ASSOCIATION BETWEEN VISCERAL FAT AND METABOLIC VARIABLES. THE METABOLIC SYNDROME

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Abstract

Obesity is an increasing health problem worldwide. Visceral fat has been associated with a higher risk of metabolic syndrome. The co-occurrence of a cluster of factors is constantly increasing, making this syndrome the leading cause of cardiovascular diseases and diabetes type 2. The present investigation showed that visceral fat in overweight and obese subjects with metabolic syndrome has a high correlation with HOMA, triacylglycerol, increased wait hip ratio and several other metabolic variables. The only parameter that did not differ among the three groups of subjects was the level of cholesterol.

INTRODUCTION

Obesity is a condition of excess body fat that results from increased energy intake relative to energy expenditure. It is to a great extent, genetically determined and is strongly influenced by fatty food and a sedentary lifestyle. A central body distribution of fat, referred to as android obesity determined by the waist to hip ratio has together with the several factors including among many as hypertension, hypertriglyceridemia, decreased glucose tolerance been associated with Metabolic syndrome. The mean dominator for this syndrome being insulin resistance. This cluster of risk factor is responsible for much of the excess cardiovascular disease morbidity among overweight and obese patients and those persons with type 2 diabetes mellitus. There are several theories about the development of metabolic syndrome. The traditional view has been that obesity is the underlying factor responsible for the metabolic syndrome. The portal theory of visceral fat being more metabolically active and its close localization to the portal vein, its high lipolytic activity and increased blood supply has a central role and is well accepted. More recently studies has included knew knowledge about different risk factors and proposes other theories explaining the metabolic syndrome such as insulin resistance, stress hormones, lifestyle and diet.

The treatment of metabolic syndrome is mainly non-pharmacological as lifestyle interventions, physical activity and low calorie diets. The main goal being decreased energy intake and increased energy expenditure. As fat metabolism is used in an increasing amount during sub maximal exercise, which is 50% of VO2 max, this combined with the right dietary interventions has been proved to be highly effective in the treatment of obesity and therefore also metabolic syndrome.

OBESITY

Obesity is a risk factor for development of cardiovascular disease, hypertension, gallbladder disease, diabetes mellitus and certain form of cancer and the prevalence are rapidly increasing worldwide. Obesity should not be defined by body weight alone, as muscular individuals may be overweight by arbitrary standards without having increased adiposity. The most widely used method to assess obesity is the body mass index (BMI), which is equal to weight/height^2 in kg/m^2. A BMI between 20 and 25 kg/m^2 is considered an appropriate for most individuals. Overweight is defined as a BMI>25-27 kg/m^2 and obesity is defined as a BMI > 30 kg/m^2.

Many treatment strategies have been used among obese people to loose weight. Most common treatment involves a low-energy diet to decrease energy intake and promote weight reduction. However when energy intake is restricted, energy expenditure decreases, resulting in a decrease in the rate of weight loss with time. This decrease includes reduction in resting metabolism due to losses of fat-free mass and decreases in postprandial energy expenditure because of smaller meals. A treatment that prevents these phenomena would be advantageous. Exercise is capable of altering energy intake and energy expenditure and body composition. Thus, it is an important variable to consider in understanding obesity development and treatment.

ETHIOPATHOGENESIS OF OBESITY

There are many associations between obesity and an unfavourable lipid profile. Lipid abnormalities related to obesity include an elevated serum concentration of cholesterol, low-density-lipoprotein (LDL) cholesterol, very low density lipoprotein (VLDL) cholesterol, triglycerides and apolipoprotein B, as well as a reduction in serum high-density-lipoprotein

(HDL) cholesterol. The mechanisms underlying this dyslipidaemia are not fully understood but involve the combination of insulin resistance and hyperinsulinaemia stimulating hepatic triglyceride synthesis from an increased adipose tissue undergoing enhanced lipolysis. This leads to postprandial hypertrigyceridaemia, smaller and denser LDL particles, and reduced HDL cholesterol concentrations.

Obesity is associated with increased risk of coronary artery disease, heart failure and atrial fibrillation. As far as the coronary artery disease is concerned, it is associated with both fatty streaks and raised atherosclerotic lesions in the right coronary and left anterior descending coronary arteries in young men, although not in women (S.D.H. Malnick et al). Individuals with obesity have a form of cardiomyopathy attributed to chronic volume overload, characterized by left ventricular dilatation, increased left ventricular wall stress and compensatory left ventricular hypertrophy. Most studies have reported abnormal diastolic function without abnormal systolic function.

