



Correlation Between Body Mass Index and Glicosilat Haemoglobin(Hba1C) of Type 2 Diabetes Mellitus patients in Primary Health Care (PHC) in Binjai City, Sumatera Utara

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Abstract :Type 2 Diabetes Mellitus (DM) is a chronic metabolic disease which is marked by increasing blood sugar level. The people that diagnosed with type 2 DM increase every year and in 2013 Indonesia was seventh rank in the world. Main factors that can cause type 2 DM are life style changes including sedentary life style. Obesity is one of the risk factors that can cause insulin resistance and will lead to increasing blood sugar level and type 2 DM. Body Mass Index (BMI) is often used to measure the amount of tissues mass (muscle, bone and fat) in an individu which can be categorized as underweight, normoweight, overweight or obese. Increasing blood sugar level and a good control of type 2 DM can be measured reliably by measuring glicosilat haemoglobin (Hba1C) level in blood. This research is aimed to analyze the relation between BMI and Hba1C of type 2 Diabetes Mellitus patients in primary health care in Binjai city, Sumatera Utara. This research is a descriptive analytic research. The population is type 2 DM patients in four primary health care in Binjai city with sample size of 80 patients. Sampling was done by consecutive sampling and data was analyzed using correlation test, found that there was no significant relation between BMI and Hba1c of type 2 DM patients in PHC in Binjai city ($p=0,150$; $CI=95\%$).

Keywords : Body Mass Index, BMI, Type 2 Diabetes Mellitus, Diabetes Mellitus.

Introduction

Type 2 Diabetes Mellitus (DM) is a chronic metabolic disease which is caused by the body disability of producing insulin hormone that marked by increasing blood sugar level.¹ The criteria for diagnosis of diabetes mellitus as recommended by the American Diabetes Association include: 1. A1C $\geq 6.5\%$ or fasting plasma glucose [FPG] value after an 8-hour fast ≥ 126 mg/dL, or 2-hour post load glucose (PG) ≥ 200 mg/dL (11.1 mmol/L) during an OGTT, or symptoms of diabetes mellitus and a random plasma glucose concentration ≥ 200 mg/dl (11.1 mmol/ L).² The people that diagnosed with type 2 DM increase every year. The data from regional International Diabetes Federation (IDF) shows that Southeast Asia ranked second in the world³ and the number of diabetics in Indonesia was ranked seventh in the world in 2013.⁴ Main factors that can cause type 2 DM are life style changes including sedentary life style.⁵ Obesity and type 2 DM are public health problems. Obesity is one of the risk factors that can cause insulin resistance and will leads to increasing blood sugar level and type 2 DM. The increase in the prevalence of diabetes parallels that of obesity.⁶ The major link between obesity and T2DM is insulin resistance. In the natural history of diabetes, obesity and insulin resistance precede abnormal glucose. Insulin resistance in both of these conditions is mani-fested by decreased insulin-stimulated

glucose transport and metabolism in adipocytes and skeletal muscle and by impaired suppression of hepatic glucose output.⁷ In obesity the initial deposition of triglycerides occurs in subcutaneous adipose tissue and as this increases in size insulin resistance will rise and limit further subcutaneous lipid accumulation. Triglycerides will then be diverted to the visceral fat depot as well as to ectopic sites. This leads to a substantial rise in insulin resistance and the prevalence of its associated disorders. Evidence supporting this hypothesis includes studies showing that in lean subjects the prime determinant of insulin resistance is BMI, that is, subcutaneous fat, whilst in overweight and obese subjects, it is waist circumference and visceral adiposity. It has also been shown that the metabolic syndrome suddenly increases in prevalence at high levels of insulin resistance and it is suggested that this is due to the diversion of lipids from the subcutaneous to the visceral depot.⁸ WHO data show that, globally, there are more than 1 billion adults overweight and 300 million obese people. The problem of obesity is increasing in the developing world with more than 115 million people suffering from obesity related problems. Obesity rates have increased 3-fold or more since 1980 in Middle East, the Pacific Islands, Australasia, and China.^{9,10} Obesity is defined as abnormal or excessive fat accumulation.¹¹ It is measured through the Body Mass Index (BMI), a simple index of weight–height relationship that indicates amount tissues mass (muscle, bone and fat) which can be categorized as underweight, normoweight, overweight or obese. The body mass index (BMI), calculated as weight in (kg) divided by height in (m) squared.¹² Elevated body mass index (BMI) were significantly associated with type 2 DM.⁸ Increasing blood sugar level and a good control of type 2 DM can be measured reliably by measuring glycosylated haemoglobin (HbA1C) level in blood. HbA1c is haemoglobin and glucose which is specifically binds to the free amino acid groups formed from the reaction of non enzymatic by haemoglobin which is exposed by elevating glucose level in plasma.¹³ HbA1C can be categorized as good (<6.5%), moderate (6.5-8%) and bad (>8%) diabetic patients with good and moderate levels of HbA1c can be classified as a controlled patients. HbA1c routinely used as a diagnostic tool to measure glucose levels in the blood and can be used to predict the risk of complications in patients with type 2 DM. glucose level control in the blood and decreasing HbA1c level associated with a decreased risk complications.¹⁴

