

To Evaluate the Relationship between Triglyceride-Glucose Index (TyG) with Left Ventricular Dysfunction

Shreeraksha H¹, MM Basavaraju²¹Post Graduate, Department of General Medicine, Mysore Medical College and Research Institute Mysuru²Professor Department of General Medicine, Mysore Medical College and Research Institute Mysuru

Received: 18-01-2023 / Revised: 21-02-2024 / Accepted: 26-03-2024

Corresponding author: Dr. MM Basavaraju

Conflict of interest: Nil

Abstract:

Background: Insulin Resistance can induce an imbalance in glucose metabolism that generates chronic hyperglycemia, which in turn triggers oxidative stress and causes an inflammatory response leading to cellular damage. Triglyceride Glucose index (TyG) index is a novel comprehensive marker composed of fasting blood glucose and fasting triglyceride levels with better predictive value, cost effective and easier to calculate in routine clinical practice. Therefore, the present study aims to determine the relationship between the TyG index and echocardiographic parameters which can be used to evaluate cardiac function and possibly predict the future cardiovascular risk.

Method: Seventy patients in the age group 18-75 years were included in the study. TyG index was calculated by using formula $\text{Log} (\text{Fasting triglyceride (mg/dl)} \times \text{fasting glucose (mg/dl)})/2$. Echocardiographic parameters including LV parameters (IVSTd, LVIDd, LVPWd), ejection fraction, LA Size were measured. LVM was calculated using ASE (American Society of Echocardiography) approved cube formula as follows: $\text{LVM} = 1.04 \times ([\text{IVSTd} + \text{LVIDd} + \text{LVPWTd}]^3 - \text{LVIDd}^3) \times 0.8 + 0.6$.

Result: Study showed higher TyG index was associated with elevated LV mass. Also Diabetics were found to have higher mean TyG and thus LV mass than non-diabetics.

Conclusion: TyG index is a simple indicator of insulin resistance which reflects cardiac remodelling and dysfunction and in near future could be a potential therapeutic strategy to reduce cardiovascular mortality.

Keywords: Insulin Resistance, Triglyceride to glucose index, cardiovascular risk.

This is an Open Access article that uses a funding model which does not charge readers or their institutions for access and distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>) and the Budapest Open Access Initiative (<http://www.budapestopenaccessinitiative.org/read>), which permit unrestricted use, distribution, and reproduction in any medium, provided original work is properly credited.

Introduction

Insulin is a key hormone that functions as a regulator of cellular metabolism in human body. Insulin resistance (IR) is defined as decrease in tissue response to insulin stimulation thus characterized by defects in glucose and lipid metabolism.

IR can induce an imbalance in glucose metabolism that generates chronic hyperglycemia, which in turn triggers oxidative stress and causes an inflammatory response leading to cellular damage [1]. IR also alters lipid metabolism which leads to dyslipidemia with high levels of plasma triglycerides, low-density lipoprotein and decreased level of high-density lipoproteins.

This, along with endothelial dysfunction contributes to cardiovascular Disease (CVD) via two independent pathways atheroma plaque formation and ventricular hypertrophy and diastolic abnormality thus accounts for one-third of all death

globally and has become a major public health burden [2]. Homeostasis Model assessment 2-Insulin Resistance (HOMA2-IR) is mathematical model that estimates insulin resistance by measuring the levels of insulin and glucose in blood which is cumbersome and impractical in peripheral centers.

Triglyceride Glucose index (TyG) index is a novel comprehensive marker composed of fasting blood glucose and fasting triglyceride levels with better predictive value, cost effective and easier to calculate in routine clinical practice. Thus can be used as a surrogate marker of Insulin resistance. It represents the combined effect of "glucotoxicity" and "lipid toxicity", which prominently contribute to the reduced endocardial collateral flow density and the impaired coronary microcirculation.

Through the generation of reactive oxygen species, hyperglycaemia impairs the structure and function

of heart, leading to myocardial fibrosis and a decrease in compliance. Hypertriglyceridemia leads to increased free fatty acid accumulation in cardiac tissue, along with lipotoxicity, impair insulin signalling resulting in reduced cardiac efficiency and function [3].

