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CIGARETTE SMOKING COULD INCREASE PREVALENCE OF CAROTID ARTERIAL PLAQUE IN OBESE MALE HEALTHY COLLEGIATE STUDENTS

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ABSTRACT : Obesity is a known risk factor for several diseases and also negatively affects physical functioning. It is identified that obesity is an independent risk factor for cardiovascular (CV) disease. However, only few studies have investigated the association of different anthropometric measures with carotid plaque. We in this study aimed to investigate, compare and find the correlation between four measures in normal and obese adults: Anthropometric, Blood Analysis, Smoking habit and presence of Carotid Arterial Plaque. We included 44 healthy young collegiate students aged 20-25 years and were divided into two based on their body mass index: normal weight (n=15) and obese (n=29). Anthropometric measurements such as body weight, body mass index (BMI), waist / hip ratio (WHR), body fat percentage (BFP) and visceral fat area (VFA) and from the collected venous blood, total cholesterol, triglycerides, HDL cholesterol, LDL cholesterol and glucose were measured. Presence of carotid arterial plaque [CAP] was identified by ultrasonography. Plaque presence was defined as e"1 plaque in any of the carotid arteries. Data were analyzed using independent t-test. Pearson's correlation coefficient was used to find the correlations between the measured parameters. After adjustment for age and sex between the groups, there was strong evidence of significant difference between groups for anthropometric measures [Weight, BMI, WHR, BFP and VFA] and also for serum HDL, LDL and triglycerides levels ($p < 0.01$). But there was no difference for serum total cholesterol and glucose concentrations ($p > 0.05$). CAP was found in 69% of obese subjects and a strong correlation was found between smoking and presence of carotid plaque. A smoker is 1.84 times subjected to plaque occurrence than non-smoker among obese participants. In this study we found high prevalence of CAP among the obese adults. Occurrence of CAP was significantly increased among the smokers' ones. Thus, evaluation of obese patients with this anthropometric, lipid profile and cigarette smoking could help the individuals to identify the risk of higher residual cardiovascular incidence.

Key words : Obesity, anthropometry, carotid arterial plaque, lipid profile, cigarette smoking.

INTRODUCTION

Obesity is a serious medical and pandemic health challenge, and it is increasing at an alarming rate in most countries, including Saudi Arabia (Al-Baghli, Al-Ghamdi *et al*, 2008; Badran and Laher, 2011; Almughamisi, George *et al*, 2017). People with this chronic disorder experience significantly increased risks of many pathological complications such as cardiovascular disease, type 2 diabetes, hepatic dysfunction, renal dysfunction, asthma, sleep disorders, certain cancers, stroke and all-cause mortality (World Health Organization, 2000; Lavie, Milani *et al*, 2009; Manna and Jain, 2015). It is believed that, obesity may lead to stroke via aggravation of hypertension, dyslipidemia, insulin resistance, and the metabolic syndrome (Deen, Raheem *et al*, 2013).

On the other hand, carotid artery plaque (CAP) is considered as an excellent substitute for determining arterial atherosclerosis (Oh, Han *et al*, 2010; Sturlaugsdottir, Aspelund *et al*, 2016) and is significantly increased in patients with coronary artery disease, acute myocardial infarction, and stroke events (Mantella, Colledanchise *et al*, 2017; Zhou, Wang *et al*, 2017). Indeed, CAP can be utilized as ultrasonographic indicator of early atherosclerosis as well as an independent predictor for cardiovascular risk (Raitakari, Juonala *et al*, 2009). Petty *et al* (1999) showed that CAPs are responsible for at least 15-20% of all ischemic strokes. Other investigators estimated that 28% of ischemic strokes are

atherothrombotic in origin, and that most resulted from carotid stenosis (Serena, Irimia *et al*, 2013). Understanding the risk factors for CAPs may lead to better screening and interventions to decrease excess risk for cardiovascular disorder and may also improve people's inspiration to alter harmful habits. Up to now, the published results regarding the effect of being overweight or obese on CAP have been conflicting (Friedlander and Lande, 1981; Ghouri, Purves *et al*, 2015; West, Juonala *et al*, 2015; Sturlaugsdottir, Aspelund *et al*, 2016) and there is a lack of information regarding this issue among young adult males, particularly in Saudi population.

