Obesity and hyperlipidaemia in adult males in a Semi-urban community in Jordan

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ABSTRACT

Objective: To describe the relationship between obesity and hyperlipidaemia.

Methods: Cluster sampling technique-probability of households from Sareeh area. A total of 400 apparently healthy adult males aged 30-50 years were invited to participate in the study, of whom 306 completed the study.

A pilot tested interview questionnaire was designed in the study to collect the dietary history. Height, weight, waist circumference, triglycerides (TRIG) and total cholesterol (CHOL) were measured. Obesity was categorized into three groups as indicated by BMI-C based on WHO (1997) for generalized obesity and for classifications of central obesity using waist circumference categories based on (Lean et al, 1995).

Results: The means of plasma triglycerides and total cholesterol increased significantly with increasing levels of waist circumference and BMI categories ($P<0.05$).

There was an increase in the prevalence rates (PRs) of hypertriglyceridaemia and hypercholesterolaemia with increasing levels of obesity and its duration. The proportion ratios of hypertriglyceridaemia and hypercholesterolaemia for subjects with high and moderate obesity levels compared with subjects with normal levels were (10.1-11.6) and (6.7 -8.1) times respectively; whereas for subjects with duration of overweight>20 years compared with subjects with duration of obesity <10 years ranged (2.11-2.96) times. Also odds ratios (ORs) of hypertriglyceridaemia and hypercholesterolaemia after controlling for confounded factors were increased with increasing levels of obesity and its duration, the highest (OR) in high obesity levels ranged (11.59-19.18, 95% CI, $P<0.001$) whereas for duration of obesity >20 years ranged (16.78-20.94, 95% CI, $P<0.001$). Central obesity had a potential risk on hypertriglyceridaemia and hypercholesterolaemia more than generalized obesity.

Conclusions: amount of body fat, location and duration are the major risk factors on hyper- trigly ceridaemia and hypercholesterolaemia which emphasizes the need for concentrated e-ffort to prevent and treat obesity rather than just any associated co-morbidities.

Keywords: Obesity, body mass index(BMI) , waist circumference , hyperlipidaemia, Jordan

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ملخص البحث

EDITORIAL

البدانة وفرط الشحميات في الدم عند الذكور البالغين في مجتمع شبه الحضر في الأردن

طريقة الدراسة: تهدف الدراسة بشكل رئيس إلى معرفة العلاقة بين السمنة و شذوذ شحميات الدم، عند الذكور البالغين، في مجتمع شبه الحضر في الأردن الذين تراوحت أعمارهم من 30-50 عاماً وبدوا اصحاء. واعتمدت منطقة الصريح حالةً دراسيةً، واستخدمت طريقة العينة العشوائية العنقودية في اختيار بيوت السكان المحليين، بسحب أفراد العينة البالغ عددهم 400 فرد. وكان معدل الاستجابة النهائي للأفراد المطابقين لشروط الدراسة الذين أكملوا خطوات الدراسة كافةً 76.5% (306) أفراد بدوا أصحاء، وبنسب أعداد متقاربة للمواقع كلها، ولتصنيفات مؤشر الكتلة الجسمية الرئيسة(BMI-C) المعتمد من قبل منظمة الصحة العالمية 1997 لتصنيف السمنة المتعممة (وزن طبيعي أقل من25 ، زيادة وزن 25-29.99 ، بدانة ≥ 30 كغم/م^2 . كما استخدم لتصنيف السمنة المركزية محيط الخصر المعتمد من قبل الباحث لين ورفاقه" (1995).

وقد استخدم لهذه الغاية مقابلة واستبيان شامل، صمم لجمع التاريخ الغذائي ، والوزن، والطول، ومحيط الخصر. وشحميات الدم ومنها: الدهون الثلاثية (TRIG) ،وإجمالي الكوليسترول (CHOL).

النتائج : أظهرت أن المعدل الحسابي(CHOL,TRIG) اظهر فروقات طردية عند مستوى حدث ≤ 0.05 بين مستويات السمنة باستخدام مؤشر كل من الكتلة الجسمية ومحيط الخصر مع زيادة رتبة نمط السمنة.

