

DYSLIPIDEMIA AND METABOLIC SYNDROME

Violeta HOXHA , Dorina YLLI , Gerond HUSI , Thanas FURRERAJ , Zamira YLLI , Pranvera DOCI , Eni CELO , Elizana PETRELA , Marjeta KERMAJ , Agron YLLI

Abstract

Dyslipidemia is a risk factor for metabolic syndrome. The aim of the study: evaluation of dyslipidemia as the risk factor in people with and without metabolic syndrome.

Materials and methods

The study involved 401 healthy individuals aged 25-55 years old. They were chosen at random in Tirana. It included generalities, sex, residence, age, weight, abdominal circumference, systolic blood pressure, diastolic blood pressure, pulse, BMI, cholesterol, Triglyceride, LDL, HDL, glycemia, insulinemia, HOMA -R. Insulin resistance and insulin sensitivity were calculated and compared within people with and without MS. MS prevalence was determined by IDF criteria.

Results

It was noticed that there is a significant link between triglyceride in persons with MS and those without MS ($P < 0,001$)

There was also an important link between HDL in persons with MS and those without MS ($P < 0,001$)

It is noticed an important connection between pulse pressure and total cholesterol ($P = 0,02$) ($r = 0,130$).

There was also between pulse pressure and triglyceride ($P = 0,002$) ($r = 0,158$)

In people with MS was a significant correlation between triglyceride and glycemia ($P = 0,022$) ($r = 0,253$)

There was also an important link between triglyceride and insulinemia ($P = 0,0002$) ($r = 0,403$)

In this people was a significant correlation between triglyceride and HOMA -IR ($P = 0,001$) ($r = 0,489$)

There was also an important link between triglyceride and insulin sensitivity ($P = 0,003$) ($r = 0,326$).

Keywords

MS – metabolic syndrome

IDF – International Diabetes Federation

Background

This dyslipidemia is characterised by a spectrum of qualitative lipid abnormalities reflecting perturbations in the struc-

ture, metabolism, and biological activities of both atherogenic lipoproteins and antiatherogenic HDL-C which includes an elevation of lipoproteins containing apolipoprotein B (apoB), elevated TGs, increased levels of small particles of LDL, and low levels of HDL-C. Insulin resistance leads to an atherogenic dyslipidemia in several ways. First, insulin normally suppresses lipolysis in adipocytes, so an impaired insulin signalling increases lipolysis, resulting in increased FFA levels. In the liver, FFAs serve as a substrate for the synthesis of TGs. FFAs also stabilize the production of apoB, the major lipoprotein of very low density lipoprotein (VLDL) particles, resulting in a more VLDL production. Second, insulin normally degrades apoB through PI3K-dependent pathways, so an insulin resistance directly increases VLDL production. Third, insulin regulates the activity of lipoprotein lipase, the rate-limiting and major mediator of VLDL clearance. Thus, hypertriglyceridemia in insulin resistance is the result of both an increase in VLDL production and a decrease in VLDL clearance. VLDL is metabolized to remnant lipoproteins and small dense LDL, both of which can promote an atheroma formation. The TGs in VLDL are transferred to HDL by the cholesterol ester transport protein (CETP) in exchange for cholesteryl esters, resulting in the TG-enriched HDL and cholesteryl ester-enriched VLDL particles. Further, the TG-enriched HDL is a better substrate for hepatic lipase, so it is cleared rapidly from the circulation, leaving a fewer HDL particles to participate in a reverse cholesterol transport from the vasculature. Thus, in the liver of insulin-resistant patients, FFA flux is high, TGs synthesis and storage are increased, and excess TG is secreted as VLDL [1]. For the most part, it is believed that the dyslipidemia associated with insulin resistance is a direct consequence of increased VLDL secretion by the liver [2]. These anomalies are closely associated with an increased oxidative stress and an endothelial dysfunction, thereby reinforcing the proinflammatory nature of macrovascular atherosclerotic disease.

Worldwide prevalence of MetS ranges from <10% to as much as 84%, depending on the region, urban or rural environment, composition (sex, age, race, and ethnicity) of the population studied, and the definition of the syndrome used [4, 5]. In general, the IDF estimates that one-quarter of the world's adult population has the MetS [9]. Higher socioeconomic status, sedentary lifestyle, and high body mass index (BMI) were significantly associated with MetS. Cameron et al. have concluded that the differences in genetic background, diet, levels of physical activity, smoking, family history of diabetes, and education all influence the prevalence of the MetS and its components [6]. The observed prevalence of the MetS in National Health and Nutrition Examination Survey (NHANES) was 5% among the subjects of normal weight, 22% among the overweight, and 60% among the obese [7]. It further increases with age (10% in individuals aged 20–29, 20% in individuals aged 40–49, and 45% in individuals aged 60–69) [8]. The prevalence of MetS (based on NCEP-ATP III criteria, 2001) varied from 8% to 43% in men and from 7% to 56% in women around the world [6]. Park et al. [7] noticed that there is an increase in the prevalence of MetS from 20 years old through the sixth and seventh decade of life for males and females, respectively.