Therefore, our major aim was to determine the prevalence of CAP among obese young adult males. In addition, we examined the association between cigarette smoking and presence of CAP among these participants.

MATERIALS AND METHODS

Subjects and study design

The study was approved by the Scientific Review Board (SRB) and Institutional Ethical committee of College of Medicine at University of Hail. A total of 44 young adult male students aged between 20 and 25 years were recruited from the medical and applied medical science college of University of Hail, Saudi Arabia. This is a cross sectional study for a complete one-day trial in which the baseline investigations were done and analyzed for the prevalence of carotid plaque among obese individuals. Individuals with chronic diseases [diabetes, coronary artery disease, vascular disease etc] were excluded. Validated questionnaire addressing age, history of chronic diseases, drugs, smoking and personal habits was self-administered. After filling the questionnaire, anthropometric measurements such as body weight (BW), body mass index (BMI), waist / hip ratio (WHR), body fat percentage (BFP) and visceral fat area (VFA) of all the students were measured using In-Body Composition Analyzer 720 (Biospace, Seoul, Korea). Subjects are then grouped into two based on the guidelines of the National Institute of Health 2017: normal weight group [NW] with BMI = 18.5 - 25 kg/m2 (n = 15) and obese group [OB] with BMI ≥ 30 (n = 29).

Blood collection and analysis

Venous blood samples (10 ml) were obtained between 09:00 and 11:00 am after overnight (>12 hr) fasting. Blood samples were drawn from an antecubital vein into vacutainer tubes without anticoagulant in a sitting position. For serum collection, tubes were incubated in an upright position at room temperature for 15-30 min to allow clotting before centrifugation (10 minutes, 3000 rpm). Serum was immediately stored at –70°C until further analysis. Laboratory blood examinations include total cholesterol (normal range 0-5.2 mmol/L), triglycerides (normal range 0.34-2.2 mmol/L), HDL cholesterol (normal range 0.9-1.55 mmol/L), LDL cholesterol (normal range 0-3.88 mmol/L) and glucose (normal range 4.2-6.1 mmol/L) were measured using commercial enzyme-linked immunosorbent assay kits (HUMAN Diagnostics, Wiesbaden, Germany).

Ultrasonography of Carotid arteries

The left and right internal carotid arteries were scanned by certified examiners using a high-resolution ultrasound Doppler system General Electric (GE) Vivid 7 ultrasound scanner, 8-MHz transducer with the participant in the horizontal position. Plaque occurrence was diagnosed during the examination in the common carotid arteries (CCAs), its bifurcation, internal carotid artery (ICA) and external carotid artery (ECA). Plaques were present if a focal widening of the vessel wall relative to adjacent segments was found (as evidenced by protrusion into the lumen or localized roughness with increased echogenicity or an area of focal increased thickness of the intima–media layer). Plaque presence was defined as ≥ 1 plaque in any of the carotid arteries.

Statistical analysis

All variables were reported as mean ± standard deviation (SD). Data were analyzed using independent "t" test. Pearson's correlation coefficient was used to evaluate the correlations between the anthropometric measurements and the measures of laboratory blood examinations and between smoking and plaque occurrence. All statistical analyses were performed with the SPSS statistical package version 23.0 (SPSS Inc., Chicago, IL, USA). An á level of 0.05 was used to denote statistical significance.

RESULTS

Mean values of anthropometric characteristics and blood parameters in the NW and OB groups are presented in Table 1. By experimental design, the groups were similar by age and significantly differed ($p < 0.01$) by weight, BMI, WHR, BFP and VFA. Significant differences were found between groups for serum HDL, LDL and triglycerides levels. No differences were noted among groups for serum total cholesterol and glucose concentrations ($p > 0.05$). Carotid plaque was not detected among NW groups whereas around 66% of OB groups had presence of carotid plaque. As shown in Table 2, a good correlation was found between smoking and presence of carotid plaque. A smoker is 1.84 times subjected to plaque occurrence than non-smoker among obese participants. As presented in Table 3, significant

Table 1 : Mean values of Anthropometric characteristics, demographic data and laboratory blood examinations in the normal-weight and obese groups.

