Relation between Body Mass Index (BMI) and Fasting Blood Sugar (FBS)

G.S. Prema[1], R. Anitha[1], O. Padmini[1]

ABSTRACT

Introduction: Increased energy intake leads to increased fat delivery from either exogenous fat in the diet or endogenous fat from hepatic lipogenesis to muscle and liver resulting in increased fat storage in these tissues and insulin resistance. Aim of the study was to establish the relationship between fasting blood sugar and body weight.

Materials and methods: The subjects for the study are 100 males in the age group of 21 to 40 years. Control group of 50 males had BMI of 18-24 kg/m2 and study group of 50 males had BMI of 25-29kg/m2. Height and weight are recorded. Ethics committee consent was taken. Study group excluded people on treatment for diabetes mellitus and cardiovascular diseases. Biochemical Analysis: The patient was asked to fast overnight for 8-10 hours. Fasting Blood Sugar was estimated by Glucose Oxidase method.

Result: The triad of obesity, diabetes and heart diseases are interlinked and are proving the major cause for the morbidity and mortality in the world population. All stages of glucose abnormalities like prediabetes and established diabetes mellitus are associated with CAD and detection of these abnormalities is of great value in early screening of cardiovascular diseases

Conclusion: The existence of a significant direct correlation between FBS and BMI was confirmed in the present study. Our results therefore suggest that a low BMI is important for maintaining normal blood glucose levels.

Keywords: Body mass index (BMI), Fasting blood sugar (FBS), Obesity Cardio vascular disease (CVD)

INTRODUCTION

There has been a renaissance in the field of obesity research in the last ten years. Traditionally, obesity was believed to be associated with affluent lifestyles in the west. Now, however, the picture has changed & research in the field of obesity has blossomed. Obesity is increasing at an alarming rate throughout the world. Several studies in India have shown that changes in dietary patterns, physical activity levels, lifestyles associated with affluence and migration to urban areas are related to increasing frequencies of obesity. The role of vitamin fortification leading to obesity is becoming more prevalent in recent days.[1]

Obesity is an epidemic disease that threatens to inundate health care resources by increasing the incidence of diabetes, heart disease, hypertension and cancer. All obese individuals do not display a chastening of metabolic & cardiovascular risk factors and all lean individuals do not present with a healthy metabolic & disease - free profile. But most of the time obesity is associated with abnormal glucose and lipid profiles. Studies indicate that the presence of obesity increases the risk for developing diabetes and cardiovascular diseases.[2]

In obesity as excessive adipose tissue accumulates, an altered metabolic profile occurs along with a variety of adaptations and alterations in cardiovascular structure and function even in the absence of co-morbidities.

Adipose tissue excess or obesity, particularly in the visceral compartment, is associated with insulin resistance, hyperglycemia, dyslipidemia. hypertension, and prothrombotic and proinflammatory states. The most common cause of insulin resistance occurs when energy intake exceeds the metabolic rate leading to obesity.

Increased energy intake leads to increased fat delivery from either exogenous fat in the diet or endogenous fat from hepatic lipogenesis to muscle and liver, resulting in increased fat storage in these tissues and insulin resistance. Fatty acid synthesis in the body can also occur from increased carbohydrates like glucose.[3]

Circulating insulin levels are elevated and correlate inversely with insulin receptor number. If insulin levels are lowered for example by diet or drugs that interfere with insulin secretion, the receptor number returns to normal even though the degree of obesity may not be significantly changed. This suggests that the number of insulin receptors in insulin resistant obese patients may result from the effects of excess insulin on normal pathway of receptor down regulation. The observation that insulin receptor number can be restored to normal if insulin levels are decreased in obese patients suggests that the changes in receptor number are secondary to the insulin resistance and not a primary causal factor.