High levels of insulin interact via several signaling pathways and genes involved in myocardial growth increase the left ventricular mass through interactions between insulin, its receptor and the receptor of insulin-like growth factor 1 (IGF-1).

IR also decreases IGF-1 levels, which is negatively related to levels of growth hormone. Growth hormone further contributes to an increase in Left ventricular Mass (LVM) by cellular proliferation and fluid retention on the heart. IR is also correlated to LVH through its inflammation-inducing property [4].

Insulin resistance and derangement of glucose metabolism leads to activation of renin angiotensin mechanism, increasing the production and activity of angiotensin 2 causing extracellular matrix deposition and cell proliferation [5].

IR and excess fatty acid lead to deposition of intramyocardial lipids leading to subsequent myocardial fibrosis and systolic dysfunction thus affecting the ejection fraction. Enlargement of the left atrium (LA) can occur as a consequence of left ventricular (LV) diastolic dysfunction [6].

Therefore, the present study aims to determine the relationship between the TyG index and echocardiographic parameters which can be used to evaluate cardiac function and possibly predict the future cardiovascular risk.

Aims and Objectives

To Study the relationship between Triglyceride to glucose index with Left ventricular dysfunction in Diabetics and Non diabetics

Materials and Method

Seventy patients in the age group 18-75 years were included in the study.

Patients with pre-existing cardiac illness, chronic kidney disease or hypertensive heart disease were excluded. Following patients BMI, Waist circumference, blood sugar values, glycated hemoglobin, lipid profile were collected.

TyG index was calculated by using formula $\text{Log}(\text{Fasting triglyceride (mg/dl)} \times \text{fasting glucose (mg/dl)})/2$. Echocardiographic parameters including LV parameters (IVSTd, LVIDd, LVPWd), ejection fraction, LA Size were measured.

- LVM was calculated using ASE(American Society of Echocardiography) approved cube formula as follows:
- $\text{LVM} = 1.04 \times ((\text{IVSTd} + \text{LVIDd} + \text{LVPWd})^3 - \text{LVIDd}^3) \times 0.8 + 0.6$.

Discussion

TyG index was stratified into 4 quartiles based on TyG index (<8.4, 8.4-8.8, 8.8-9.3, >9.3).

In all age groups majority was noted in >9.3 TyG quartile. In <40, 40-60,>60 age groups 50%, 57.1% and 45.5% were noted respectively in highest TyG quartile with highest being in 40-60 year group.

Study showed higher TyG index was associated with elevated LV mass in between groups (Alpha 0.015). Additionally there was correlation between TyG Index and cardio metabolic risk factors like BMI, Waist circumference, fasting Plasma glucose and Total Cholesterol.

Highest mean LVIDd, LVPWd, IVSTd, LA size were noted in highest quartile as shown below. Diabetics were found to have higher mean TyG and thus LV mass than non-diabetics as depicted in bar chart below.

Table 1:

Sl. No	TyG(<8.4)	TyG(8.4-8.8)	TyG(8.8-9.3)	TyG(>9.3)
LVIDd(Left Ventricular Internal Dimension)	4.3800	4.399	4.45	4.56
LVPWd(Left Ventricular Posterior wall thickness)	0.8600	0.9429	0.9600	1.050
IVSTd(Interventricular septal thickness)	0.8600	0.9467	1.0786	1.1306
EF	62.800	63.667	64.071	63.7778
LA Size	2.9	2.9667	2.8786	3.0872
LV Mass	125.3324	140.7902	154.5705	182.0324