The aim of the study

Evaluation of dyslipidemia as the risk factor in people with and without metabolic syndrome.

Materials and methods

The study involved 401 healthy individuals aged 25-55 years old.. There were chosen at random in Tirana it included generalities,sex,residence,age,weight,abdominal circumference, systolic blood pressure,diastolic blood pressure,pulse,BMI, cholesterol,Trigiceride,LDL, HDL, glicemia,insulinemia ,HOMA –R. Insulin resistance and insulin sensitivity were calculated and compared within people with and without MS. MS prevalence was determined by IDF criteria.

Statistical analysis

Continuous data were presented in the average value and standart deviation .

The relationships between variables were studied with the Pearson correlation .

Discrete data presented in absolute value and percentage .

Differences between the two groups for continuous quantitative variables was performed through student test .

Data analysis was performed with SPSS statistical package , version 20, (Statistical Package for Social Sciences).

The level of significance was accepted at $P < 0.05$

Results

The study involved 401 healthy individuals 166 man and 235 women (41,4% males and 58,6% females).

Figures/Captions

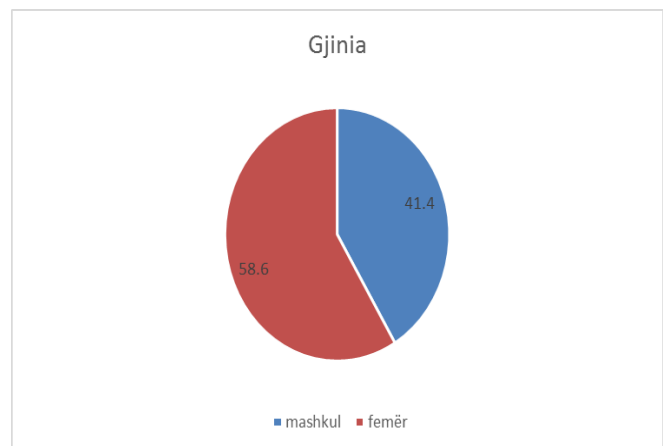


Figure 1. Percentage distribution by gender of individuals in study

It was noticed that there is a significant link between triglyceride in persons with MS and those without MS ($P < 0,001$)

Table 1.

Variables	N	Average	SD	t	Df	Sig. (2-tailed)
TG yes	83	183.80	138.25	8.339	399	<0.001
TG no	318	105.56	48.47			
HD L yes	83	39.37	5.01	-4.487	399	<0.001
HD L no	318	43.37	7.70			
LD yes	83	116.95	31.0	-	39	.521

L	s			0	.64	5	
	no	31	119.64	33.9	3		
Cho	ye	83	190.23	37.1	1.7	39	.086
lest	s			5	21	9	
erol	no	31	182.33	37.2			
		8		6			

There was also an important link between triglyceride and insulinemi ($P=0,0002$), ($r=0,403$)
 In this people was a significant correlation between triglyceride and HOMA -IR ($P=0,001$), ($r=0,489$)
 There was also an important link between triglyceride and insulin sensitivity ($P=0,003$), ($r=0,326$) .

Conclusions

As a result of our study, it was noticed that hypertriglicerydemia that was present in persons with MS , associated with low HDL has a major effect in insulin resistance and endothelial dysfunction. High levels of cholesterol had also an effect in endothelial dysfunction.

References

- [1] G. F. Lewis and G. Steiner, "Acute effects of insulin in the control of VLDL production in humans: implications for the insulin-resistant state," Diabetes Care, vol. 19, no. 4, pp. 390–393, 1996. [View at Google Scholar](#) · [View at Scopus](#)
- [2] H. N. Ginsberg, Y.-L. Zhang, and A. Hernandez-Ono, "Regulation of plasma triglycerides in insulin resistance and diabetes," Archives of Medical Research, vol. 36, no. 3, pp. 232–240, 2005. [View at Publisher](#) · [View at Google Scholar](#) · [View at Scopus](#)
- [3] S. Desroches and B. Lamarche, "The evolving definitions and increasing prevalence of the metabolic syndrome," Applied Physiology, Nutrition and Metabolism, vol. 32, no. 1, pp. 23–32, 2007. [View at Publisher](#) · [View at Google Scholar](#) · [View at Scopus](#)
- [4] G. D. Kolovou, K. K. Anagnostopoulou, K. D. Salpea, and D. P. Mikhailidis, "The prevalence of metabolic syndrome in various populations," The American Journal of the Medical Sciences, vol. 333, no. 6, pp. 362–371, 2007. [View at Publisher](#) · [View at Google Scholar](#) · [View at Scopus](#)
- [5] A. J. Cameron, J. E. Shaw, and P. Z. Zimmet, "The metabolic syndrome: prevalence in worldwide populations," Endocrinology and Metabolism Clinics