كما أظهرت الدراسة أن معدل انتشار شذوذهما يزداد مع زيادة مستوى كل من رتبتها، ومدتها، حيث تبين أن نسب معدل الانتشار للنمط العالي للسمنة تراوحت مابين (10.1 –11.6) ضعف، وان المستوى المعتدل للسمنة تراوح مابين (6.1 - 8.6) ضعف إذا ما قورنا بالمستوى الطبيعي(غير البدناء). بينما كان معدل انتشار شذوذهما لمدة السمنة ≤ 20 سنة إذا ما قورنت بمدة السمنة > 10سنة بالمستوى يتراوح مابين (2.11-2.96)ضعف.

وتوصلت الدراسة إلى أن عامل الخطر (OR) لمؤشرات السمنة، ومدتها بعد السيطرة على عوامل بيولوجية، وبيئية مختارة على شذوذ (CHOL,TRIG) يزداد مع زيادة مستوى كل من رتبتها، ومدتها. حيث كان يتراوح لذوي النمط العالي للسمنة مابين (11.59-19.18) ،ولمدة السمنة ≤ 20 سنه مابين(16.78-20.94) عند مستوى ثقة 95% ومستوى حدث ≤0.001. ووجدت الدراسة أن تأثير السمنة المركزية اكثر حدة من السمنة المتعممة في شذوذ (CHOL,TRIG).

الخلاصة: وخلصت الدراسة إلى أن كمية دهن الجسم، وموقعه، ومدته هي متنبئات رئيسة لشذوذ(CHOL,TRIG). وهذا يؤكد ضرورة تركيز الجهود للوقاية من السمنة ومعالجتها وليس لمعالجة الأمراض المتعلقة بها فقط.

Introduction. Obesity is a widespread metabolic abnormality. It is a growing epidemic worldwide including Jordan and has affected people with different ethnic and cultural backgrounds[1,2,3]. Currently, nearly one quarter of the US population is considered to be overweight (defined as BMI=25-29.9 kg/ m^2), and an additional one quarter is clinically obese, (defined as BMI ≥30 kg/ m^2) [1].

Obesity can be defined as a disease of extensive fat accumulation to the extent that health and wellbeing are affected. However, the degree of excess fat, its distribution within the body, duration and the associated health consequences vary considerably between obese individuals[2]. Obesity has long been recognized as an associated factor with a variety of adverse health consequences chiefly among which are diabetes[4,5], hypertension[6,7], dyslipidaemia[8,9,10], increased cardiovascular events and mortality[11], and hyperuricaemia[12] . These patients are also more likely to present with silent disease[14,15] and as a cluster of metabolic syndrome[2,16]. The most commonly recognized risk factors in the metabolic syndrome are highly correlated with each other[17] and are pre-sumed to reflect common metabolic pathways and they interact to increase risk in a synergistic fashion[18].

Furthermore, several epidemiological studies showed a positive association between obesity and hyperlipidemia[19,20]. Increases in plasma lipoproteins concentration showed positive association with body mass index (BMI), waist circumference, and waist to hip ratio (WHR) [21,22,23]. The risk was increased among men who had been overweight in adolescence [24]. It has been suggested that other factors, such as dietary fat from animal source, genetic susceptibility, sedentary life style and age, may also play a role in producing hyperlipidemia[25, 26,27,28,29,30,31,32,33,34.] It seems from the above that several ethnic groups are more prone to the development of obesity and its complications. Furthermore, a change from the traditional to affluent sedentary life style and accompanying diet availability, low price of fat-rich energy-dense diet, and reduced activity from unemployment might be considered as main causes of obesity or/and may facilitate expression of genetic predisposition of obesity and related diseases[35]. Also[36] and [37] reported that weight reduction could reverse the abnormal biochemical characteristics of obesity. Most Clinical studies assessing the health effect of peripheral obesity rely on BMI,[38] whereas central obesity is determined by waist circumference and waist to hip circumference ratio[22,39,23] .