Variable	Normal weight	Obese	P value
N	15	29	
Age (years)	21.8±1.30	22.6±2.57	NS
Weight (kg)	68.13 ± 6.70	112.43±15.26*	NS
BMI (kg/m^2)	21.8 ± 2.25	37.7±5.20*	NS
BFP (%)	19.7 ± 5.24	44.6±4.92*	NS
WHR	0.82 ± 0.0399	0.98±0.0255*	NS
VFA(cm^2)	44.63 ± 1.94	149.83±7.94*	NS
Cholesterol (m mol/L)	3.96 ± 0.50	4.19±0.72	NS
HDL (m mol/L)	1.63 ± 0.33	0.80±0.09*	NS
LDL (m mol/L)	2.08 ± 0.37	2.77±0.62*	NS
Triglycerides (m mol/L)	0.56 ± 0.29	1.36±0.75*	NS
Fasting serum glucose (m mol/L)	5.18 ± 0.099	5.24±0.14	NS

Data are expressed as means ± SEM. BMI: body mass index; WHR: waist/hip ratio; BFP: body fat percentage; VFA: visceral fat area; ns, non-significance. *Significantly different from normal-weight group, $p < 0.01$.

Table 2 : Correlation between smoking and arterial carotid plaques among obese participants.

Group	CPQ+	CPQ-	Total
Smoking	12	2	14
Non-smoking	7	8	15
Total	19	10	29

Odds ratio (of having CPQ+ for smoking Vs non-smoking) = 7.84
Relative risk (of having CPQ+ for smoking Vs Non-smoking) = (12/14) / (7/15) = 1.84.
The risk of a CPQ+ for smoking people is 1.84 that of non-smokers people

correlations were observed between serum HDL, LDL and triglycerides concentrations and all measured anthropometric parameters. No associations were found between serum total cholesterol with blood glucose or tested anthropometric parameters. Mean values of anthropometric characteristics and blood parameters in the subjects with carotid plaque and subjects without carotid plaque (within OB groups) are presented in Table 4. Obese participant with carotid plaque had statistically significantly lower BFP (%) and LDL concentrations and significantly higher HDL concentrations compared to OB subjects without plaque. No differences were noted among groups for serum total cholesterol, triglycerides, glucose concentrations, WHR and VFA ($p > 0.05$).

Table 3 : Correlation between laboratory blood results and Anthropometric.

Variables	BMI	BFP	WHR	VFA
Cholesterol	0.136	0.190	0.211	0.170
HDL	-0.794***	-0.816***	-0.886***	-0.880***
LDL	0.512***	0.493**	0.543***	0.536***
Triglycerides	0.570***	0.578***	0.581***	0.539***
Glucose	-0.264	-0.175	-0.146	-0.171

***P = 0.000, **p = 0.001
BMI – Body Mass Index, BFP – Body Fat Percentage, WHR – Waist Hip Ratio, VFA – Visceral Fat Area.

Table 4 : Mean values of Anthropometric characteristics, demographic data and laboratory blood examinations in the CPQ+ and CPQ- within obese groups.

Group	CPQ+	CPQ-	Total
N	19	10	
BMI (kg/m^2)	36.67±4.83	39.18±5.01	NS
BFP (%)	43.36±4.367	47.33±5.225	0.042*
WHR	0.9927±0.027	0.9925±0.021	NS
VFA(cm^2)	148.23±7.63	153.40±7.86	NS
Cholesterol (m mol/L)	4.16±0.44	4.04±1.03	NS
HDL (m mol/L)	0.87±0.09	0.79±0.09	0.042*
LDL (m mol/L)	2.40±0.22	2.93±0.88	0.049*
TGL (m mol/L)	0.91±0.21	1.23 ± 0.66	NS
Fasting serum glucose (m mol/L)	5.16±0.54	4.85 ± 0.43	NS

