

Association of Type 2 Diabetes mellitus with Obesity

T. Alaguveni, P. Sai Kumar and S. Kaviprasanna

Department of Physiology, Sree Balaji Medical College, Bharath University, India

Abstract: *Background:* Type 2 Diabetes Mellitus (DM) is the most common health problem in India. Obesity increases the risk for type 2 DM. Lipotoxicity constitutes an important pathogenic link between obesity and type 2 DM. *Aim & Objectives:* The aim of the study is to find out the association of type 2 DM with obesity. *Methods & Materials:* A cross sectional study was done among type 2 DM in the year of 2013. A sample of 300 type 2 DM patients was randomly selected for this study. Among these, 129 were men and 171 were women. All patients completed an interviewer-administrated questionnaire and underwent complete clinical examination. BMI was calculated as per WHO criteria. Results revealed that in among these 300 type 2 DM patients, 249 were obese (Men-78, Women-171). The study indicated more prevalence of obesity among women than men. *In Conclusion:* The risk of type 2 diabetes increases exponentially as BMI increases above about 25 kg/m². There is a positive relationship between obesity and type 2DM. If BMI increases, the risk on type 2 DM will also increases.

Key words: Type 2 Diabetes Mellitus • Obesity • Body Mass Index

INTRODUCTION

Type 2 DM is strongly associated with obesity [1]. The risk of type 2 diabetes increases exponentially as BMI increases above about 25 kg/m². Compared with a normal BMI of 22 kg/m², the risk of type 2 diabetes is increased by two to eight-fold at BMI 25, 10-40-fold at BMI > 30 and > 40-FOLD AT BMI > 35 depending on age, gender, duration and distribution of adiposity. Excessive deposition of lipid in muscle and liver also enhances the risk of type 2 DM through mechanisms of intracellular lipotoxicity.

Obesity is more common amongst people of Hispanic and black ethnicities and the increased risk of type 2 diabetes may be slightly greater than in obese people of European origin [2, 3]. A BMI of 30-35 increased the occurrence of type 2 diabetes by > 20 fold in women and > 10 fold in men [4,5].

It is generally held that an accumulation of abdominal fat ('central' obesity), as indicated by an increased waist:hip ratio is an independent risk for type 2 diabetes irrespective of the extent of obesity[6]. Excess adiposity is well known to promote the onset and severity of insulin resistance, contributing to emergence and progression of IGT and type 2 diabetes. Indeed, around two thirds to

three-quarters of individuals who develop diabetes have a history of significant overweight or obesity making chronic excess adiposity the strongest risk factor for type 2 diabetes.

WHO criteria (underweight: BMI<18.5 kg/m²; normal: BMI 18.5-24.9; overweight: BMI 25.0-29.9; obese BMI 30.0). Participants were considered centrally obese if waist-hip ratio (WHR) was 0.95 or over for men and 0.85 or over for women.

MATERIALS AND METHODS

General Cross-sectional study in a random sample of 300 Type 2 DM patients treated under primary care during the year of 2013. This study was done to find out the association of Type 2 DM with obesity. Study was approved by institutional research and ethics committee. Patients were screened for eligibility for this study. After receiving a prior informed written consent that is written one from literate patient, orally informed one from illiterate patients. Patients with congestive cardiac failure, UTI, recent MI, febrile patients, marked hypertension, pregnant patients, patients who undergone bariatric surgeries, patients confined to bed for more than 2 weeks were excluded from the study.

Participant patients were interviewed about DM type, duration, treatment profile, level of control. Other causes of obesity like endocrine disorders, hormonal imbalance and steroid overuse were ruled out.

Complete clinical examination was done in all patients. BMI was calculated from the height and weight measurements of the patients. Height was measured without shoes. BMI was calculated by weight in kg divided by height in meters squared. The WHO classification for BMI was used to find out the degree of obesity [7]. BP was measured in the early morning before taking blood samples by using standard mercury sphygmomanometer after a 15 minutes rest.

Statistical Analysis: Data was collected using standardized protocol. The blood sugar was measured by glucose oxidation test and by digital glucometer at the community level due to logistic reason. BMI was calculated as per WHO criteria by using height and weight. Obtained data were cleaned, coded, recoded and edited. The analysis was done using SPSS 15.0 statistical software. Chi square test and student t-test were used for statistical analysis. P-value below 0.05 considered as statistically significant. To measure association between variables in diabetics and non diabetics a multiple logistic regression was performed.

RESULTS

The table showed overall gender prevalence of obesity among type 2 DM.

As displayed in the table, 123 (41.0%) of the respondents were overweight (BMI =25 kg/m²) while 63 (21.0%) were obese (BMI =30 kg/m²), 42 (14.0%) were class1 obesity, 14 (4.7%) were class2 obesity and 7 (2.3%) were class3 obesity. Among 129 men 54 (41.9%) were overweight, 12 (9.3 %) were border line obesity. 10 (7.8%) were class1 obesity, 2 (1.6%) were class2 obesity and no man attained the level of class3 obesity as compared to the women (0% vs 4.1%) The prevalence of overweight was significantly higher among women compared to man

respondents (40.4% vs 41.9%, $p < 0.001$); likewise, obesity was more common in women (29.8% vs 9.3%, $p < 0.001$). All classes of obesity were also more common in woman respondents. As the p value was < 0.05 , this study was statistically significant.