Recent studies conducted in Jordan indicate high prevalence of diabetes mellitus, hypertension and hyperlipolipidemia[6,40 ,41 ,42]. Also cardiovascular diseases are the first leading cause of death in Jordan as reported by annual reports of Royal medical services[43], (RMS, 2001). Data are needed regarding the relationship between obesity and hyperlipidemia in Jordan. It is important that factors inducing hyperlipidemia should be addressed in any coordinated strategy to tackle the problem of hyperlipidemia and associated co-mobidities.

MATERIALS AND METHODS.Cluster sampling technique-probability of households from Sareeh area in the northern of Jordan was used to recruit the studied sample .A total of over three hundred of apparently healthy adult males aged 30-50 years were actually included in the study which was conducted in 2001. The sample was categorized into approximately equal three groups, normal body weight, overweight and obese as indicated by BMIs: < 25, 25-29.99 and ≥30 kg /m²

EDITORIAL

respectively. Also central obesity was categorized in three groups as indicated by waist circumference: normal< 94 cm; moderate ≥ 94- 102; high > 102 cm based on waist circumference classifications[44].

A modifiable and pilot tested interview questionnaire was designed in the study to collect demographic, socio-cultural, dietary and physical activity factors, current smoking, history of weight and disease for participants and their parents, in addition to blood lipids triglycerides (TRIG) and cholesterol (CHOL),

Anthropometric measurements were performed using the Anthropometric Standardization Reference Manual[45] . Laboratory measurements were performed using standard automated procedures (Hitachi 911) with commercially available kits (Randox manual procedures edition 111, 1990 or Roche Diagnostics, 2000). Hypertriglyceridaemia is defined as serum triglycerides level of ≥ 200 mg/dl whereas elevated plasma cholesterol defined as plasma cholesterol level of ≥ 200 mg/dl for men aged < 40 years, ≥ 240 mg/dl for men aged ≥ 40 years. An informed consent was obtained from each subject. The study was approved by the Research Ethics Committee of Philadelphia University- Jordan.

Data management and analysis. The data was entered into a computer using the SPSS (statistical Package for Social Sciences, Windows version 8x, 1997; SPSS Inc, Chicago, 1L, USA. Frequency and range checks were performed initially to detect errors in data entry. Detected errors were corrected by rechecking the original data formed.

The prevalence of hypertriglyceridaemia and hypercholesterolaemia and comparing proportions in the study sample among population subgroups based on overall and central obesity index categories were analyzed using X^2 test. Analysis of variance (ANOVA) was performed to test significance for mean values of variables between overall and central obesity categories.

Logistic regression analysis was carried out to estimate risk or odds ratios (ORs) of obesity indices, obesity duration categories, selected biological and environmental factors on hypertriglyceridaemia and hypercholesterolaemia. Hypertriglyceridaemia and hypercholesterolaemia were subdivided into binary variables (normal and abnormal values) and used as a dependent variable, whereas obesity indices, duration of obesity categories and selected biological and environmental factors were used as independent variables. A P value of < 0.05 was used as the criterion of statistical significance.

Results. The mean of plasma triglycerides and total cholesterol increased with increasing BMI and waist circumference categories and showed significant mean differences among BMI-C and W-C ($P<0.05$) as shown in Table (1a,b).

Table 2a,b shows prevalence rates and ratios of the proportion of subjects with hypertriglyceridaemia and hypercholesterolaemia among obesity categories increased as (BMI-C and W-C) increased.

The results shown in Table (2a,b) indicate that all the absolute proportions of hypertriglyceridaemia and hypercholesterolaemia increased as obesity levels increased. Prevalence rates of

hypertriglyceridaemia and hypercholesterolae-mia among subjects with high level of central obesity or obese category were more than ten times in comparison with subjects with low level of central obesity level or non-obese category, whereas for subjects with moderate level of central obesity or overweight category were above six times for hypertriglyceridaemia and above eight for hypercholesterolaemia in comparison with subjects with low level of central obesity or non-obese category.

Table 1a Descriptive and comparative data for plasma triglycerides and total cholesterol. The study sample classified into non-obese, overweight and obese using body mass index categories (BMI-C).