The previous research found that insulin resistance and HbA1c level significantly higher in obese children compared to normoweight children.^{15,16} Study about relation between age, BMI and prevalence of type 2 DM showed that there was a significant correlation between the variables where people with BMI \geq 25kg/m² has 1,496 times greater risk to become a type 2 DM.¹⁷ Another research also showed that the major percentage of diabetic patients 61,5% has BMI \geq 25kg/m².¹⁸ But The earlier study which compared HbA1c levels in obese, overweight and normoweight adults found that there was no significant difference between each groups.¹⁹ Another study also showed no significant correlation between BMI and HbA1c and no significant difference in HbA1c level in women with central obesity than those who do not.^{20,21}

Based on the difference found on the previous research studies, this study aimed to analyze the relation between BMI and HbA1c in diabetic patients.

1. Method

This study involved 80 subjects, Sample population is all of diabetic patients in binjai scattered in four PHC with 20 patients in each PHC which are HAH Hasan, Jati Makmur, Tanah Tinggi and Kebon Lada in accordance with the inclusion and exclusion criteria. All subjects gave informed consent.

Research subjects criteria

A. inclusion criteria

1. diagnosed as type 2 diabetic patients
2. aged 40 – 65 years old
3. has the ability to do the activity by themselves
4. has the ability to read
5. live with their family

B. Exclusion criteria

1. type 2 diabetic patients with physical, mental or cognitive limitation that can be a disturbance for this research (blind, deaf, mental disability)

2. type 2 diabetic patients with any complications that can be a disturbance for this research (chronic renal failure, heart failure, impaired vision and others)
3. type 2 diabetic patients that has no will to include in this research.

Method of data collection using primary data, weight and height as the basis to calculate BMI. We also measured abdominal circumference. HbA1c levels collect by using secondary data results of laboratory tests of blood with immunoassay method. Bivariate data analyzed by using correlation test.

Results and Discussions

Table 1. Baseline characteristic of the 80 samples

	HAH HASAN		JATI MAKMUR		TANAH TINGGI		KEBON LADA	
	N	%	N	%	N	%	N	%
IMT								
-Obesitas	6	30	9	45	4	20	3	15
-Kelebihan berat badan (Overweight)	12	10	3	15	5	25	6	30
-Normal (normoweight)	11	55	7	35	11	55	11	55
-BB dibawah normal (underweight)	1	5	1	5	0	0	0	0
TOTAL	20	100	20	100	20	100	20	100
Hba1c								
-<6.5 (baik)	1	5	1	5	1	5	1	5
-6.5-8 (sedang)	0	0	3	15	6	30	6	30
->8 (buruk)	19	95	16	80	13	65	13	65
TOTAL	20	100	20	100	20	100	20	100

Data from table above shows that most of the diabetic patients in HAH Hasan PHC categorized as normoweight which is 11 people (55%) and the less is underweight which is 1 people (5%). In Jati Makmur PHC most of diabetic patients categorized as obese which is 9 people (45%) and the less is underweight which is 1 people (5%). In Tanah Tinggi PHC most of diabetic patients categorized as normoweight which is 11 people (55%) and the less is obese which is 4 people (20%). In Kebon Lada PHC most of diabetic patients categorized as normoweight which is 11 people (55%) and the less is obese which is 3 people (15%) and non of the samples categorized as underweight.

Most of the diabetic patients have HbA1c levels in bad categorized which is 19 people (95%) and the less is categorized as good which is 1 people (5%) and non of the samples have HbA1c levels categorized as moderate in HAH Hasan PHC. Most of the diabetic patients in Jati Makmur PHC have HbA1c levels categorized as bad which is 16 people (80%) and the less is categorized as good which is 1 people (5%). Most of the diabetic patients in Tanah Tinggi and Kebon Lada is categorized as bad which is 13% (65%) and the less is good which is 1 people (5%).

Table 2. Hba1C Levels of the 80 samples

		Hba1c kelompok		Total
		tdk terkontrol	terkontrol	
bmi kategori	kegemukan	6	11	17
	kurus	3	0	3
	normal	35	3	38
	obesitas	20	2	22
Total		64	16	80

Data was analyzed by using correlation test using SPSS and found that there was no significant relation between BMI and Hba1c of type 2 DM patients in PHC in Binjai city ($p=0,150$; $CI=95\%$). This result is supported by previous research which compared Hba1c levels in obese, overweight and normoweight adults found that there was no significant difference between each groups.¹⁶ There was a research study that also showed no significant correlation between BMI and Hba1c and the study stated that obese which is defined by BMI is not sensitive enough in depicting metabolic disorders. The research conducted in 2015 also found that there was no significant difference in Hba1c level in women with central obesity than those who do not.^{20,21}

High Hba1c levels that found in the most subjects with no elevating in weight or BMI it might happen because the subject was an uncontrolled diabetic patients as we already knew that the uncontrolled diabetic patients will experience decreasing body weight with unknown causes.²²

StasticalAnalysisis

		Bmi hasil	kadar HbA1C pada waktu penelitian
Bmi hasil	Pearson Correlation	1	-,163
	Sig. (2-tailed)		,150
	N	80	79
kadar HbA1C pada waktu penelitian	Pearson Correlation	-,163	1
	Sig. (2-tailed)	,150	
	N	79	79

The data were stastical analyzed using the statistical analysis with correlation test, found that there was no significant relation between BMI and Hba1c of type 2 DM patients in PHC in Binjai city ($p=0,150$; $CI=95\%$).

