International Invention Journal of Medicine and Medical Sciences (ISSN: 2408-7246) Vol. 2(6) pp. 96-100, June, 2015
Available online http://internationalinventjournals.org/journals/IIJMMS
Copyright ©2015 International Invention Journals

Full Length Research Paper

The Effects of Smoking and/or Drinking on Visceral Adiposity Index

Edibe Saricicek[1], Ayten Oguz[2], Dilek Tuzun[2], Ahmet Celik[3]*, Elif Sahin[3], Metin Kilinc[3]

[1.]Zirve University, Medical Faculty, Department of Medical Biochemistry, Gaziantep, Turkey
[2.]Sutcu Imam University, Medical Faculty, Department of Endocrinology, Kahramanmaras, Turkey
[3.]Sutcu Imam University, Medical Faculty, Department of Medical Biochemistry, Kahramanmaras, Turkey

Abstract

Body mass index (BMI), waist circumference (WC), serum triglyceride (TG) and HDL-cholesterol (HDL-C) concentrations are evaluated together in calculation of visceral adiposity index (VAI), is a newer described index. We aimed to investigate the relationships of smoking and/or alcohol intake and VAI in healthy adult men in this study. For this purpose 96 healthy adult men were included to the study and divided into four groups; 52 both non-smokers and non-drinkers (Group NSNA), 21 smokers (Group S), 9 drinkers (Group A) and 14 both smoker and drinker (Group SA). Serum concentrations TG and HDL-C, body weight and waist circumference were measured, then BMI and VAI were calculated. VAI, TG and HDL-C were significantly different between the groups (p values were 0.002; 0.008 and 0.044 respectively). VAI, TG and HDL-C were significantly different between Group NSNA and SA (p values were 0.001; 0.004 and 0.035 respectively). There was no significant correlation between cigarettes smoked per years and VAI in smokers. The rise of VAI is associated with both alone alcohol intake and smoking and alcohol intake together. VAI may be lower, in case of absence of both smoking and drinking, non-elevated VAI may be seen in smokers due to lower TG.

Keywords: Visceral Adiposity Index, Smoking, Drinking.

INTRODUCTION

Several parameters have been used to appraise the obesity such as body mass index (BMI), body adiposity index, waist circumference (WC), waist circumference to hip circumference (WHR), waist circumference to height ratio (WHtR) and visceral adiposity index (VAI) (Athyros et al., 2010; Karakas et al., 2012; Lee et al., 2008; Amato et al., 2010). VAI, is a newer described index. BMI, WC, triglyceride (TG) and HDL-cholesterol (HDL-C) were evaluated together in calculation of VAI. It has been proposed as a surrogate marker of adipose tissue dysfunction. It is independently correlated with cardiometabolic risk and thought to be capable of indicating both fat distribution and function. VAI is an indicator of visceral fat dysfunction independently associated to coronary heart disease, myocardial infarction, transient ischemic attack, ischemic stroke and Diabetes Mellitus (Athyros et al., 2010; Karakas et al., 2012; Lee et al., 2008; Amato et al., 2010; Du et al., 2013).

Cigarette smoking is the greatest preventable cause of health disability and premature mortality around the world (Doll et al., 2004; Hozawa et al., 2004; Kamsa-Ard et al., 2013). Alcohol intake is one of the top five risk factors for global burden of disease, particularly in developed nations, and poses increased risk for all-cause mortality (Di Castelnuovo et al., 2006).

The effects of smoking and alcohol intake on some obesity indices and biochemical parameters have been demonstrated in previous studies (Hata and Nakajima, 2000; Xu et al., 2007; Brandhagen et al., 2012; Slagter et al., 2013). The present study was designed to determine the effects of cigarette smoking and/or alcohol intake on the VAI in healthy adult men.

*Corresponding Author Email: drahmetcelik@gmail.com; Tel:+903442803360; Fax:+903442801037

MATERIALS AND METHOD

This study was designed in Departments of Medical Biochemistry and Endocrinology in Kahramanmaras Sutcu Imam University Hospital. All subjects gave informed consent and the study protocol was approved by the local ethics committee. Patients with acute infection, neoplasia, previous stroke and MI history, DM, hypertension, thyroid disorders, taking drugs such as vitamins, anti-inflammatory agents or antibiotics were excluded from the study. All subjects were examined physically. Age, height, weight, WC, alcohol intake and smoking status were recorded.