DEPENDENT VARIABLE	BMI-C	NO	MEAN	± SD.	BMI-C		MEAN DIFFERENCE	± STD. ERROR	P
Age/years	0	103	38.60	6.31	0	1	-0.23	0.91	0.969
	1	100	38.83	6.63	2	0	0.90	0.90	0.612
	2	103	39.49	6.50	2	1	0.67	0.91	0.764
TRIG	0	103	126.41	59.01	0	1	-52.61	11.40	0.000
	1	100	179.02	84.81	2	0	84.24	11.32	0.000
	2	103	210.65	95.58	2	1	31.63	11.40	0.022
CHOL	0	103	179.80	29.75	0	1	-33.71	5.71	0.000
	1	100	213.51	45.02	2	0	52.50	5.66	0.000
	2	103	232.29	45.36	2	1	18.78	5.71	0.005
* Results of one-way ANOVA and Scheffe post hook analysis (0) non-obese, (1) overweight, (2) obese									

Table (3) shows adjusted odds ratio (OR) of hypertriglyceridaemia of obesity categories after controlling for age, weight history, parental (CVD, and hypertension), current smoking, physical activity and percentage of calorie from fat in the study sample

The adjusted risk of hypertriglyceridaemia among subjects with moderate central obesity or overweight, and high central obesity or obese levels compared with subjects with low level of central obesity categories or non-obese were (ORs 5.08-6.27, 95%CI) and (ORs11.59 -13.8, 95%CI) respectively.

Parental cardiovascular disease (CVD), age, percentage of calories from dietary fat (%CDF), childhood and adolescence obesity were associated significantly with hypertriglyceridaemia ($P<0.05$). These results show obesity is the dominant risk factor on hypertriglyceridaemia.

Table 1b Descriptive Characteristics (Mean+SD) and Multiple Comparisons plasma hypertriglyceridaemia and hypercholesterolaemia level using waist circumference categories (W-C) for Study Population

EDITORIAL

Dependent variable	W-C	No	Mean	±SD.	W-C		Mean Difference	±Std. Error	P
TRIG	0	103	126.37	59.01	0	1	-54.24	11.22	0.000
	1	107	180.61	85.60	2	0	84.86	11.53	0.000
	2	96	211.23	95.70	2	1	30.62	11.43	0.000
CHOL	0	103	179.81	29.75	0	1	-32.59	5.57	0.000
	1	107	212.39	45.03	2	0	55.09	5.72	0.000
	2	96	234.90	44.48	2	1	22.50	5.67	0.000
* Results of one-way ANOVA and Scheffe post hook analysis (0) Low (1) moderate (2) high									

Table 2a: Prevalence rate and ratios proportions of subjects with biochemical abnormalities among body mass index categories (BMI-C) in the study sample

Indicators	Overall obesity index Body mass index categories (BMI-C)							
	Non-obese		rweight		R	Obese		R
	N.	%	N.	%		N.	%	
	103	33.7	100	32.6		103	33.7	
TRIG	5	4.9	30	30.0	6.2	52	50.5	10.4
CHOL	5	4.9	43	43.0	8.9	53	51.5	10.6

Table 2b: Prevalence rate and ratios proportions of subjects with hypertriglyceridaemia and hypercholesterolaemia among waist circumference categories (W-C) in the study sample

Waist circumference (W-C)								
Indicator	Low		Moderate		R	High		R
	N.	%	N.	%		N.	%	
	103	33.6	107	35.0		96	31.4	
TRIG	5	4.9	35	32.7	6.7	47	49.0	10.1
CHOL	5	4.9	42	39.3	8.1	54	56.0	11.6
All (R): calculated with each category by dividing the proportions of biochemical abnormalities overweight or moderate or high or obese level / low-level proportion or non-obese proportion. All ratios of proportions were significantly larger than 1.00 at P<0.5 based on Chi-square statistic.								

Table (4)shows significance of adjusted risk (ORs)of high plasma cholesterol for participants with moderate level and high levelcentral obesity in comparison with low level of central obesity were (10.9, 95%CI) and (19.18, 95%CI) respectively. Whereas odds ratios of high plasma cholesterol for overweight and obese subjects in comparison with non-obese subject were (6.5, 95%CI) and (14.03, 95%CI), respectively. Central obesity categories had a higher (ORs) than overall obesity categories.