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Type II diabetes mellitus is strongly associated with overweight in both genders in all ethnic groups.[4]
Smith etal found that human adipocytes with proper invitro conditions will take up glucose and enlarge their fat content. Insulin sensitivity is inversely correlated with cell size.[5]
Through the use of BMI the epidemic of obesity that began in the 1980s has been tracked through the end of the century. The original alarm was sounded in 1994 by the National center for health statistics in USA when they reported the data from the National health and Nutrition Examination survey (NHANES).
In recent years, the body mass index(BMI) has become the medical standard used to measure overweight and obesity. BMI can be considered to provide the most useful, albirt crude population - level measure of obesity. Men and women with a BMI of 25.0 to 29.9 kg/m2 are considered overweight, and those with a BMI 30 kg/m2 or greater are considered obese. The prevalence of obesity - related diseases, such as diabetes, begins to increase at BMI values around 25 kg/m^2. Obese persons with excess abdominal fat are at higher risk for diabetes, hypertension, dyslipidemia, and ischemic heart disease than obese persons whose fat is located predominantly in the lower body[6]
In obese adults, type 2 diabetes develops over a long period, and impaired glucose tolerance can be a predictor for the risk of development of diabetes and cardiovascular disease.[7]
FBS < 100 mg/dl is considered normal and between 100–125 mg/dl along with Hb A1C 5.7%-6.4% is marked as impaired fasting glucose or prediabetes. FBS≥ 126 mg/dl along with Hb A1C ≥6.5% is defined as diabetes mellitus.[8]
It has been recognized that prediabetic hyperglycemia -fasting plasma glucose of 110 to 125 mg/dl confers an increased risk for cardiovascular disease.[9] The prevalence of diabetes and prediabetes are known to relate with higher ranger of waist circumference, waist /hip ratio and body mass index.[10]
According to ADA guidelines, the risk factors for diabetes are:-

- Physical inactivity
- First-degree relative with diabetes
- High-risk race/ethnicity
- Women who delivered a baby >9 lb or were diagnosed with gestational diabetes mellitus
- HDL-C <35 mg/dL ± TG >250 mg/dL
- Hypertension (≥140/90 mm Hg or on therapy)
- Hb A1C ≥5.7%
- Conditions associated with insulin resistance: severe obesity, acanthosis nigrica, and

Present study was under taken to emphasie the relation between BMI >25kg/m^2 and fasting blood sugar.

MATERIALS AND METHODS

The subjects for the study were 100 males in the age group of 21 to 40 years. Control group of 50 males had BMI of 18-24 kg/m2 and study group of 50 males had BMI of 25-29kg/m2. Height and weight are recorded. Ethics committee consent was taken.
Study group excluded people on treatment for diabetes mellitus and cardiovascular diseases.

The commonly employed measurements and calculations are as follows
Height: standing height is measured using a stadiometer, with an accuracy of 0.1 cm graduations and sliding headpiece. The measurement is taken with the subject wearing no shoes, standing erect on a horizontal surface with heels together, the shoulders relaxed and arms at the sides.
Weight: weight assessment provides important data in assessing status of an individual and serves as an indicator of intentional or unintentional weight loss.
Body weight = body fat + lean body mass (fat free mass).
BMI (Body Mass Index) or Quetelet Index:-
It is a statistical measure of the weight of a person scaled according to height. It is used as a simple means of classifying inactive individuals of an average body composition according to their body fat content. It was originally developed between 1830 and 1850 by the Belgian polymath, Adolphe Quetelet during the course of developing Social Physics.
It is a reliable and easily obtainable objective anthropometric criterion for the definition and diagnosis as well as an estimate of the severity of undernutrition or chronic energy deficiency (CED) in adults.[11]
BMI was calculated from the following equation

$$\text{Body Mass Index (kg/m}^2\text{)} = \frac{\text{Weight in Kg}}{\text{Height in m}^2}$$

The Body Mass Index value ranging between 18.5 -25.0 is considered as normal, < 18.5 indicates the status as undernourished, while value above 25 as overweight and above 30.0 as obese.
Biochemical Analysis:- The patient was asked to fast overnight for 8-10 hours. Fasting Blood Sugar was estimated by Glucose Oxidase method.

RESULTS

Paired T test is done . Results show that 40/50 subjects with BMI<25kg/m^2 have FBS of <100mg/dl (Graph-1). 38/50 Subjects with BMI of >25kg/m^2 have FBS of >100mg/dl. (Graph-2) Significant p value is seen. The study shows that Body Mass Index is directly related to Fasting Blood Sugar.