Participants were grouped in three different forms. Firstly, neither smoker nor drinkers were grouped as Group NSNA (n=52); only smokers as Group S (n=21); only drinkers as Group A (n=9) and both smoker and drinkers as Group SA (n=14). Secondly, they were grouped as MetS (Metabolic Syndrome) and non-MetS, with regard to a cut off level of VAI (14). Thirdly, they were grouped according to BMI as normal's (BMI <25 kg/m^2); overweights (25≤ BMI≥29.99 kg/m^2) and obeses (BMI ≥30 kg/m^2).

Height, body weight (BW) and WC were measured and BMI and VAI were calculated (Amato et al., 2010) (VAI was calculated after biochemical analysis).

BMI= BW/height^2

VAI=[WC/(39.68+1.88xBMI)]x[TG(mmol/L)/1.03]x[1.31/HDL-C(mmol/L)] (for males)

Participants were asked to refrain from smoking for 12 h and abstain from alcohol for 3 days prior to the blood sampling. After an overnight fasting, blood was drawn from ante-cubital vein. Serum was separated by centrifugation for 1700 g, 10 min at +4 degrees within 10 min of sampling and the samples were stored at -70 degrees until the analysis. Serum TG and HDL-C concentrations were determined with commercial kits and auto-analyzer (Advia 1800 Chemistry System, Siemens, NY 10591-5097 USA).

Data were analyzed in SPSS program 15.0 (SPSS Inc, Chicago, IL, USA). For comparison Kruskal-Wallis, Dunnett tests, Pearson's and Spearman's correlation tests were used as appropriate. Statistical significance was assumed when the p-value was less than 0.05. Results were expressed as the mean±SD and percent.

RESULTS

The mean±SD age of participants was 23.8±3.0 (ranges were 18-28). 19 persons (19.85 %) were MetS, 77 (80.2 %) were non-MetS (according to the cut off level of VAI described by Amato et al. 2011), 67 (69.8 %) were normal, 24 (25 %) were overweight and 5 (5.2 %) were obese. All smokers were light smokers (≤20 cigarettes per day). Biochemical and anthropometric data of groups according to smoking and alcohol intake were summarized in table 1.

The rate of MetS was significantly higher in Group A and Group SA than in other groups. ($p<0.05$). The rate of normals was significantly lower and the rate of obeses was significantly higher in Group SA than in other groups. In addition the rate of overweights was significantly lower in Group NSNA (p values were <0.05) (Table 1).

There were no significantly differnece in BMI and WC ($p >0.05$). However, VAI, TG and HDL-C were significantly different between the groups (p values were 0.002; 0.008 and 0.044 respectively). According to post hoc test (Dunnett test), VAI, TG and HDL-C were significantly different between the Group NSNA and SA (p values 0.001; 0.004 and 0.035 respectively). In addition, VAI and TG were significantly different between the Group S and SA (p values 0.004; and 0.005 respectively) (Table 1). There was no significant correlation between cigarettes smoked per years and VAI in Group S and SA.

DISCUSSION

According to the cut off level of VAI (Amato et al., 2011), we found that the rate of MetS was significantly higher in Group A and Group SA than in other groups. Since VAI is a newer index, there are no studies in the literature commenting on direct association of VAI with smoking and/or alcohol intake. However, relations between smoking and parameters using in calculation of VAI investigated in many previous studies and various results were obtained. In a cohort study Slagter et al. have reported that smoking is associated with lower HDL-C, higher TG and WC and an increased prevalence of MetS, independent of sex and BMI class (Hata and Nakajima, 2000; Slagter et al., 2013).

On the other hand, some studies have reported contrary results. It has been reported that smokers had lower WC, body fat percentage, and BMI compared with non-smokers (Chatkin and Chatkin, 2007; Clair et al., 2011; Mieczkowska et al., 2012). Xu et al. 2007 established that cigarette smoking was negatively associated with BW indicated by BMI but not with central obesity indexed by WC in Chinese men.

In our study, significant differences were found in VAI and biochemical parameters (TG and HDL-C) between the groups. However other parameters of VAI (BMI and WC) were not different between the groups. In this study, the difference in VAI is due to TG and HDL-C. Slagter et al. have reported that smoking is associated with lower HDL-C (Slagter et al., 2013).