Results shown in Table (5) indicate that the prevalence rates of hypertriglyceridaemia and hypercholesterolaemia increase with increasing duration of overweight ((BMI $\geq$ 25 kg/m^2). The prevalence rate (PR) of hypertriglyceridaemia among subjects with duration two and subjects with duration three, in comparison with the reference group, the subjects with duration one; were (1.82; 2.69) fold,respectively.Whereas the prevalence rate of hyperchole- sterolaemia among subjects with duration two and subjects with duration three in comparison with the reference group subjects with duration one were (1.94; 2.11) fold, respectively.

Table 3: Adjusted odds ratio (ORs) of hypertriglyceridaemia of obesity categories after controlling for age, weight history, cardiovascular disease(CVD), and hypertension among parents, current smoking, physical activity and percentage of calorie from dietary fat in the study sample

		95% CI for Exp (B)		
Variable	Exp (B)	Lower	Upper	P
Body mass index categories(BMI)				
Parental (CVD)	22.11	1.16	3.82	0.0145
Childhood obesity	0.29	0.10	0.88	0.0 283
Adolescence obesity	1.82	0.76	4.36	0.0401
%CDF (3)	2.71	1.20	6.12	0.0168
BMI-C (1)	5.08	1.79	14.43	0.0023
BMI-C (2)	13.18	4.57	19.5	0.0000
Waist circumference categories(W-C)				
Parental (CVD)	2.13	1.18	3.84	0.117
Childhood obesity	0.28	0.09	0.84	0.0229
Adolescence obesity	2.06	0.88	4.81	0.0958
%CDF(3)	2.79	1.24	6.27	0.0134
W-C (1)	6.27	2.25	17.48	0.0004
W-C (2)	11.59	3.94	34.07	0.0000
* Each obesity indicator entered separately with controlling factors*%CDF-C(3) = % of calorie from dietary fat$\geq$ 34.5				

EDITORIAL

The results shown in Table (6) indicate that duration of overweight had a risk on hypertriglyceridaemia and hypercholesterolaemia, which increased, steeply in duration 1 and sharply in duration two and three. Also, the results showed that the risk of hypercholesterolaemia increased steeply with small difference between duration two and three. Also, the results showed that the risk of hypertriglyceridaemia increased sharply between duration two and three.

Table 4: Adjusted odds ratio (ORs) of hypercholesterolaemia of obesity categories after controlling for age, weight history, CVD, and hypertension among parents, current smoking, physical activity and percentage of calorie from dietary fat in the study sample

		95% CI for Exp. (B)		
Variable	Exp. (B)	Lower	Upper	P
Body mass index categories(BMI)				
Childhood obesity	2.00	0.90	4.40	0.0870
%CDF-C(3)	1.89	0.92	3.89	0.0823
BMI-C (1)	12.6.5	4.62	34.59	0.0000
BMI-C (2)	14.03	5.03	39.10	0.0000
Waist circumference categories(W-C)				
%CDF-C(3)	1.71	0.83	3.51	0.1471
W-C (1)	10.90	4.01	29.59	0.0000
W-C (2)	19.18	6.87	53.60	0.0000

* Each obesity indicator entered separately with controlling factors *%CDF-C(3) = % of calorie from dietary fat≥ 34.5

Table 5: Prevalence rates and ratios proportions of the subjects with hypertriglyceridaemia and hypercholesterolaemia among duration of overweight (BMI≥ 25 kg/m^2) in the study sample

Indicator	Duration1		Duration2		R	Duration3		R
	N	%	N	%		N	%	
	49	16.0	70	23.0		84	27.0	
TRIG- C(1)	10	20.4	26	37.1	1.82	46	54.8	2.69
CHOL- C(1)	13	26.5	36	51.4	1.94	47	56.0	2.11

* Ratios were significant in all categories
*All (R): calculated with each categories by dividing the proportions of hypertriglyceridaemia and hypercholesterolaemia in duration 2 or duration 3 / duration 1 proportion.
*All ratios of proportions were significantly larger than 1.00 at P<0.5 based on Chi-square statistic.
*Duration of obesity 1= <10 yrs; 2 = 10-20; 3= >20

