated. Fasting blood sugar measured using single prick glucometer technique.

RESULTS

SPSS software 16.0 was used for statistical analysis and Pearson correlation was done. Descriptive statistics of age, visceral fat, subcutaneous fat, fasting blood sugar, and BMI of 100 middle-aged male offspring of diabetic parent mentioned in Tables 1 and 2 (enclosed) show Pearson correlation of fasting blood sugar with BMI, subcutaneous fat, and visceral fat.

DISCUSSION

Our study showed a significant positive correlation between visceral fat and subcutaneous fat with fasting blood sugar in the middle-aged male offspring of diabetic parents similar to a study conducted by Shahid *et al*.[23] Our study did not show positive correlation between BMI and fasting blood sugar which is a strong predictor of insulin resistance.[25,26]

A study conducted by Usui *et al*. mentioned that in non-diabetic people sex, age, and VO2max did not show significance, but visceral fat showed positive correlation with insulin resistance.[27] Swinburn *et al*. and Durenberg *et al*. have shown that the WHO defined range of

Table 1: Description of age, visceral fat, subcutaneous fat, and fasting blood sugar

Age mean±SD (in years)	Fasting blood Sugar mean±SD (g %)	Visceral fat mean±SD (%)	Subcutaneous fat mean±SD (%)	BMI kg/m²
39±4	89±8	11±5	29±6	25±3

BMI: Body mass index, SD: Standard deviation

Table 2: Correlation between visceral fat and subcutaneous fat with fasting blood sugar

	Fasting blood sugar g %
Visceral fat %	r value+0.329
Subcutaneous fat %	r value+0 0.464
BMI kg/m²	r value+0.050

BMI: Body mass index

overweight in relation to body mass index (BMI) did not adequately reflect the actual insulin resistance status[28,29] to assess obesity-related morbidity. The study conducted by Sikri *et al*. said even in the normal range of BMI put up by the WHO showed risk factors for hypertension and type 2 diabetes.[30] Gómez-Ambrosi *et al*. reported in their study male subjects over the age of 40 years with BMI <25 kg/m²; body fat percentage (BF%) may help to predict insulin resistance better. A study conducted by Shea *et al*.[32] also indicated appropriate parameters which can increase the risk of cardiovascular diseases are subcutaneous fat and visceral fat percentage even in the normal range of BMI. Our study provides screening tools to identify these individuals to prevent them from obesity-associated disease by measuring subcutaneous fat and visceral fat percentage.

The close relationship between metabolic disorders and BF distribution has been reported.[33,34] A study conducted by Fujimoto *et al*. also mentioned the measurement of visceral fat accumulation is the important step in the assessment of atherosclerosis risk.[35] Subcutaneous fat remains as a major contributor for metabolic disturbances as shown by Pavankumar *et al*. Subcutaneous truncal adipose tissue has significant impact in the development of insulin resistance. Subcutaneous adipose tissue acts as a metabolic buffer. Adipocyte hypertrophy decreased adipogenesis, and angiogenesis makes the subcutaneous adipose tissue dysfunctional. Tissue damage can be caused by the excess free fatty acids spill over into adjacent ectopic tissues.[36] A study conducted by Suk Jung *et al*. and Kim *et al*.[37,38] showed visceral fat remains as better predictor than subcutaneous fat for insulin resistance.

Our study is strengthened by measuring regional fat such as visceral and subcutaneous fat percentage instead of whole BF and correlated with fasting blood sugar. Limitation of our study is that we did not measure plasma insulin level which could have substantiated the study result.

Implication

Lifestyle modification remains as key intervention for the better functioning of adipose tissue and prevents the inclination toward insulin resistance and subsequent morbidity due to cardiovascular and metabolic diseases.

CONCLUSION

Visceral fat and subcutaneous fat remain a better predictor than BMI in early assessment of insulin resistance.