DISCUSSION

The present study demonstrated that carotid arterial plaques (CAP) were not detected among NW subjects; however, a significant high prevalence of CAP was observed among OB participants. The occurrence of CAP provides strong evidence that premature vascular structural changes are present in obese young adult males. Our findings are discordant with results of Notwithstanding, Friedlander and Altman (1981), who reported a lack of association between obesity and presence of atheromatic plaques in the carotid arteries. Also, Ghouri and Yang established that BMI had no connection with CAP (Yang, Sun *et al*, 2014; Ghouri, Purves *et al*, 2015). Contrary to our results Sturlaugsdottir demonstrated that obesity reduces the risk of developing CAP (Sturlaugsdottir, Aspelund *et al*, 2016). However, the results of this study are in line with West who reported that increasing BMI appeared to be a risk factor for carotid plaque (West, Juonala *et al*, 2015; Lou, Li *et al*, 2017). The contradictory reports about the involvement of obesity to the presence of CAPs may be attributed to the fact that most studies were conducted in old or diseased participants. Ethnic and other

environmental factors may also play a key role leading to this obvious disagreement. Importantly, we found that more than 80% of plaques were observed in left common carotid artery. Due to anatomical differences, peak systolic velocity (PSV) and flow volume (FV) in left vertebral artery were found to be significantly higher than right side in young adults (Nemati, Bavil *et al*, 2009).

Further our results demonstrated a remarkable association between cigarette smoking and CAP within obese participants. It was found that CAPs were present in more than 85% of obese cigarette smokers comparing to 47% in obese non-smokers participants. These finding are in agreement with many other results which recommended smoking as a risk factor for CAPs (Guo, Jiang *et al*, 2003; Yang, Iyer *et al*, 2015; Taramasso, Mirabella *et al*, 2018). However, no study yet investigated the relationship between smoking and presence of CAPs among obese young adults. A significant positive connections between BMI and blood glucose, serum TGs, cholesterol and LDL, together with a negative correlation between BMI and serum HDL demonstrated in our study are in line with previous studies (Al-Amodi, Abdelbasit *et al*, 2017).

These results demonstrated that obese adolescents have multiple cardiovascular risk factors including high BMI, higher serum cholesterol level, higher serum triglycerides level, higher serum level of LDL, low level of serum HDL and high VSA. These findings coincides with that of Szczygielska *et al* (2003), who found that obesity and overweight are accompanied by unfavorable blood lipids patterns and in a considerable proportion of overweight or obese patients other risk factors for coronary heart disease, such as hypertension, smoking, diabetes or family history of cardiovascular diseases (Szczygielska *et al*, 2003). Also EI Ugwuja *et al* (2013) found that those with higher BMI and advanced in age, exhibited unfavorable plasma lipids which may predispose them to CVD. Our results as shown in fig. I found a good correlation between smoking and presence of carotid plaque. A smoker is 1.84 times subjected to plaque occurrence than non-smoker among obese participants. These results coincides with those of Dixon Yang *et al* (2015), who observed a non-linear, V shaped like relationship between current cigarette smoking and plaque echodensity. Also, current smokers were more likely to have either soft or calcified plaques and former smokers were at greater risk of only echodense plaques when compared against never smokers and recommended further researches to determine if plaque morphology mediates an association between smoking and clinical vascular events. Asdrúbal Nóbrega Montenegro-Neto *et al* (2011) suggested that additional cardiovascular risk could be demonstrated by the high prevalence of being overweight and central obesity presented by the population and the presence of subclinical inflammation amongst hypertensive ones.

Our study shows in table III a highly significant reverse correlation between BMI and HDL and highly significant correlation between BMI and LDL which coincides with recent studies done by Y. Zhu et al 2016 showing that BMI is the best anthropometric indicator to identify cardiovascular risk factors (Zhu *et al*, 2016). Also, De Onis *et al* (2013) demonstrated that the increase in BMI is accompanied by a reduction in HDL-c and elevation in plasma insulin, HOMA, TG and LDL-c levels (de Onis *et al*, 2013).