Table 6: Adjusted odds ratios (ORs) of duration of overweight (BMI ≥ 25kg/m^2) on hypertriglyceridaemia and hypercholesterolaemia after controlling for age, weight history, CVD,

and hypertension among parents, current smoking, physical activity and percentage of calories from dietary fat in the study sample

Hypertriglyceridaemia (TRIG- C(1))				
95% CI for Exp (B)				
Variable	Exp (B)	Lower	Upper	P
parental (CVD)	1.7466	0.9619	3.1715	0.0669
%CDF-C(3)	2.9661	1.3216	6.6572	0.0084
Duration (1)	3.3650	1.0359	10.9314	0.0435
Duration (2)	8.5595	2.9702	24.6667	0.0001
Duration (3)	16.7750	5.7349	49.0683	0.0000
Hypercholesterolaemia(CHOL- C(1))				
Age-C (2)	0.5208	0.2886	.9399	0.0303
%CDF-C(3)	1.7996	0.8602	3.7651	0.0303
Duration (1)	4.7569	1.5264	14.8248	0.0072
Duration (2)	18.0647	6.5531	51.4020	0.0000
Duration (3)	20.9417	7.2924	60.138	0.0000
Duration of obesity: 0 non-obese; 1= <10 yrs; 2 = 10-20; 3= >20* age (2) ≥ 40 years				

These results revealed that a relative risk for hypertriglyceridaemia and hypercholesterolaemia is dangerous in participants with duration of overweight more than 10 years. Also, parental CVD and subjects whose caloric intake from dietary fat was ≥34.5%(%CDF-C(3)) showed significant relative risk on hypertriglyceridaemia. Also, subjects whose caloric intake from dietary fat was ≥33.5% and aged ≥ 40 years(age-C(2)) showed significant risk on hypercholesterolaemia.

Discussion

Obesity and hypertriglyceridaemia

In this study the relationship between overall obesity and central obesity with hypertriglyceridaemia was investigated using different statistical tests. As shown in Table (1a,b), the mean difference of plasma triglycerides when comparing obese with non-obese was 84.1 mg/dl and between non-obese with overweight was 52.61 mg/dl, also, between obese and overweight was 31.63 mg/dl. These results are in agreement with the results revealed by[46] who reported that, the difference in triglycerides ranged from (62-118) mg/dl between obese and lean. Also, the mean of plasma triglycerides for obese and high level of central obesity categories was above the recommendations by American Heart Association (AHA) to keep triglycerides below 200 mg/dl as reported by[47,48] .

EDITORIAL

Furthermore, Table (2ab) shows that the prevalence rates increased as obesity categories increased. Prevalence rate of hypertriglyceridaemia among subjects with obese or high level central obesity categories was 10 times than those subjects with non-obese or low level of central obesity categories. Also prevalence rate of high plasma triglycerides among subjects with overweight and moderate level of central obesity categories was 5.6 times than those subjects with non-obese or low level of central obesity categories. These results are in agreement with the results of a study conducted in Jordan on a community based survey of a sample of 2152 persons aged 25 years or more selected from three locations in Jordan. The results indicate that obese subjects had a higher rate of hypertriglyceridaemia (27.1%) compared to that of non-obese subjects, despite some differences in the two populations. Furthermore, hypertriglyceridaemia has been associated with obesity in several national studies [46,49,50,51].

Furthermore, the study findings showed central obesity and overall obesity in relation to plasma elevated plasma triglycerides were the dominant risk factors on elevated triglycerides after controlling for selected biological and environmental factors as shown in Table (3,4). Adjusted risk of hypertriglyceridaemia for overweight or moderate level of central obesity and obesity or high level of central obesity compared with low central obesity level or non-obese ranged (OR 5.08-6.27, 95% CI) and (ORs11.33-13.97, 95% CI) respectively. It is concluded that obesity is associated significantly and independently with hypertriglyceridaemia, in agreement with the findings of several studies[52, 53,37,23,54,55.] This result may be considered as indirect evidence that two basic mechanisms exist for hypertriglyceridaemia of obesity, overproduction of very low-density lipoprotein (VLDL) and defective lipolysis of the triglycerides (TG) rich lipoprotein[56].

