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RFM IS A BETTER METABOLIC PREDICTOR THAN BMI IN A COHORT OF PATIENTS WITH DIABETES MELLITUS

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Abstract

As well-known there is a strong connection between obesity and Diabetes Mellitus. Numerous studies are conducted in this field, most of them based on obesity, taking the BMI as a reference value. The aim of our study is to highlight the importance of RFM, especially given that in its formula is incorporated the waist circumference value, as an assessing obesity risk factor for Diabetes Mellitus. We enrolled 137 diabetic patients, hospitalizes in the Endocrine department of "Mother Teresa" Hospital, of Tirana. The variables taken into consideration were age, gender, HbA1c, stature, weight, waist circumference, years with diabetes and from our data we obtained BMI and RFM. We used an IBM SPSS Statistics 23.0 program to analyze the data. Was observed a significative connection between BMI and RFM. It was observed a strong and positive connection between RFM and years with diabetes and HbA1c, that shows that the more the years from diagnosis with diabetes go, the more the chance of obesity increases and the more obese the patient is, the higher get the HbA1c. We do believe that RFM is a better metabolic predictor than BMI in Diabetic patients. Our study was conducted in a small group of patients. We invite researchers to improve the data in this field of study and investigate furtherly.

Keywords: Diabetes Mellitus, Obesity, RFM, BMI, Metabolism, Dyslipidemia.

DM 1	***Diabetes Mellitus type 1***
DM2	***Diabetes Mellitus type 2***
LDL	***Low Density Lipoprotein***
VLDL	***Very Low-Density Lipoprotein***
Tg	***Triglyceride***
HDL	***High Density Lipoprotein***
RFM	***Relative Fat Mass***
BMI	***Body Mass Index***
FFA	***Free Fatty Acids***

I. INTRODUCTION

DM 2 is a multifactorial syndrome, which is thought to be caused by alterations in lipid metabolism associated with genetic and life style factors. The main problem consists in a damaged function of the beta-cells and in a low or absent sensitivity of insulin in the peripheral tissues (muscle, liver, adipose tissue, pancreas). It is not yet clear the prompt pathogenesis of these disease but certainly both of these pathways, synergistically or separately plays an important role in its genesis and development. A great number of patients affected by Diabetes Mellitus type 2 suffer of obesity as well.(1)Because of the insulin resistance, in obese patients are observed increased levels of insulinemia and with the progression of the disease over time can lead to beta cells depletion, therefore to insulin deficiency.(2) A strong relationship is observed between weight and glycemia.

II. MATERIAL AND METHODS

In our clinic we start to collect some data of some patients recovered during two years. The patients were randomly chosen. The parameters taken into consideration were age, gender, HbA1c, stature, weight, waist circumference, years with diabetes and other parameters. From clinically collected data we have calculated BMI and RFM. To calculate BMI the applicated formula was: weight (kg)/ stature(m) Xstature(m).(3)To calculate RFM the applicated formula for adult females was: 76 – (20 x height(m) / waist circumference(m)) while for adult males: 64 – (20 x height (m) / waist circumference (m)).(4)

In our study were enrolled 137 patients, from them 68 were males and 69 females, from 18 to 73 years old, with a mean age of 50.8 years old. We used an IBM SPSS Statistics 23.0 program to explore the data.

III. RESULT ANALYSIS

BMI and RFM both resulted as variables with an abnormal distribution, so we used Spearman's rho correlation, in which came on a significative connection between both variables, as presented in Table 1, with a $p< 0,05$. HbA1c is a variable with a normal distribution, while years with diabetes is a variable with an abnormal distribution. We correlate HbA1c and years with diabetes with BMI and RFM. For all the correlations we used Spearman's rho correlation. As a result of our study we noticed a significative connection between RFM and HbA1c and years with diabetes (duration of the pathology), this association was not observed with BMI as well. It was observed a strong

and positive connection between RFM and years with diabetes, as presented in Table 2, with a $p < 0,05$. This shows that the more the years from diagnosis with diabetes go, the more the chance of obesity increases. It was observed a strong and positive connection between RFM and HbA1c, as presented in Table 3, with a $p< 0,05$. This means that the more obese the patient is, the higher get the HbA1.