TG was lower in Group S, Lower TG may be caused by increase of basal metabolic rate due to

Table 1. Demographic and biochemical data of groups according to smoking and alcohol intake

parameters	Group NSNA (n=52)	Group S (n=21)	Group A (n=9)	Group SA (n=14)	p values
Age. year	23.9±3.0	23.4±2.9	22.7±3.3	23.8±3.0	0.637
Light smokers. %	0	100	0	100	
Heavy smokers. %	0	0	0	0	
Smoking. pack-years	0	356±204	0	439±158	
MetS. n (%)	7 (13.5)	2 (9.5)	2 (22.2)	8 (57.1)	0.008
Normals. n (%)	40 (76.9)	15 (71.4)	6 (66.6)	6 (42.9)	0.235
Overweights. n (%)	10 (19.2)	6 (28.6)	3 (33.3)	5 (35.7)	0.034
Obeses. n (%)	2 (3.8)	0 (0)	0 (0)	3 (21.4)	0.007
BMI. kg/m^2	23.7±3.3	23.3±2.5	24.7±3.1	25.8±3.8	0.105
VAI	1.52±0.87	1.52±0.88	2.40±1.97	2.74±1.30	0.002
WC. cm	87.8±10.9	87.1±9.9	89.1±9.5	94.4±12.4	0.160
Triglyceride. mg/dL	101.1±43.2	95.4±40.1	142.1±85.0	151.0±60.8	0.008
HDL-C. mg/dL	41.3±8.1	39.2±9.1	38.4±8.8	34.8±9.4	0.044

Abbreviations: BMI. body mass index; HDL-C. high density lipoprotein cholesterol; MetS. metabolic syndrome; VAI. visceral adiposity index; WC. waist circumference

hypercatecholaminemia and decrease of appetite. Cigarette smoking is accompanied by hypercatecholaminemia (Zevin et al., 2001). The inhalation of nicotine acutely raises the levels of dopamine and serotonin in the brain, reducing the need for energy intake and consequently suppressing appetite (Klein et al., 2004; Hussain et al., 2012). In addition nicotine has a direct effect on adipose tissue metabolism causing a higher oxidation of lipids. Which help explain the fact that TG is lower in smokers than in non-smokers. Body weight tends to be lower among smokers than among non-smokers. Smoking cessation results in an increase in body weight (Klein et al., 2004; Hussain et al., 2012; Filozof et al., 2004; Ferrara et al., 2001; Nikoloutsou et al., 2014). As a result VAI may be found as non-elevated in smokers. Smoking itself is a cause of health disability and premature mortality (Hozawa et al., 2004; Kamsa-Ard et al., 2013). It increases the risk of metabolic syndrome diabetes and these factors increase risk of cardiovascular disease (Chiolero et al., 2008). Non-elevated VAI doesn't mean that there is no risk for health disability and premature mortality in smokers.

We found no significant correlation between smoking pack-years and VAI. Kim et al. found that while BMI and total body fat percentage were not associated with smoking pack-years, abdominal and visceral obesity were associated with it (Kim et al., 2012). Clair et al. 2011 reported that while WC and body fat increased with cigarettes smoked per day. There was no relation between cigarettes smoked per day and BMI. Among smokers, cigarettes smoked per day were positively associated with central fat accumulation. In our study, TG, HDL-C, WC, BMI and VAI were not significantly correlated with smoking pack-years. These results may resource from features of study population for example all smokers were light smoker or other factors such as diet exercise and age.

Findings obtained from previous studies on the effects of alcohol consumption on biochemical and anthropometric parameters are clearer and support our findings about higher VAI in drinkers. Hata and Nakajima 2000 have reported that drinking elevated triglycerides by a mean of 10 mg/dl and also HDL-C by 2.5 mg/dl. Wakabayashi 2013 has reported that the ratio of TG to HDL-C (TG/HDL-C). TG and WC were significantly higher in drinkers than in non-drinkers. Brandhagen et al. 2012 found that alcohol intake was positively related to central and general obesity in men.

VAI is 1 in healthy non-obese subjects with normal serum TG and HDL-C levels and normal fat distribution (Karayannis et al., 2012). In case of lower HDL-C, higher TG and WC, VAI is expected to be found as markedly higher. So, these parameters are evaluated together by VAI. From 10% to 20% of obese people (according to BMI), do not show the metabolic changes common in obese patients. These metabolic changes elevate hepatic enzymes, HDL-C, TG to HDL-C ratio and decrease insulin sensitivity etc. These patients are classified as "metabolic healthy obese" (MHO) persons. On the other hand, some of non-obese patients have these metabolic changes. They were classified as "metabolic unhealthy non-obese" (MUNO) persons (Griera Borrás et al., 2013; Messier et al., 2010). In our study all of these parameters and the rates of MHO and MUNO persons weren't considered. However, we can hypothesized that there is an association between the

rate of MUNO and non-elevated VAI and smoking may play a central role in both conditions. This hypothesis needs to be corrected.