CONCLUSION

In this study, we found that CAP was common in the obese adults in comparison to the age, gender and occupation matched normal weight adults. Among the obese smokers, high prevalence of CAP [86%] was found. Thus smoking is proved to be a risk factor for CAP in obese adults. Also, significantly higher anthropometric [body weight, BMI, WHR, BFP & VFA] and lipid profile [cholesterol, triglycerides, LDL cholesterol] were found in the obese adults. Thus, a periodic screening of these anthropometric measures, lipid profile and cigarette smoking could help the individuals to identify the risk of higher residual cardiovascular incidence such as CAP.

Conflict of interest : none.

REFERENCES

Al-Amodi H S, Abdelbasit N A, Fatani S H, Babakr A T and Mukhtar M M (2017) The Effect of Obesity and Components of Metabolic Syndrome on Leptin Levels in Saudi women. *Diabetes Metab Syndr* **12**, 357-364.

Al-Baghli N A, Al-Ghamdi A J, Al-Turki K A, El-Zubaier A G, Al-Ameer M M and Al-Baghli F A (2008) Overweight and obesity in the eastern province of Saudi Arabia. *Saudi Med J*. **29**, 1319-1325.

Almughamisi M, George T and Harding S (2017) Prevalence of overweight and obesity among children and adolescents in Saudi Arabia. *Proceedings of the Nutrition Society* **76**, (OCE4).

Badran M and Laher I (2011) Obesity in arabic-speaking countries. *J Obes*. **2011**, 686430.

Deen O J, Raheem S A and Kirby D F (2013) Obesity and stroke. *Handbook of Clinical Nutrition and Stroke,* Springer 95-112.

De Onis M, Martínez-Costa C, Núñez F, Nguefack-Tsague G, Montal A and Brines J (2013) Association between WHO cut-offs for childhood overweight and obesity and cardiometabolic risk. *Pub Health Nut*. **16**, 625-630

Dixon Yang, Sunil Iyer, Hannah Gardener, David Della-Morte, Milita Crisby, Chuanhui Dong, Ken Cheung, Consuelo Mora-McLaughlin, Clinton B Wright, Mitchell S Elkind, Ralph L

Sacco and Tatjana Rundek (2015) Cigarette smoking and carotid plaque echodensity in the Northern Manhattan Study. *Cerebrovasc Dis*. **40**, 136–143.

Friedlander A H and Lande A (1981) Panoramic radiographic identification of carotid arterial plaques. *Oral Surgery, Oral Medicine, Oral Pathology* **52**, 102-104.

Ghouri N, Purves D, Deans K A, Logan G, McConnachie A, Wilson J, Gill J M and Sattar N (2015) An investigation of two-dimensional ultrasound carotid plaque presence and intima media thickness in middle-aged South Asian and European men living in the United Kingdom. *PloS one* **10**(4), e0123317.

Guo Y, Jiang X, Chen S, Zhang S, Zhao H and Wu Y (2003) Aortic arch and intra-/extracranial cerebral arterial atherosclerosis in patients suffering acute ischemic strokes. *Chinese Medical Journal* **116**, 1840-1844.

Lavie C J, Milani R V and Ventura H O (2009) Obesity and cardiovascular disease: risk factor, paradox, and impact of weight loss. *J Am Coll Cardiol.* **53**, 1925-1932.

Lou Y, Li B, Su L, Mu Z, Sun M, Gu H, Ni J, Wu Y, Tu J and Wang J (2017) Association between body mass index and presence of carotid plaque among low-income adults aged 45 years and older: a population-based cross-sectional study in rural China. *Oncotarget* **8**, 81261.

Manna P and Jain S K (2015) Obesity, Oxidative Stress, Adipose Tissue Dysfunction and the Associated Health Risks: Causes and Therapeutic Strategies. *Metab Syndr Relat Disord*. **13**, 423-444.

Mantella L-E, Colledanchise K, Zhu T, Bullen M, Hetu M-F, Herr J E, Abunassar J and Johri A M (2017) Carotid Plaque Neovascularization is Increased in Patients With Significant Coronary Artery Disease and Acute Myocardial Infarction. *Am Heart Assoc*. **136**, A15978.

Nemati M, Bavil A S and Taheri N (2009) Comparison of normal values of Duplex indices of vertebral arteries in young and elderly adults. *Cardiovascular ultrasound* **7**, 2.