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References

1. PERKENI 2011. Konsensus pengelolaan dan pencegahan diabetes melitus tipe 2 di indonesia, PB. PERKENI.
2. Reaven, G. (2004) The metabolic syndrome or the insulin resistance syndrome? Different names, different concepts, and different goals. *Endocrinology Metabolism Clinics of North America*, 33, 283-303.

3. IDF. 2013. Idf diabetes atlas[Online].Brussels, Belgium: International DiabetesFederation.Available: <http://www.idf.org/diabetesatlas>[Accessed march, 23 th 2014].
4. Riskesdas 2013. Riset kesehatan dasar.
5. Whiting, D. R., Guariguata, L., Weil, C., Shaw, J. 2011. Idf diabetes atlas: Global estimates of the prevalence of diabetes for 2011 and 2030. *Diabetes research and clinical practice*, 94, 311-321.
6. Nguyen, N.T., *et al.* (2008) Association of hypertension, diabetes, dyslipidemia, and metabolic syndrome with obesity: Findings from the National Health and Nutrition Examination Survey, 1999 to 2004. *Journal of the Ame- rican College of Surgeons*, 207, 928-934.
7. Reaven, G.M. (1995) Pathophysiology of insulin resis-tance in human disease. *Physiological Reviews*, 75, 473- 486.
8. Ali, A.T., *et al.* (2011) Insulin resistance in the control of body fat distribution: A new hypothesis. *Hormone me-tabolism Research*, 43, 77-80.
9. World Health Organization Controlling the global obesity epidemic.2008.
10. World Health Organization Global strategy on diet, physical activity and health. 2008.
11. Chris Burslem, October. The Changing Face of Malnutrition. *IFPRI Forum*,International Food Policy Research Institute: Washington, D.C. 2004.
12. Katz DL, O'Connell M, Yeh MC, Nawaz H, Njike V, Anderson LM, Cory Sand Dietz W. Public health strategies for preventing and controlling overweight and obesity in school and worksite settings: a report on recommendations of the Task Force on Community Preventive Services. *MMWR Recomm Rep.* 2005; 54:1-12.
13. Saudek, C., Kalyani, R. R., Derr, R. 2005. Assessment of glycemia in diabetes mellitus:Hemoglobin a1c. www.japi.org
14. Kunkel HG, Wallenius G. New hemoglobin in normal adult blood. *Science*. 1955;122(3163):288.
15. Shalitin S, Abrahami M, Lilos P, Phillip M (2005). Insulin resistance and impaired glucose tolerance in obese children and adolescents referred to a tertiary-care center in Israel. *Int J Obes (Lond)*, 29(6):571-8.
16. Fujii C, Sakakibara H (2012). Association between insulin resistance, cardiovascular risk factors and overweight in Japanese school children.*Obesity Research &Clinical Practice*, 6:e1-e90.
17. Weiss R, Dziura J, Burgert TS, Tomborlane WV, Taksali SE, Yeckel CW,et al. (2004). Obesity and the metabolic syndrome in children and adolescents. *N Engl J Med*, 350(23):2362-74..
18. Distribution of body mass index of adults and children in the US population. *International Journal of Obesity and Related Metabolic Disorders*, 24, 807-818.
19. Grundy S, 2004. Obesity, Metabolic Syndrome, and Cardiovascular Disease. *The Journal ofClinical Endocrinology & Metabolism* June 1, 2004 vol. 89 no. 6 2595-2600(Online), (<http://jcem.endojournals.org>, diakses 24 Agustus 2009).
20. Bays, H.E, R. H. Chapman, S. Grandy. 2007. *The relationship of body mass index todietabetes mellitus, hypertension and dyslipidaemia: comparison of data from twonational surveys*. *International Journal of clinical Praticce*.
21. Sandeep, S.K., Gokulakrishnan, K., Velmurugan, K., Deepa, M., and Mohan, V., 2010. Viseral & Subcutaneous Andominal Fat in Relation to Insulin Resistance & Metabolic Syndrome in Non-Diabetic South Indians. *Indian J Med Res*, 131, 629-635.
22. Sudoyo, A.W., 2009. *Buku Ajar Ilmu Penyakit Dalam*, Volume III Edition IV. Jakarta: FKUI, 1919-1925.
