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ORIGINAL ARTICLE

Obesity: An Important Predictor of Metabolic Syndrome

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Abstract

Background/Aim: The leading avoidable cause of mortality in the world is obesity. It modifies how the body reacts to insulin, which might result in insulin resistance and an elevated risk of type 2 diabetes. Recent investigations have revealed a link between obesity and the metabolic syndrome. Therefore, it was intended for the current study to look at the prevalence of obesity and how it relates to the metabolic syndrome.
Methods: 120 healthy males between 30-50 years of age were chosen from the general population of Kota District, Rajasthan, India and screened for obesity and divided in three groups: normal, overweight and obese. Each group comprised of forty subject. Serum was separated and run in department of biochemistry, GMC Kota. Anthropometric parameters were taken. The completely automated analyser ERBA EM 360 performed lipid profile and blood sugar analyses on serum.
Result: Obesity increased metabolic syndrome prevalence. In comparison to overweight and normal weight people, obese subjects exhibited considerably lower levels of high-density lipoprotein (HDL) cholesterol and significantly higher levels of blood glucose, triglycerides, total cholesterol and low-density lipoprotein (LDL) cholesterol. Additional coronary artery disease (CAD) risk prediction ratios, such as total cholesterol/HDL, LDL/HDL and triglycerides/HDL ratios that have gradually increased from the normal to obese group, show that obese subjects have a relatively higher risk of developing cardiovascular diseases (CVD) than do those in the overweight and normal groups.
Conclusion: One of the key elements of metabolic syndrome, which is a collection of clinical and metabolic anomalies including abdominal obesity, insulin resistance, hypertension and dyslipidaemia, is obesity. Each of these conditions raises the risk of CVD and diabetes mellitus type 2 directly. The progression towards diabetes and CVD can be prevented by lifestyle modification programmes and regular health checks (to explore the risk factors of metabolic syndrome).

Key words: Obesity; Metabolic syndrome; Lipid profile.

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Introduction

In cases of obesity, extra body fat builds up to the point that it may have a negative impact on health, reducing life expectancy and/or increasing health issues. Obesity is a leading avoidable cause of mortality globally. Most cases of obesity are assumed to be caused by a combination of excessive dietary energy consumption and a lack of physical exercise.[1] Insulin resistance may result from altered insulin responses caused by increased body fat. Fat accumulation in the subcutaneous and visceral depots is vulnerable to metabolic and cardiovascular problems because it induces a pro-inflammatory and pro-thrombotic state.[2]

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Metabolic syndrome (MetS) is defined as a number of clinical and metabolic problems, as abdominal obesity, insulin resistance, hypertension and dyslipidaemia, have been demonstrated to enhance the chance of developing diabetes mellitus type 2 (DMT2) and cardiovascular diseases (CVD).[3] Right now, the NCEP's ATP III and IDF definitions, which employ waist circumference as a proxy for central obesity, are the two most often used definitions. According to the NCEP ATP III definition, MetS is present if three or more of the following five criteria are met: a waist circumference greater than 40 inches for men (100 cm) and 35 inches for women (90 cm); blood pressure greater than 130/85 mmHg; a fasting TG level greater than 150 mg/dL; a fasting high-density lipoprotein (HDL) level less than 40 mg/dL for men and 50 mg/dL for women; and a fasting blood sugar level greater than 100 mg/dL. One of the most often used criteria for the MetS is the NCEP ATP III definition. Visceral obesity, atherogenic dyslipidaemia, hypertension and hyperglycaemia are all included as important characteristics. Oxidative stress and the depletion of antioxidants are essential pathogenic mechanisms in the development of obesity-associated MetS, diabetes and its consequences, according to experimental and clinical studies.[4]