REFERENCES

1. Brozak J, Keys A. The evaluation of leanness-fatness in man: Norms and interrelationships food and agricultural organization of the United Nations. Dietary Surv 1949;62:194-206.
2. Després JP, Lemieux A. Abdominal obesity and metabolic syndrome. Nature 2006;444:881-7.
3. Go A, Mozaffarian D, Roger VL, Benjamin EJ, Berry JD, Blaha MJ, *et al*. Heart disease and stroke statistics-2015 update a report from the American Heart Association. Circulation 2015;131:e29-322.
4. Ritchie SA, Connell JM. The link between abdominal obesity, metabolic syndrome and cardio vascular disease. Nutr Metab Cardiovasc Dis 2007;17:319-26.
5. Fox CS, Massaro JM, Hoffmann U, Pou KM, Maurovich-Horvat P, Liu CY, *et al*. Abdominal visceral and subcutaneous adipose tissue compartments: Association with metabolic risk factors in the Framingham heart study. Circulation 2007;116:39-48.
6. Oh TH, Byeon JS, Myung SJ, Yang SK, Choi KS, Chung JW, *et al*. Visceral obesity as a risk factor for colorectal neoplasm. J Gastroenterol Hepatol 2008;23:411-7.
7. Von Hafe P, Pina F, Pérez A, Tavares M, Barros H. Visceral fat accumulation as a risk factor for prostate cancer. Obes Res 2004;12:1930-5.
8. Diehl AK, Stern MP. Social health problems of Mexican-American: Obesity, gallbladder disease, diabetes mellitus, and cardiovascular disease. Adv Intern Med 1989;34:73-96.
9. Hamman RF. Genetic and environmental determinations of non-insulin dependent diabetes mellitus (NIDDM). Diabetes Metab Rev 1992;8:287-338.
10. Golay A, Thompson J, Kolka R. International Association for the Study of Obesity Nick Finer. Available from: http://www.iaso.org/iotf/obesity/obesitytheglobalepidemic. [Last accessed on 24 Apr 2018].
11. Menon S, Venugopal R. A comparative study of lipid profile, body mass index, and waist circumference among Type 2 diabetes mellitus patients with poor and good metabolic control and normal age-matched control group. Natl J Physiol Pharm