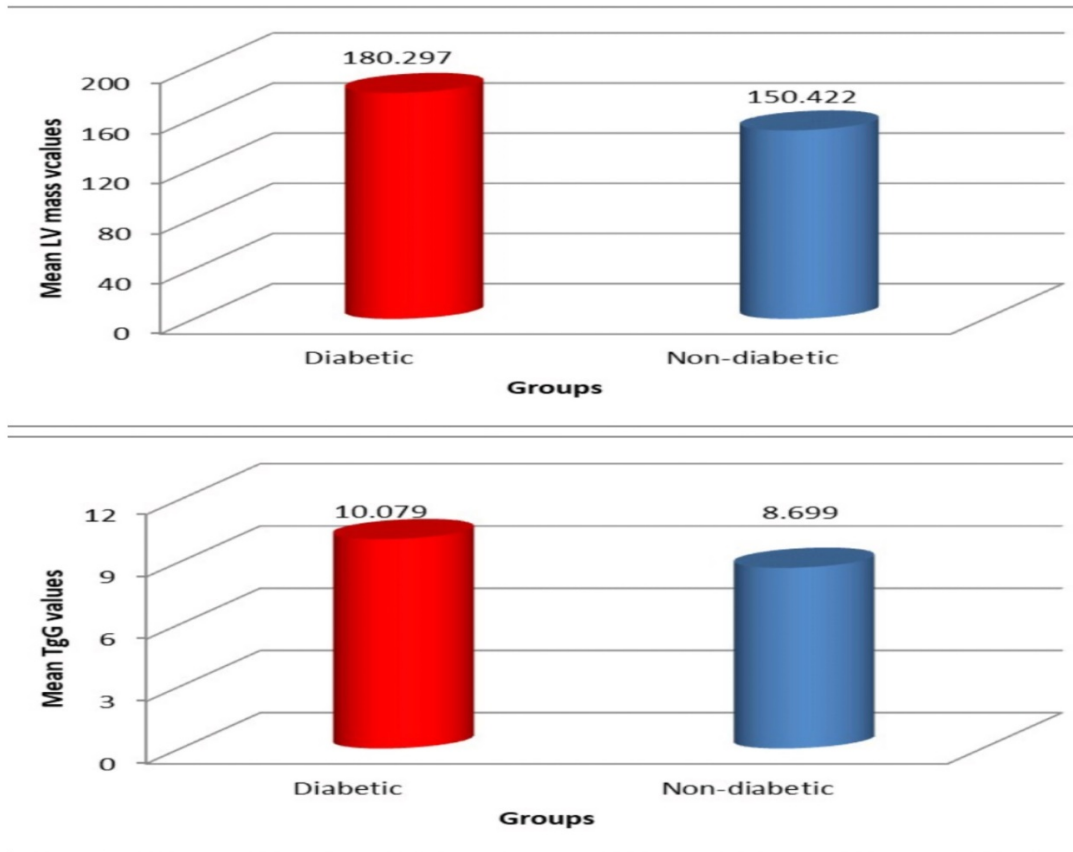


Figure 1:

Conclusion

Thus, TyG index is a simple indicator of insulin resistance which reflects cardiac remodelling and dysfunction.

This study also reflects the diastolic dysfunction being higher in diabetic than non-diabetics which could be correlated to Triglyceride glucose index and could be a potential therapeutic strategy to reduce cardiovascular mortality.

References

1. Laakso M, Kuusisto J. Insulin resistance and hyperglycaemia in cardiovascular disease development. *Nature Reviews Endocrinology*. 2014 May; 10(5):293-302.
2. Jia G, DeMarco VG, Sowers JR. Insulin resistance and hyperinsulinaemia in diabetic cardiomyopathy. *Nature Reviews Endocrinology*. 2016 Mar;12(3):144-53
3. Reaven G, Abbasi F, McLaughlin T. Obesity, insulin resistance, and cardiovascular disease. *Recent progress in hormone research*. 2004 Jan 1; 59:207-24.
4. Ormazabal V, Nair S, Elfeky O, Aguayo C, Salomon C, Zuñiga FA. Association between insulin resistance and the development of cardiovascular disease. *Cardiovascular diabetology*. 2018 Dec;17:1-4
5. Geloneze B, Vasques AC, Stabe CF, Pareja JC, Rosado LE, Queiroz EC, Tambascia MA. HOMA1-IR and HOMA2-IR indexes in identifying insulin resistance and metabolic syndrome: Brazilian Metabolic Syndrome Study (BRAMS). *Arquivos Brasileiros de Endocrinologia & Metabologia*. 2009; 53:281.