Excessive fat build-up in the MetS promotes oxidative damage in adipose tissue and releases free radicals. These free radicals have a high degree of reactivity with nearby molecules including lipids, proteins and carbohydrates and they can harm cells.[5] Oxidative stress is a situation that develops when the generation of harmful free radicals outpaces the body's antioxidant defences' ability to cleanse them. Reactive oxygen species (ROS) are all free radicals that include oxygen.[6] The superoxide anion radical (O_2 •–) and the hydroxyl radical (•OH) are the ROS that are most often generated.[7] The production of O_2•– starts a chain reaction that quickly produces additional free radicals and, in the end, results in the synthesis of H_2O.[8] Antioxidants are substances that can protect bodily tissues from the harmful effects of oxidation. The production of atherosclerotic plaque requires the oxidative alteration of low-density lipoprotein (LDL), which has been associated to low plasma antioxidant levels.[9] The most significant factors may be dietary and lifestyle choices. Obesity has been linked to inadequate fruit and vegetable consumption.[10]

The goal of the this study was to determine the prevalence of obesity and how it relates to the MetS.

Methods

This study was conducted from September 2011 to August 2012 in Department of Biochemistry, Central Laboratory NMCH and MBS Hospital Kota, Rajasthan, India. For this study 120 healthy males between 30-50 years of age were selected from the general population of Kota District and accordance to recent recommendations made by the Indian Ministry of Health, who have been classified by body mass index (BMI) in 3 groups: normal, overweight and obese. Each group comprised of forty subjects.

Following anthropometric measurements were performed: body mass (kg), height (m) using a standard measuring tape, BMI, waist size (cm), hip size (cm), waist to hip ratio, heart rate (beats per minute – bpm). Serum was used for the following biochemical investigations: fasting blood glucose (mg/dL), level of serum HDL cholesterol (mg/dL), level of serum LDL cholesterol (mg/dL), level of serum total cholesterol (mg/dL), level of serum triglycerides (mg/dL), total cholesterol/HDL ratio, LDL/HDL ratio, triglyceride/HDL ratio.

Participants underwent revised NCEP ATP III criteria for MetS evaluation. New NCEP ATP III Standards:

1. Men should have a waist circumference of at least 90 cm, while women should have one at least 80 cm (according to newly released recommendations issued by the Indian Ministry of Health).
2. A triglyceride level of 150 mg/dL or more is considered to be high.
3. Low HDL ("good") cholesterol level is less than 40 mg/dL for men and less than 50 mg/dL for women.
4. Blood pressure is elevated if it is measured 130/85 mm Hg or above, or if patient is using a hypertension medication.
5. Elevated fasting glucose: 100 mg/dL or above, or patient is using a hyperglycaemia treatment.

Subjects were categorised as having MetS if they satisfied three out of these five criteria.

Excel was used to enter the data and SPSS software version 21.0 was used for analysis. Qualitative data was expressed in percentage and quantitative data expressed as mean ± standard deviation (SD).

Results

Prevalence of MetS is shown in Table 1.

Table 1: Prevalence of metabolic syndrome

Body mass	N	Metabolic syndrome	
		N	%
Normal	40	2	5.00
Overweight	40	6	15.00
Obese	40	25	62.50

N: number of subjects;

Prevalence of MetS increased with obesity (increasing BMI). As shown in the Table 1, 62.5 % obese subjects were having MetS while 15 % overweight and 5 % normal subjects had this syndrome. Comparison of anthropometric parameters of all 3 groups is shown in Table 2.

Waist circumference and the waist-to-hip ratio significantly increased along with the rise in BMI in obese people. Further, as blood pressure rose with rising BMI, waist circumference and waist/hip ratio, both systolic and diastolic blood pressure were linked to obesity ($p < 0.05$).