Pharmacol 2018;8:239-43.
12. Garg AJ. Regional adiposity and insulin resistance. Clin Endocrinol Metab 2004;89:4206-10.
13. 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.
14. Basu A, Basu R, Shah P, Vella A, Rizza RA, Jensen MD. Systemic and regional fatty acid metabolism in Type 2 diabetes. Am J Physiol 2001;280:E1000-6.
15. Bonora E, Del Prato S, Bonadonna RC, Gulli G, Solini A, Shank ML, *et al*. Total body fat content and fat topography are associated differently with *in vivo* glucose metabolism in nonobese and obese nondiabetic women. Diabetes 1992;41:1151-9.
16. Abate N, Garg A, Peshock RM, Stray-Gunderson J, Grundy SM. Relationship of generalized and original adiposity to insulin sensitivity in men. J Clin Invest 1995;96:88-98.
17. Abate N, Garg A, Peshock RM, Stray-Gunderson J, Adams-Huet B, Grundy SM. Relationship of generalized and original adiposity to insulin sensitivity in men with NIDDM. Diabetes 1996;45:1684-93.
18. Frayn KN. Visceral fat and insulin resistance-causative or correlative? Br J Nutr 2000;83 Suppl 1:S71-7.
19. Pischon T, Schlattmann P, Fankhaenel T, Goebel H. General and abdominal adiposity and risk of death in Europe. N Engl J Med 2008;359:2105-20.
20. Cohen P, Levy JD, Zhang Y, Frontini A, Kolodin DP, Svensson KJ, *et al*. Ablation of PRDM16 and beige adipose causes metabolic dysfunction and a subcutaneous to visceral fat switch. Cell 2014;156:304-16.
21. Nielsen S, Guo ZK, Johnson M, Hensrud DD, Jensen MD. Splanchnic lipolysis in obesity. J Clin Invest 2004;113:1582-8.
22. Mohamed-Ali V, Pinkney JH, Coppack SW. Adipose tissue as an endocrine and paracrine organ. Int J Obesity 1999;22:1145-58.
23. Shahid A, Lone KP, Saeed S, Arslan M. Male offspring of both diabetic parents have higher insulin resistance and serum leptin levels compared to those with one diabetic parent. Hormones 2008;7:313-9.
24. Jebb SA, Cole TJ, Doman D, Murgatroyd PR, Prentice AM. Evaluation of the novel Tanita body-fat analyser to measure body composition by comparison with a four-compartment model. Br J Nutr 2000;83:115-22.
25. Stovall DW, Bailey AP, Pastore LM. Assessment of insulin resistance and impaired glucose tolerance in lean women with polycystic ovary syndrome. J Womens Health (Larchmt) 2011;20:37-43.
26. Lin-Tan DT, Lin JL, Wang LH, Wang LM, Huang LM, Liu L, *et al*. Fasting glucose levels in predicting 1-year all-cause mortality in patients who do not have Diabetes and are on maintenance hemodialysis. J Am Soc Nephrol 2007;18:82385-91.
27. Usui C, Asaka M, Kawano H, Aoyama T, Ishijima T, Sakamoto S, *et al*. Visceral fat is a strong predictor of insulin resistance regardless of cardiorespiratory fitness in non-diabetic people. J Nutr Sci Vitaminol (Tokyo) 2010;56:109-16.
28. Swinburn BA, Ley SJ, Carmichael HE, Plank LD. Body size and composition in polynesians. Int J Obes 1999;23:1178-83.
29. Durenberg P, Yap M, Stavern van WA. Body mass index and percent body fat. A meta analysis among different ethnic groups. Int J Obes 1998;22:1164-71.
30. Sikri G, Garg MK. Body mass index and obesity. Tailoring "cut off" for an Asian Indian population. Med J Armed Force India 2008;64:350-3.
31. Gómez-Ambrosi J, Silva C, Galofré JC, Escalada J, Santos S, Gil MJ. Body adiposity and Type 2 diabetes: Increased risk with a high body fat percentage even having a normal BMI. Obesity (Silver Spring) 2011;19:1439-44.
32. Shea JL, King MT, Yi Y, Gulliver W. Body fat percentage is associated with cardiometabolic dysregulation in BMI defined normal weight subjects Nutrition, metabolism, and cardiovascular diseases. Nutr Metabol Cardiovasc Dis 2012;22:741-7.
33. Nakamura T, Tokunaga K, Shimomura I, Nishida M, Yoshida S, Kotani K, *et al*. Contribution of visceral fat accumulation to the development of coronary artery disease in non-obese men. Atherosclerosis 1994;107:239-46.
34. Després JP, Lemieux A. Abdominal obesity and metabolic syndrome. Nature 2006;444:881-7.
35. Fujimoto WY, Bergstrom RW, Boyko EJ, Chen KW, Leonetti DL, Newell-Morris L, *et al.* Visceral adiposity and incident coronary heart disease in Japanese-American men. The 10-year follow-up results of the Seattle Japanese-American Community Diabetes Study. Diabetes Care 1999;22:1808-12.
36. Patel P, Abate N. Body fat distribution and insulin resistance. Nutrients 2013;5:2019-27.
37. Jung SH, Ha KH, Kim DJ. Visceral fat mass has stronger associations with diabetes and prediabetes than other anthropometric obesity indicators among Korean adults. Yonsei Med J 2016;57:674-80.
38. Kim K, Park SM. Association of muscle mass and fat mass with insulin resistance and the prevalence of metabolic syndrome in Korean adults: A cross-sectional study. Sci Rep 2018;8:2703.

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