Nóbrega Montenegro-Neto A, da Silva-Simões MO, Dantas de Medeiros AC, Portela AdS and Ramos de Queiroz MdS (2011) The correlation between anthropometric measurements and biochemical cardiovascular risk markers in the hypertensive elderly. *Rev. salud pública* **13**, 421-32.

Oh P C, Han S H, Kim J S, Park J B and Koh K K (2010) Carotid plaque in absence of traditional and non-traditional cardiovascular risk factors. *International journal of cardiology* **143**, e57-e59.

Petty G W, Brown R D Jr, Whisnant J P , Sicks J D, O'Fallon W M and Wiebers D O (1999) Ischemic stroke subtypes : a population-based study of incidence and risk factors. *Stroke* **30**, 2513-2516.

Raitakari O T, Juonala M, Taittonen L, Jula A, Laitinen T, Kahonen M and Viikari J S (2009) Pulse pressure in youth and carotid intima-media thickness in adulthood: the cardiovascular risk in young Finns study. *Stroke* **40**, 1519-1521.

Serena J, Irimia P, Calleja S, Blanco M, Vivancos J and Ayo-Martín O (2013) Ultrasound measurement of carotid stenosis: recommendations from the Spanish Society of Neurosonology. *Neurología (English Edition)* **28**, 435-442.

Sturlaugsdottir R, Aspelund T, Bjornsdottir G, Sigurdsson S, Thorsson B, Eiriksdottir G and Gudnason V (2016) Prevalence and determinants of carotid plaque in the cross-sectional REFINE-Reykjavik study. *BMJ open* **6**, e012457.

Szczygielska A, Widomska S, Jaraszkiewicz M, Knera P and Muc K (2003) Blood lipids profile in obese or overweight patients. *Ann Univ Mariae Curie Sklodowska Med.* **58**, 343-349.

Taramasso L, Mirabella M, Cappadona F, Bonino B, Riccardi N, Cenderello G, De G S, Viscoli C, Viazzi F and Di A B (2018) Hypertensive versus HIV-Infected Patients: Who Has the Greatest Target Organ Damage? Comparison of Carotid Plaque Prevalence, Intima Media Thickness and Renal Resistive Index in the Two Groups of Patients. *Current hypertension reviews* **14**, 48-55.

Ugwuja E, Ogbonna N, Nwibo A and Onimawo I (2013) Overweight and Obesity, Lipid Profile and Atherogenic Indices among Civil Servants in Abakaliki, South Eastern Nigeria. *Ann Med Health Sci Res*. **3**, 13–18.

West H W, Juonala M, Gall S L, Kähönen M, Laitinen T, Taittonen L, Viikari J S, Raitakari O T and Magnussen C G (2015) Exposure to parental smoking in childhood is associated with increased risk of carotid atherosclerotic plaque in adulthood: the Cardiovascular Risk in Young Finns Study. *Circulation* **131**, 1239-1246.

World Health Organization (2000) Obesity: preventing and managing the global epidemic. Report of a WHO consultation. *World Health Organ Tech Rep Ser* **894**, i-xii, 1-253.

Yang C, Sun Z, Li Y, Ai J, Sun Q and Tian Y (2014) The correlation between serum lipid profile with carotid intima-media thickness and plaque. *BMC cardiovascular disorders* **14**, 181-88.

Yang D, Iyer S, Gardener H, Della-Morte D and Crisby M (2015) Cigarette smoking and carotid plaque echodensity in the Northern Manhattan Study. *Cerebrovascular Diseases* **40**, 136-143.

Zhou H, Wang X, Zhu J, Fish A, Kong W, Li F, Liu L, Yuan X, Gao X and Lou Q (2017) Relation of Carotid Artery Plaque to Coronary Heart Disease and Stroke in Chinese Patients: Does Hyperglycemia Status Matter? *Experimental and Clinical Endocrinology & Diabetes* **126**, 134-140

Zhu Y, Shao Z, Jing J, Ma J and Chen Y (2016) Body mass index is better than other anthropometric indices for identifying dyslipidemia in Chinese children with obesity. *PLOS ONE* **11**, e0149392.