DISCUSSION

The triad of obesity, diabetes and heart diseases are interlinked and are proving the major cause for the morbidity and mortality in the world population.
Diabetes mellitus is one of the leading risk factors of Coronary Artery Disease and is growing in developing countries because of the changes in lifestyles, increasing high-calorie diet and physical inactivity.[11] Obesity, diabetes mellitus and

BMI Kg/m²	FBS<100 mg/dl	FBS>100 mg/dl	Total
BMI<25	40	10	50
BMI>25	12	38	50
Total	48	52	100

Table-1: Comparision of BMI with fasting blood sugar

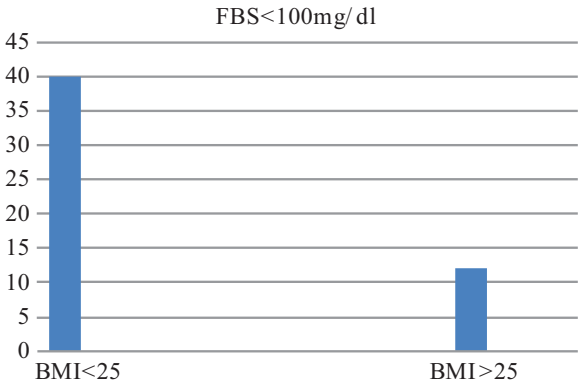

Figure-1: BMI versus FBS

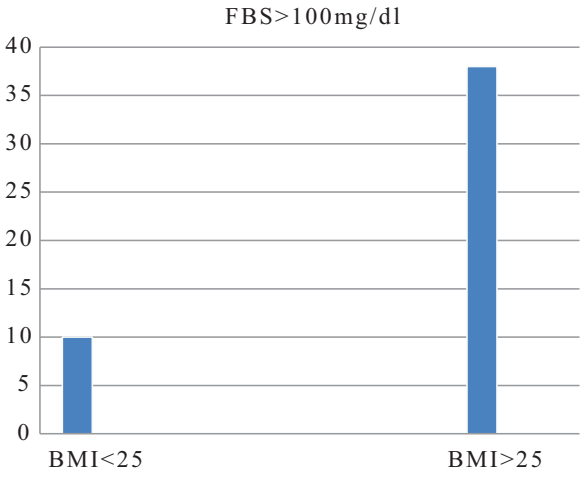

Figure-2: BMI versus FBS

hypertension are common, interrelated medical problem in westernized, industrialized societies

All stages of glucose abnormalities like prediabetes and established diabetes mellitus are associated with CAD and detection of these abnormalities is of great value in early screening of cardiovascular diseases.[12,13]

Patients put on hypolipidemic drugs like fenofibrate are known to show increased insulin sensitivity thereby effecting glycemic control of the patients.[14]

Elevated fasting serum glucose level and a diagnosis of diabetes are independent risk factors for several major cancers and the risk tends to increase with an increased level of fasting serum glucose.

Recent studies point towards RESISTIN. Resistin is an adipokine secreted from adipose tissue and monocytes. It is named for its ability to resist or interfere with insulin action It was proposed as a link between obesity and diabetes.[15,16]

In obese individuals, adipose tissue releases increased amounts of non-esterified fatty acids, glycerol, hormones, pro-inflammatory cytokines and other factors that are involved in the development of insulin resistance.[17]

The life style of present generation adolescents makes them more prone for obesity and diabetes. Early detection and therapy of the obese adolescent with a family history of type 2 diabetes may interrupt the cycle of weight gain and insulin resistance that leads to glucose intolerance in adulthood.[18]

Studies show that therapy with extended release niacin may increase fasting blood sugar into the diabetic range, especially for obese patients.[19]

Our study is done to re-establish the direct relationship between increased body weight and fasting blood sugar. In the control group of BMI <25, majority of people showed fasting blood sugar of less than 100mg/dl (Graph-1). In the study group with BMI>25, majority of people showed FBS >100mg/dl (Graph-2). As we have excluded patients of diabetes mellitus and cardiovascular diseases in our study, other causes for the increased FBS leading to prediabetes, like the role of stress in daily life should be considered. Stress hormones are known to increase blood glucose levels. Physical and emotional stress increases these hormones thereby increasing blood glucose levels.

Increased blood glucose levels increase insulin levels in the body which in turn increase nor adrenalin. It is known to induce insulin resistance. Family history of diabetes should also be considered. Genetic cause for insulin resistance cannot be ruled out. As untreated prediabetes can progress to diabetes which can lead to microvascular complications effecting heart, kidney, retina, nerves etc., and can cause morbidity in the effected.

Measures should be taken by the prediabetics to keep the sugar levels normal from reaching diabetic levels by changing the lifestyle, increasing physical activity, consuming food having low glycemic index with high fiber content and frequent monitoring of blood glucose levels along with Hb A1c.