Table 2: Comparison of anthropometric parameters of the groups (normal, overweight and obese subjects)

Parameters	Group 1: normal	Group 2: overweight	Group 3: obese	ANOVA	
				F-score	p-value
Body mass (kg)	63.87 ± 5.33	68.75 ± 4.08	84.75 ± 8.79	116.9	< 0.001
Height (cm)	172.03 ± 5.14	169.5 ± 4.68	170.5 ± 5.36	2.51	> 0.05
BMI	21.49 ± 0.93	23.81 ± 0.48	29.2 ± 2.91	196.2	< 0.001
Waist circumference (cm)	78.02 ± 3.64	87.65 ± 2.5	100.27 ± 7.85	183.9	< 0.001
Hip circumference (cm)	82.97 ± 3.85	91.22 ± 3.56	99.1 ± 6.42	113.4	< 0.001
Waist / hip ratio	0.93 ± 0.02	0.95 ± 0.04	1.01 ± 0.04	30.02	< 0.001
Systolic BP (mm Hg)	121.74 ± 10.94	126.22 ± 7.89	127.42 ± 8.84	4.16	< 0.05
Diastolic BP (mm Hg)	80.45 ± 7.44	83.2 ± 6.89	85.05 ± 6.89	4.26	< 0.05

Values are Mean ± SD; BP: blood pressure; BMI: body mass index;

Table 3: Comparison of lipid and blood glucose parameters between the groups (normal, overweight and obese)

Parameters	Group 1: normal	Group 2: overweight	Group 3: obese	ANOVA	
				F-score	p-value
Fasting blood glucose (mg/dL)	87.3 ± 12.41	91.5 ± 10.92	102.6 ± 10.77	19.24	0.001
Triglycerides (mg/dL)	116.42 ± 19.95	117.25 ± 20.4	142.92 ± 26.46	17.98	0.001
Total cholesterol (mg/dL)	185.32 ± 32.83	199.22 ± 32.28	231.75 ± 42.46	12.83	0.001
LDL cholesterol (mg/dL)	106.17 ± 17.14	112.77 ± 20.88	133.95 ± 24.02	19.33	0.001
HDL cholesterol (mg/dL)	50.12 ± 7.31	43.92 ± 3.68	42.4 ± 4.52	22.94	0.001
Total cholesterol/HDL ratio	3.96 ± 1.37	4.64 ± 1.09	5.61 ± 1.33	17.06	0.001
LDL/HDL ratio	2.2 ± 0.63	2.6 ± 0.64	3.21 ± 0.77	22.43	0.001
TG/HDL ratio	2.41 ± 0.71	2.73 ± 0.63	3.41 ± 0.77	21.18	0.001

LDL: low-density lipoprotein; HDL: high-density lipoprotein; TG: triglycerides; Values are presented as Mean ± SD;

Table 4: Prevalence of components of metabolic syndrome

Parameters	Group 1: normal	Group 2: overweight	Group 3: obese
WC ≥ 90 (cm)	0	12	39
BP ≥ 130/85 (mm Hg)	7	16	19
FBG ≥ 100 (mg/dL)	10	10	24
TG ≥ 150 (mg/dL)	3	5	14
HDL < 40 (mg/dL)	2	5	11

WC: waist circumference; BP: blood pressure; FBG: fasting blood glucose; TG: triglycerides; HDL: high-density lipoprotein;

The blood sugar and lipid values of healthy, overweight and obese people are shown in the Table 3. Obesity had a significant impact on cholesterol and blood glucose levels. In comparison to overweight and normal weight people, obese subjects exhibited considerably lower levels of HDL cholesterol and significantly higher levels of blood glucose, triglycerides, total cholesterol and LDL cholesterol ($p < 0.001$). Additional CAD risk prediction ratios, such as total cholesterol/HDL, LDL/HDL and TG/HDL ratios that had grad-

ually increased from the normal to obese group, showed that obese subjects had a relatively higher risk of developing CVD than do those in the overweight and normal groups ($p < 0.001$).

Table 4 shows that the prevalence of components of MetS increased with obesity (increasing BMI).