One of the important findings in this study indicate that other factors such as %CDF$\geq$34.5 is associated significantly and independently with hypertriglyceridaemia compared with subjects whose %CDF $\leq$ 33, with significant adjusted (OR) (2.0-3.5; 95% CI). This result is in agreement with several studies[57,58,59].

Furthermore, hypertriglyceridaemia was associated significantly with subjects who reported childhood obesity, adjusted relative risk was about 29% higher than those subjects who did not report obesity during childhood. Also subjects who had reported adolescence obesity in comparison with subjects who had not had relative risk of hypertriglyceridaemia (OR 2.0, 95% CI), as have been reported [60,61,62,24]. The results of relative risk of childhood and adolescence obesity on hypertriglyceridaemia may explain indirectly the changes in body fat distribution that occur at adolescent fatness on adult morbidity. Visceral fat occupies approximately 50% of the cross-section of visceral fat area in 11-13 years old children[63]. Between adolescence and childhood the cross-sectional visceral fat area increases four to five fold [23]or may be due to other mechanisms including genetic factors that entrain both body fatness and adult morbidity [64] .

Parental CVD was associated significantly and positively with hypertriglyceridaemia ($P<0.05$). This result indirectly may indicate role of genetic factors[32,65].

The study results revealed the potential risk of overall and central obesity on hypertriglyceridaemia. Furthermore, potential risk of hypertriglyceridaemia was higher among subjects with moderate levels of central obesity compared with overweight subjects,

while high level of central obesity and obese subjects revealed approximately the same magnitude of relative risk. It is concluded that obesity is the dominant risk factor on hypertriglyceridaemia, whereas central obesity had a potential effect on hypertriglyceridaemia more than overall obesity.

Obesity and high plasma total cholesterol

All statistical investigations performed in this study revealed that the mean level of plasma cholesterol increased according to BMI categories and central obesity categories. The mean difference between obese or high level of central obesity compared with non-obese or low level of central obesity was (52.0 – 55.0) mg/dl as shown in Table (1 a, b)

Comparison of the current study findings with those from other Arab countries is hampered by the different methods used. The mean values of plasma cholesterol among overweight and obese participants were higher than that reported by [66]in central Saudi Arabia but the two populations were totally different. [67]reported that the mean cholesterol level of men from Western Saudi Arabia was (183.0) mg/dl. In Jordan the mean cholesterol level of men was (203) mg/dl [41]. Han et al; 1995 conducted a study on a random sample of 2183 men and 2698 women, 20-59 years old from Netherlands. The mean cholesterol level for men with waist circumference less than 94 cm was 201.6 mg/dl and for men with waist circumference ≥94-<102 cm was 222.7 mg/dl and for men with waist circumference more than 102 cm 229.3 mg/dl. In the present study we observed higher values of cholesterol for overweight or moderate level of central obesity (212.34) and for obese and high level of central obesity (232.29 -234.9) mg/dl. Explanation for the high level of plasma cholesterol in the study population may be because food consumption, life style and genetic factors could be of minor role while obesity was largely responsible for our findings.

Furthermore, investigations in this study indicate that the prevalence rates of high plasma cholesterol increased with increasing BMI categories and central obesity categories as shown in Tables (2a, b). The prevalence rates (PRs) of hypercholesterolaemia level were 10 times among subjects with obesity or high level of central obesity and 8 times in subjects with moderate level of central obesity or overweight compared with non-obese or low level of central obesity respectively. These results are in agreement with several studies which indicate that high level of cholesterol increased as body weight increased [21]and confirmed with the NHANES III on 16884 adults, 25 years and older[68] and as has been found by [41]. But the magnitude of the prevalence rate was higher among our study population.