Prediabetic state is a 'grey zone' which implies a declining glucose homeostatic efficiency. Though only 25% of cases progress to full blown T2DM, when combined with obesity (BMI >25), it is a definite predictor of onset of T2DM in due course. Moreover, complications particularly cardiovascular abnormalities begin in prediabetic phase surreptitiously even before overt Diabetes is medically diagnosed. So as altered glycemic control points to insulin resistance in early diabetes and obesity, identifying this phase by simple tests like estimation of FBS, HbA1c goes a long way in preventing the onslaught of T2DM. Hence our study was an attempt at correlating body weight with FBS levels to prevent this metabolic syndrome with lifestyle modifications before it does major systemic damage

CONCLUSION

The existence of a significant direct correlation between FBS

and BMI was confirmed in the present study. Our results therefore suggest that a low BMI is important for maintaining normal blood glucose levels.

This study highlights the critical importance of early intervention directed at treatment of obesity in association with normal blood glucose levels to avert the long-term consequences of obesity and diabetes mellitus on the development of various complications.

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REFERENCES

1. Zhou SS[1], Zhou Y - Excess vitamin intake: An unrecognized risk factor for obesity. World J Diabetes. 2014 15;5:1-13.
2. Cassano PA, Segal MR, Vokonas PS Weiss ST: Body fat distribution, blood pressure and Hypertension Ann Epidomology 1990: 1: 33 - 48.
3. Nelson DL and Cox MM: Lehninger Principles of Biochemistry. 4th edition, W.H Freeman and Company, New York. 2005.
4. Colditz GA Willett WC Rotintzky A Manson JE 1995 weight gain as a risk factor for clinical diabetes mellitus in women Ann Intern Med 122; 481-486.
5. Smith U: Insulin responsiveness and lipid synthesis in human fat cells of different sizes: effect of the incubation medium Biochem Biophys Acta 218: 417-423; 1970
6. Kissebah AH, Videlingum N, Murray R, et al. Relation of body fat distribution to metabolic complications of obesity. J Chin /endoccrinol Metab 1982; 54: 254-260.
7. Sinha R, Fish G, Teagu B, Tamborlan Wv, Banyas B, Allen K. Prevalence of impaired glucose tolerance test among children and adolescents with marked obesity. N Engl J Med 2002;346:802-10.
8. Diabetes Care. 2015;38(suppl 1):S1-S93.
9. Levitzky YS, Pencina MJ, D'Agostino RB, Meigs JB, Murabito JM, Vasan RS, et al. Impact of Impaired Fasting Glucose on Cardiovascular Disease. J Am Coll Cardiol 2008;51:264-70.
10. Azizi F, Esmaillzadeh A, Mirmiran P, Ainy E. Is there an independent association between waist-to-hip ratio and cardiovascular risk factors in overweight and obese women? Int J Cardiol 2005; 101:39-46
11. Sadeghi M, Roohafza H, et al. Diabetes and associated cardiovascular risk factors in iran: The isfahan healthy heart programme. Ann Acad Med Singapore 2007;36:175-80.
12. Mellbin LG, Anselmino M, Rydén L. Diabetes, prediabetes and cardiovascular risk. Eur J Cardiovasc Prev Rehabil. 2010;17:S9-S14.
13. Schnell O. The links between diabetes and cardiovascular disease. J Interv Cardio 2005;18:413-6.
14. Kei A, Liberopoulos E, Elisaf M - Effect of hypolipidemic treatment on glycemic profile in patients with mixed dyslipidemia. . World J Diabetes. 2013;4:365-71.
15. Steppan CM, Bailey ST et al - The hormone resistin links obesity to diabetes. Nature. 2001a;409:307–312.
16. Savage ge DB, Sewter CP, Klenk ES, et al. Resistin/Fizz3 expression in relation to obesity and peroxisome proliferator-activated receptor-gamma action in humans. Diabetes. 2001;50:2199–2202
17. Kahn SE, Hull RL, Utzschneider KM - Mechanisms linking obesity to insulin resistance and type 2 diabetes - Nature. 2006;444(7121):840-6.
18. Freemark M, Bursey D - The effects of metformin on body mass index and glucose tolerance in obese adolescents with fasting hyperinsulinemia and a family history of type 2 diabetes - Pediatrics. 2001;107:E55
19. Libby A, Meier J, et al. The effect of body mass index on fasting blood glucose and development of diabetes mellitus after initiation of extended-release niacin. Meta Synder Relat Disord. 2010;8:79-84.

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