Limitations

Participants were not considered by means of exercise and diet. None of the subjects were heavy smokers and alcohol consumption was not degreed. As all participants were men, sex differences were not taken into consideration about studied subjects.

CONCLUSION

We concluded that even though elevated VAI was associated with alcohol intake alone. there were no association and correlation between the VAI and smoking alone. However. smoking and alcohol intake were together associated with higher VAI and increased rate of MetS and obesity. This result is due to higher TG and lower HDL-C. In case of absence of both smoking and drinking. VAI and MetS rate may be found lower. Smoking alone may suppress the elevation of VAI due to hypercatecholaminemia and non elevated serum triglyceride level. In this condition to management of alone VAI may be insufficient to estimate the cardiometabolic risk.

Conflict of interest

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

ACKNOWLEDGEMENTS

We thank Prof. Dr. Fatma Inanc Tolun for critically reviewing the manuscript and the staff of Sutcu Imam University Medical Faculty Hospital for their help and technical support

REFERENCES

Amato MC, Giordano C, Galia M, Criscimanna A, Vitabile S, Midiri M, Galluzzo A (2010). AlkaMeSy Study Group. Visceral adiposity index. A reliable indicator of visceral fat function associated with cardiometabolic risk. Diabetes Care.33(4):920-2.

Amato MC, Giordano C, Pitrone M, Galluzzo A (2011). Cut-off points of the visceral adiposity index (VAI) identifying a visceral adipose dysfunction associated with cardiometabolic risk in a Caucasian Sicilian population. Lipids Health Dis. Oct 19;10:183. doi: 10.1186/1476-511X-10-183.

Athyros VG, Tziomalos K, Karagiannis A, Anagnostis P, Mikhailidis DP (2010). Should adipokines be considered in the choice of the treatment of obesity-related health problems? Curr Drug Targets;11(1):122-35.

Brandhagen M, Forslund HB, Lissner L, Winkvist A, Lindroos AK, Carlsson LM, Carlsson LM, Sjöström L, Larsson I (2012). Alcohol and macronutrient intake patterns are related to general and central adiposity. Eur J Clin Nutr.66(3):305-13.

Chatkin R, Chatkin JM (2007). Smoking and changes in body weight: can physiopathology and genetics explain this association? J Bras Pneumol. Nov-Dec;33(6):712-9.

Chiolero A, Faeh D, Paccaud F, Cornuz J (2008). Consequences of smoking for body weight. body fat distribution. and insulin resistance. Am J Clin Nutr. Apr;87(4):801-9.

Clair C, Chiolero A, Faeh D, Cornuz J, Marques-Vidal P, Paccaud F, Mooser V, Waeber G, Vollenweider P (2011). Dose-dependent positive association between cigarette smoking. abdominal obesity and body fat: cross-sectional data from a population-based survey. BMC Public Health. Jan 11;11:23. doi: 10.1186/1471-2458-11-23.

Di Castelnuovo A, Costanzo S, Bagnardi V, Donati MB, Iacoviello L, de Gaetano G (2006). Alcohol dosing and total mortality in men and women. Arch Intern Med; 166: 2437–45.

Doll R, Peto R, Boreham J, Sutherland I (2004). Mortality in relation to smoking: 50 years' observations on male British doctors. BMJ.328: 1519-28.

Du T, Sun X, Huo R, Yu X (2013). Visceral adiposity index. hypertriglyceridemic waist and risk of diabetes: the China Health and Nutrition Survey 2009. International Journal of Obesity accepted article preview 19 September 2013; doi: 10.1038/ijo.181.

Ferrara CM, Kumar M, Nicklas B, McCrone S, Goldberg AP (2001). Weight gain and adipose tissue metabolism after smoking cessation in women. Int J Obes Relat Metab Disord.;25(9):1322-6.

Filozof C, Fernandez Pinilla MC, Fernandez-Cruz A (2004). Smoking cessation and weight gain. Obes Rev.;5(2):95-103.