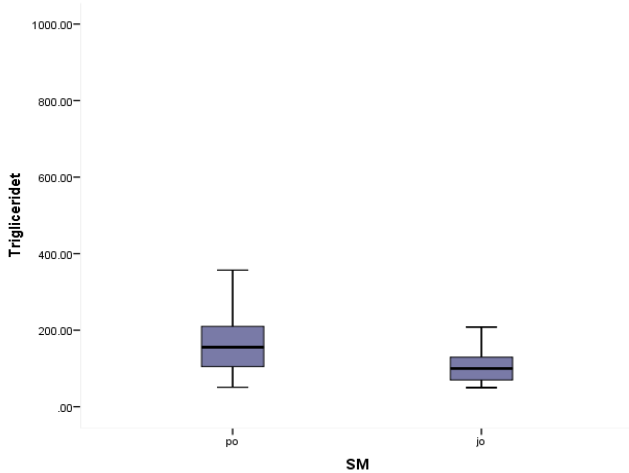


Figure 2. Triglyceride level distribution with and without MS

There was also an important link between HDL in persons with MS and those without MS ($P<0,001$)

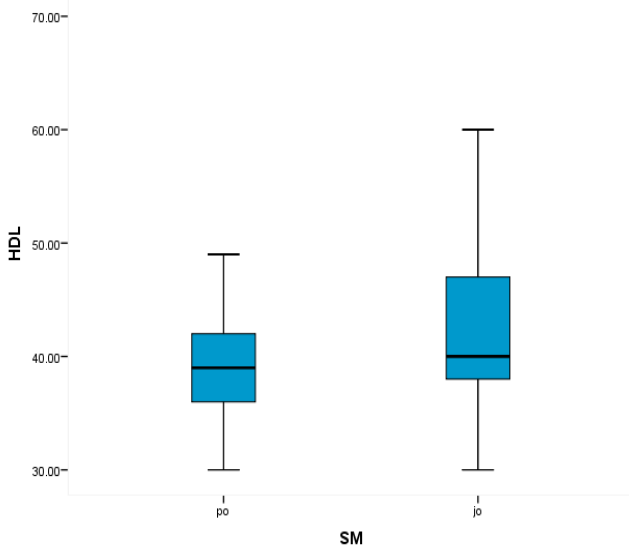


Figure 3. Distribution of values of HDL in people with and without MS

It is noticed an important connection between pulse pressure and total cholesterol ($P=0,02$) ($r=0,130$).
 There was also between pulse pressure and triglyceride ($P=0,002$), ($r=0,158$)
 In people with MS was a significant correlation between triglyceride and glicemi ($P=0,022$), ($r=0,253$)

- of North America, vol. 33, no. 2, pp. 351–375, 2004. [View at Publisher](#) · [View at Google Scholar](#)
- [6] Y.-W. Park, S. Zhu, L. Palaniappan, S. Heshka, M. R. Carnethon, and S. B. Heymsfield, “The metabolic syndrome: prevalence and associated risk factor findings in the US population from the Third National Health and Nutrition Examination Survey, 1988–1994,” *Archives of Internal Medicine*, vol. 163, no. 4, pp. 427–436, 2003. [View at Publisher](#) · [View at Google Scholar](#) · [View at Scopus](#)
- [7] E. S. Ford, W. H. Giles, and W. H. Dietz, “Prevalence of the metabolic syndrome among US adults: findings from the Third National Health and Nutrition Examination Survey,” *Journal of the American Medical Association*, vol. 287, no. 3, pp. 356–359, 2002. [View at Google Scholar](#) · [View at Scopus](#)
- [8] International Diabetes Federation: The IDF consensus worldwide definition of the metabolic syndrome, <http://www.idf.org/metabolic-syndrome>

Biography

VIOLETA HOXHA received the B.S. degree in Medicine from the Medical University of Tirana, Albania, in 1994, specialized in Endocrinology from the Medical University of Tirana, Albania in 2001, earned a Master Degree in 2008 from Medical University of Tirana, Albania, and is doctorate in process.

Currently, she is working as an endocrinologist in “Mother Theresa” Hospital, and teaching First aid and Medical emergencies to medical students in Medical University of Tirana, Albania.

She has authored and co-authored 56 other research articles and a European project about the genetics of metabolic syndrome in 2015.

Violeta Hoxha may be reached at hoxha_violeta@yahoo.com