DISCUSSION

The earliest alterations of beta cell function are a small delay and reduction in the acute 'first' phase of glucose-induced insulin secretion and an increased and protracted chronic 'second' phase response. The normal pulsatile rhythm of basal insulin secretion is also disturbed and the proportion of prandially secreted proinsulin is increased.

These observations suggest the initial sensing of an increased glucose stimulus and the intracellular processing of proinsulin to insulin become defective [8, 9]. By the time insulin resistance is established and glucose tolerance is becoming impaired the first phase of glucose-induced insulin secretion is substantially reduced. While the second phase is markedly enhanced and extended. This is customarily interpreted as a compensatory adaptation of the beta cells to address the modest emergent hyperglycaemia in the face of insulin resistance. Obesity is typically associated with an increase in beta cell mass involving hypertrophy and hyperplasia, Butler et al. [10] supporting the concept of an adaptation to meet an increased demand for insulin. At this stage the beta cells remain rich in insulin granules, suggesting that it is the acute component of the stimulus secretion process that is inadequate.

While loss of normal insulin sensitivity is well recognized to adversely affect nutrient metabolism in adipose tissue, muscle and liver of obese individuals, it is also apparent that the pancreatic beta cells themselves are susceptible to insulin resistance. Gene knockout studies have shown that insulin signaling via IRS proteins is important for beta cell viability and replication. Loss of insulin sensitivity of the beta cell appears to restrict the adaptive changes in beta cell mass and could hasten progression from IGT to type 2 diabetes.

Table 1: The total studied 300 patients was 129 men and 171 women.

BMI*	Overall and gender prevalence of obesity among type 2DM			p value
	Men (n = 129); ----- n (%)	Women (n = 171); ----- n (%)	Overall (n = 300); ----- n (%)	
Overweight	54 (41.9)	69 (40.4)	123 (41.0)	0.0005*
Obesity	12 (9.3)	51 (29.8)	63 (21.0)	0.00002*
Class 1	10 (7.8)	32 (18.7)	42 (14.0)	0.007*
Class 2	2 (1.6)	12 (7.0)	14 (4.7)	0.026*
Class 3	0 (0.0)	7 (4.1)	7 (2.3)	0.05

*Body mass index; * $p < 0.05$

The finding that a physically active lifestyle is associated with a lower incidence of type 2 diabetes has been shown in several prospective studies. Physical activity was inversely related to the incidence of type 2 diabetes among man alumni from the University of Pennsylvania [11], a relation that was particularly evident in men at high risk for developing diabetes (defined as those with a high body mass index, a history of hypertension, or a parental history of diabetes).

CONCLUSIONS

Obesity plays an important role in the development of type 2 diabetes. This study showed that prevalence of obesity is more common in women compared to men as per previous studies. There is a positive linear relationship between obesity and type 2 DM. Further investigations are needed to study the molecular mechanism of obesity for insulin resistance and diabetes in different populations. Since genetic predisposition plays an important role in the genesis of insulin resistance.

REFERENCES

1. Fagot-Campagna, A., B. Balkau, D. Simon, J.M. Warnet, J.R. Claude, P. Ducimetere, E. Eschwege, 1998. High free fatty acid concentration: an independent risk factor for hypertension in the Paris Prospective study. *Int J. Epidemiol*, 27: 808-813.
2. Mokdad, A.H., E.S. Ford, B.A. Bowman, W.H. Dietz, F. Vinicor, V.S. Bales and J.S. Marks, 2001. Prevalence of obesity, diabetes and obesity-related health risk factors *JAMA* 2003, 289:76-9.
3. Bray, G.A., K.A. Jablonski, W.Y. Fujimoto, E. Barrett-Connor, S. Haffner, R.L. Hanson, J.O. Hill, V. Hubbard, A. Kriska, E. Stamm and F.X. Pi-Sunyer, 2008. Relation of central adiposity and body mass index to the development of diabetes in the Diabetes Prevention Program. *Am J. Clin Nutr*; 87: 1212-8.
4. Hu, F.B., J.E. Manson, M.J. Stampfer, G. Colditz, S. Liu, C.G. Solomon and W.C. Willett, 2001. Diet, lifestyle and the risk of type 2 diabetes mellitus in women. *N Engl J. Med*, 345: 730-37.
5. Must, A., J. Spandano, E.H. Coakley, A.E. Field, G. Colditz and W.H. Dietz, 1999. The disease burden associated with overweight and obesity. *JAMA*, pp: 282:153-9.
6. Montague, C.T. and S.O'. Rahilly, 2000. The perils of portliness: causes and consequences of visceral adiposity. *Diabetes*, 49: 883-8.
7. WHO, 2004. Expert consultation: Appropriate BMI for asian populations and its implications for policy and intervention strategies. *The Lancet*, 363: 157-163.
8. DeFronzo, R.A., 1988. The triumvirate: beta cell, muscle, liver. A collusion responsible for NIDDM. *Diabetes*, 37: 667-87.
9. Kahn, S.E., 2003. The relative contributions of insulin resistance and beta-cell dysfunction to the pathophysiology of type 2 diabetes. *Diabetologia*, 46: 3-19.
10. Butler, A., J. Janson, S. Bonner-Weir and P. Butler, 2003. B-cell deficit and increased b-cell apoptosis in humans with type 2 diabetes. *Diabetes*, 52: 102-10.
11. Helmrigh, S.P., D.R. Ragland, R.W. Leung and R.S. Jr. Paffenbarger, 1991. Physical activity and reduced occurrence of non-insulin-dependent diabetes mellitus. *N Engl J. Med*, 325: 147-52.