The current study findings as shown in Table (4) indicate that risk of hypercholesterolaemia for subjects with overweight or moderate level of central obesity level and subjects with obese category or high level of central obesity, in comparison with the reference group non-obese or low level of central obesity categories, had significant relative risk (OR 6.12, 95% CI) and (OR >10.0, 95% CI) respectively. Furthermore, estimated adjusted relative risk (ORs) for the following factors; comparing: smoking with non smoking, subjects aged 40-50years with 30-40 years old, %CDF≥ 34.5 with ≤ 33.0%, childhood obesity, history of coronary heart diseasess were independently and significantly associated with high level of plasma cholesterol, and had significant adjusted relative

EDITORIAL

risk (OR) on high plasma cholesterol ranging from (1.72-3.93, 95% CI). Our findings in the current study are in agreement with several studies [41,58,59,22,34].

Relationship of duration of obesity with hypertriglyceridaemia and hypercholesterolaemia

Obesity with both its magnitude and location is the most powerful risk factor on hypertriglyceridaemia and hypercholesterolaemia shown in the study results. The current findings of this study shown in Table (5) indicate that prevalence rates of hypertriglyceridaemia and hypercholesterolaemia increased significantly as duration of obesity (BMI≥ 25kg/ m^2) increased. Furthermore, obese subjects with duration of obesity ≥ 20 years had the highest prevalence rates of hypertriglyceridaemia and hypercholesterolaemia. Furthermore, our findings indicate that duration of obesity is the dominant independent risk factor on hypertriglyceridaemia and hypercholesterolaemia Table (6). The scarcity of such data and the different methods of presentation hamper comparison of our data with those from Arab countries or worldwide. Obesity may exert its effects only after a long follow-up period. Duration of obesity may reflect the time it takes for the metabolic and other effects of obesity to have their full impact. In addition to that the age of the subjects was 30-50 years, so obese subjects who had reported long duration revealed that the onset of obesity may be in childhood or adolescence or early adulthood. Indirectly several studies findings support our data.[61] concluded from his study on 508 lean and overweight adolescents 13 to 18 years old who participated in the Harvard Growth study of 1922-1935 overweight in adolescence predicted a broad range of adverse health effects that were independent of adult weight after 55 years of follow-up. Also, [24]reported that from cross-sectional survey as a part of the Bogalusa heart study of cardiovascular risk in 1176 individuals (62% female, 44%) aged 5 through 17 years at baseline were followed-up for 8 years. That plasma lipid and lipoprotein level, blood pressure and plasma insulin all followed-up from childhood into your adulthood with obesity at base line, become a significant predictor of adult values. Also [69,70]reported that, dyslipidemia, hypertension and insulin resistance is frequently present in obese children. The current data of this study emphasize that obesity may generate its associated risk factors at a subsequent time. Obesity that develops at an early age in adults and is sustained effect on life long is different from the effect of obesity that develops in middle age[71]. Findings from the Manitoba,[72] study, along with findings from the analysis from the Framingham using study[73] suggest that the duration of being obese has an important bearing on the putative relationship of body weight and longevity. Data from the Framingham Heart study were analyzed using a longer time interval between measurement of obesity and subsequent outcome show that obesity clearly, was a significant predictor for cardiovascular disease, independent of age, cholesterol level, systolic blood pressure, cigarette smoking, left ventricular hypertrophy and glucose intolerance. Furthermore, associations between body mass indices and CVD became apparent only after 16 [73] or 8 to 14 [74]years follow-up.

Duration of obesity when taken into account will lead to reduction of many discrepancies between studies of populations with different ages. Different initial health status and different follow-up can be resolved. Furthermore, in short term studies, duration of obesity should be taken into account. A number of adverse health effects that occur in adulthood are associated with long duration of obesity. Because body mass index appears to be programmed early in life, the

prevention of overweight in childhood, adolescence and early adulthood may be the most effective means of decreasing the associated co-morbidities in middle and late adulthood.

Conclusions. Results of the study indicate that amount of body fat, its location and duration are the dominant risk factors on hypertrigly- ceridaemia and hypercholesterolemia, which emphasizes that prevention and treatment of obesity may be the most effective means of decreasing hypertriglyceridaemia and hypercholesterolemia and associated comorb-idities.

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