Correlations			
			BMI
Spearman's rho	**RFM**	Correlation Coefficient	.641*
		Sig. (2-tailed)	.000
		N	97
***Correlation is significant at the 0.01 level (2-tailed).**			

Correlations			
			Years with DM
Spearman's rho	RFM	Correlation Coefficient	.320*
		Sig. (2-tailed)	.004
		N	80
***Correlation is significant at the 0.01 level (2-tailed).**			

Correlations			
			RFM
Spearman's rho	HbA1c	Correlation Coefficient	.333*
		Sig. (2-tailed)	.036
		N	40
***Correlation is significant at the 0.05 level (2-tailed).**			

IV. DISCUSSIONS

Diabetes very often is associated with dyslipidemia. The most distinctive sign of dyslipidemia is hyper-Tg, partly due to the increased FFA fluxes into the liver, because of the adipocyte lypolysis.(5)This makes possible Tg accumulation into the liver, which are responsible for the increased synthesis of VLDL. FFA are increased in obese patients as a combination of an increased synthesis and clearance reduction from the adipose tissue. Increased levels of FFA and obesity-induced-inflammation play a fundamental role in the development of insulin-resistance. This is called lipotoxicity. LDL and VLDL are known as apoptosis inducers, while HDL are considered protective.(6)

Insulin facilitate the entrance of glucose in the adipocytes, where it is transformed in α-glycerol-phosphate, which gives rise to the esterification of FFA in Tg. An endocellular lipase hydrolyze the deposits of Tg in FFA and returns them back into the circulation. These enzymatic process is inhibited by insulin.(7)An overproduction of VLDL from the liver appears to be the primary and the crucial defect of the insulin resistance state accompanying by obesity and compensatory hyperinsulinemia.(8)

As known obesity, especially central obesity, is one of the main cause of the metabolic syndrome (insulin resistance, DM 2, hypertension, dyslipidemia, etc.). In obese patients is observed a decline in beta cell function and in insulin sensitivity. With the increased insulinemic demand of the tissues and impaired beta cells, abnormal glucose tolerance and Diabetes Mellitus will develop. Further increases of the glycemic index will worsen the created vicious circle, a process called the glucotoxic effect on the pancreatic β-cells. Elevated FFA levels are related to the damaged insulin secretion and biosynthesis and it might contribute to a persistent loss of function of β-cells. As well as the insulin resistance and the damaged mechanism of β-cells lead to increase of the lipotoxic FFA levels. In this way FFA has a double contribute that links β-cell dysfunction and insulin resistance in both diabetic patients and at-risk individuals, a process known as glucolipotoxic effect (9). Insulin resistance and impairment of β-cell function leads to the development of diabetes. The adipose tissue is considered an important source of energy acting as a caloric reservoir. In obesity induced from an increased caloric intake, there is an enlargement of the adipocytes. Hyperalimentation increase Tg deposits, therefore is observed hypertrophia and hyperplasia of adipocytes(10). The risk of developing DM1 in an earlier age is increased by obesity. Also other comorbidities, like metabolic syndrome have an increased risk in obese patients with DM1.(11)As a conclusion we can say that gaining weight is associated with the development of diabetes(12). These alterations can be reversible with the reduction of the weight. Some studies in diabetic patients have confirmed that waist circumference is more strongly associated with metabolic function than BMI(13).

V. CONCLUSIONS

BMI is a formula used to identify obese patients, while there are data showing that RFM is being considered as a better predictor of the body fat mass. The adipose distribution has

the greatest impact and holds the main role in the side effects caused. As well-known central obesity is associated with fatty liver, damaged insulin secretion and sensibility and other metabolic disorders.(14) Based on our study we do believe that RFM is a better predictor of the diabetes progression than BMI. We invite researchers to improve our study with other more detailed studies about obesity and diabetes. Managing and preventing diabetes in obese individuals must be studied and investigated furtherly.

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