Griera Borrás JL, Contreras Gilbert J (2013). Are there healthy obese? Endocrinol Nutr. 2013 Oct 24. pii: S1575-0922(13)00140-X. doi: 10.1016/j.endonu.03.010.

Hata Y, Nakajima K (2000). Life-style and serum lipids and lipoproteins. J Atheroscler Thromb.7(4):177-97.

Hozawa A, Ohkubo T, Yamaguchi J, Ugajin T, Koizumi Y, Nishino Y, Tsubono Y, Shibuya D, Tsuji I, Fukao A, Hisamichi S (2004). Cigarette smoking and mortality in Japan: the Miyagi Cohort Study. J Epidemiol.14(1): 12-7.

Hussain T, Al-Daghri NM, Al-Attas OS, Draz HM, Abd Al-Rahman SH, Yakout SM (2012). Plasma neuropeptide Y levels relate cigarette smoking and smoking cessation to body weight regulation. Regul Pept. Jun 10;176(1-3):22-7.

Kamsa-Ard S, Promthet S, Lewington S, Burrett JA, Sherliker P, Kamsa-Ard S, Wiangnon S, Parkin DM (2013). Association between smoking and mortality: Khon Kaen cohort study. Thailand. Asian Pac J Cancer Prev.14(4):2643-7.

Karakas P, Bozkir MG (2012). Anthropometric indices in relation to overweight and obesity among Turkish medical students. Arch Med Sci.;8(2):209-213.

Karayannis G, Giamouzis G, Tziolas N, Georgoulias P, Skoularigis J, Mikhailidis DP, Triposkiadis F (2013). Association between epicardial fat thickness and weight homeostasis hormones in patients with noncachectic heart failure. Angiology. 64(3): 173-80.

Kim JH, Shim KW, Yoon YS, Lee SY, Kim SS, Oh SW (2012). Cigarette smoking increases abdominal and visceral obesity but not overall fatness: an observational study. PLoS One. 2012;7(9):e45815. doi: 10.1371/journal.pone.0045815. Epub Sep 24.

Klein LC, Corwin EJ, Ceballos RM (2004). Leptin. hunger. and body weight: Influence of gender. tobacco smoking. and smoking abstinence. Addict Behav.;29(5):921-7.

Lee CMY, Huxley RR, Wildman RP, Woodward M (2008). Indices of abdominal obesity are better discriminators of cardiovascular risk factor than BMI: a meta-analysis. J Clin Epidemiol.;61(7): 646-53.

Messier V, Karelis AD, Robillard ME, Bellefeuille P, Brochu M, Lavoie JM, Rabasa - Lhoret R (2010). Metabolically healthy but obese

individuals: relationship with hepatic enzymes. Metabolism. Jan;59(1):20-4.
Nikoloutsou I, Vasileiou V, Litsiou E, Kolokytha S, Tsoutsa A, Zakynthinos S, Katsaounou P (2014). Smoking cessation changes basic metabolism. body weight. leptin and insulin levels. adipose tissue percentage. index of insulin resistance and index of insulin secretion. Tobacco Induced Diseases. 12(Suppl 1):A24
Slagter SN, Vliet-Ostaptchouk JV, Vonk JM, Boezen HM, Dullaart RP, Kobold AC, Feskens EJ, van Beek AP, van der Klauw MM, Wolffenbuttel BH (2013). Associations between smoking. components of metabolic syndrome and lipoprotein particle size. BMC Med. Sep 3;11:195. doi: 10.1186/1741-7015-11-195.
Wakabayashi I (2013). Associations between heavy alcohol drinking and lipid-related indices in middle-aged men. Alcohol. 2013 Oct 11. doi: 10.1016/j.alcohol.10.001. [Epub ahead of print]
Xu F, Yin XM, Wang Y (2007). The association between amount of cigarettes smoked and overweight. central obesity among Chinese adults in Nanjing. China. Asia Pac J Clin Nutr.16(2):240-7.
Zevin S, Saunders S, Gourlay SG, Jacob P, Benowitz NL (2001). Cardiovascular effects of carbon monoxide and cigarette smoking. J Am Coll Cardiol. Nov 15;38(6):1633-8.

How to cite this article: Saricicek E. Oguz A. Tuzun D. Celik A. Sahin E. Kilinc M (2015). The Effects of Smoking and/or Drinking on Visceral Adiposity Index. Int. Inv. J. Med. Med. Sci. Vol. 2(6): 96-100