Discussion

It is commonly acknowledged that the MetS significantly raises the chance of developing cancer, DMT2 and CVD. Obesity and insulin resistance are key elements of the MetS.[11, 12] The objective of the current study, which involved 120 participants, was to determine the prevalence of the MetS in obese individuals. Atherosclerosis, DMT2, hyperlipidaemia and hypertension are now recognised as typical illnesses associated with a sedentary lifestyle. Obesity is a frequent risk factor for several disorders. The key precursor of MetS is obesity, particularly visceral obesity, which has increased in incidence during the past ten years. Although MetS patients frequently have obesity, not all fat persons have MetS.[4, 13-16] On the other side, this condition can also manifest in lean individuals.[17] Additionally, it was shown that obese people are more likely to have MetS compared to overweight and normal subjects (63 %, 15 % and 2 %, respectively), supporting the findings of earlier researchers that though the obesity is a major component of MetS, all obese people may not have it and even lean person may develop this syndrome.

The results of the current investigation also showed that blood glucose and cholesterol levels are considerably impacted by obesity. In comparison to overweight and normal weight people, obese subjects exhibited considerably lower levels of HDL cholesterol and significantly higher levels of blood sugar, triglycerides, total cholesterol and LDL cholesterol. Additional CAD risk prediction ratios, including total cholesterol/HDL, LDL/HDL and TG/HDL ratio, showed that obese patients had a considerably higher chance of developing cardiovascular illnesses than do persons in the overweight and normal category. Adipose tissue produces more non-esterified fatty acids, glycerol, hormones, pro-inflammatory cytokines and other substances in obese people, which contribute to the emergence of insulin resistance. Failure to manage blood glucose levels leads to the development of DMT2 when insulin resistance is coupled with the malfunctioning of pancreatic islet cells, the cells that release insulin. This information is encouraging research into the disease's molecular and genetic roots as well as the development of fresh methods for treating and preventing it. Increased triglycerides, lower HDL levels and an aberrant LDL composition are the hallmarks of the main dyslipidaemia brought on by obesity. Without a doubt, the dyslipidaemia connected to obesity significantly contributes to the development of atherosclerosis and CVD in obese people. It has been shown that every aspect of dyslipidaemia, including greater triglyceride levels and lower HDL levels as seen in the current study, is atherogenic.

Obesity can accelerate the atherosclerotic process by affecting endothelial function and by activating oxidative stress-related pathways.[4, 16] TNF-α, resistin, leptin and adiponectin are only a few of the physiologically active chemicals that adipocytes generate, collectively known as adipokines.[18] These adipocytokines are produced in an uncontrolled manner as a result of adipocyte malfunction and they play a role in the pathophysiology of the MetS linked to obesity. For instance, increasing insulin resistance in obesity is accompanied with increased synthesis of TNF-α from stored fat.

It is generally known that individuals with the MetS phenotype experience oxidative stress more frequently than individuals without it,[4, 14-16] even if similar findings are not seen in all research.[17] Whether oxidative stress manifests at an early stage, before complications manifest, or whether it is merely a common result of cell damage, reflecting the presence of complications, will determine whether oxidative stress is a cause or a consequence in complications associated with the MetS (probably, both). Some people think that oxidative stress is a result of MetS, which includes insulin resistance, hypertension and obesity,[4] while some contend that the MetS, which includes insulin resistance, hypertension, atherosclerosis, obesity and more, is brought on by oxidative stress.[16, 19-21] The problem is made worse by a smoking habit, an urban lifestyle, an atherogenic diet and a lack of exercise as primary dyslipidaemia associated with antioxidant depletion was observed in subjects with urban background and smokers.

Conclusion

Obesity is one of the key elements of MetS, which is a collection of clinical and metabolic disorders including abdominal obesity, insulin resistance, hypertension and dyslipidaemia, all of which raise the risk of CVD and DMT2. In the MetS, excessive fat storage promotes the formation of free radicals and oxidative damage in adipose tissue. Free radicals can play a substantial role in the onset and advancement of numerous disease processes, including CVD, cerebrovascular accidents and diabetes complications.

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Conflict of interest

None.

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