METABOLIC SYNDROME

Metabolic syndrome, also called insulin resistance syndrome or Syndrome X, is a cluster of risk factors that is responsible for much of the excess cardiovascular disease morbidity among overweight and obese patients and those persons with type 2 diabetes mellitus. There are differences in body-fat distribution (i.e., gynecoid versus android) associated with an altered metabolic profile. Each component of the syndrome has been associated with an increased risk of cardiovascular disease.

The major characteristics of metabolic syndrome include a co-occurrence of:

- Insulin resistance,
- Abdominal obesity
- Elevated blood pressure
- Lipid abnormalities (i.e., elevated levels of triglycerides and low levels of high-density lipoprotein [HDL] cholesterol).

Metabolic syndrome is associated with a proinflammatory/prothrombotic state that may include elevated levels of C-reactive protein and TNFa, endothelial dysfunction, hyperfibrinogenemia, increased platelet aggregation, increased levels of plasminogen activator inhibitor 1, elevated uric acid levels, microalbuminuria, and a shift toward small, dense particles of low-density lipoprotein (LDL) cholesterol.

The etiology of the metabolic syndrome has not been established definitively. One hypothesis resumes that the primary cause is insulin resistance, and once an individual develops insulin resistance they will then develop the other characteristics of the syndrome if they have the genetic predisposition. Insulin resistance correlates with visceral fat measured by waist circumference or waist to hip ratio. The link between insulin resistance and cardiovascular disease probably is mediated by oxidative stress, which produces endothelial cell dysfunction, promoting vascular damage and atheroma formation. (Lopez-Candalez A et al)

The second hypothesis blames hormonal changes for the development of abdominal obesity. One study demonstrated that persons with elevated levels of serum cortisol (caused by chronic stress) developed abdominal obesity, insulin resistance, and lipid abnormalities. The investigators concluded that this inappropriate activation of the hypothalamic-pituitary-adrenal axis by stress is responsible for the link between psychosocial and economic problems, and acute myocardial infarction. (Bjørntorp P et al)

BODY DISTRIBUTION OF FAT

Fat is not uniformly distributed in the body. The majority of adipose depot is subcutaneous (about 80% of all body fat). In the obese and non-obese states there are characteristic gender differences in the distribution of subcutaneous fat. Non-obese women have relatively more subcutaneous fat in the gluteofemoral area than in other subcutaneous regions, whereas in non-obese men the subcutaneous fat is distributed in a uniform fashion.
The general differences are further pronounced in obesity. Obese men usually accumulate fat in the subcutaneous abdominal area. (C.Bouchard et al). This male obesity is called central, upperbody, android or ‘apple-shaped’ obesity.

Obese woman usually accumulate subcutaneous fat in the lower part of the abdominal wall and the gluteofemoral region. This female type of obesity is called peripheral, gynoid or 'pear-shaped' obesity. The link between gender and regional obesity is not absolute. There are many obese men who have peripheral fat distribution, and the other way around.

- Female: Gynoid, "pear-shaped" or Peripheral
- Male: Android, "Apple-shaped, upperbody or Central.
- Non-Obese women: Gluteofemoral distribution

The metabolic activity is lowest in the subcutaneous gluteofemoral area, followed by the abdominal subcutaneous area, and highest in the visceral region. These regional differences might be of physiological importance. Only the visceral fat has direct access to the liver by the portal vein. In situations where there is a need for rapid energy supply, such as during physical activity, more fat is mobilized from the visceral fat in comparison to the other regions in relative terms. #6 Some fat depots might be reserved for utilization during a particular situation. For example, the metabolic activity of gluteofemoral fat is activated in lactating mothers. The mechanisms behind regional differences in metabolism are partly elucidated, at least as regards lipid mobilization through lipolysis in fat cells. The lipolytic hormones (i.e. catecholamines) are most active in the visceral fat, followed by abdominal subcutaneous fat, which, in turn, is more active than gluteofemoral subcutaneous fat. The anti-lipolytic hormones and parahormones (i.e. insulin, prostaglandins and adenosine) have a more pronounced inhibitory effect on lipolysis in subcutaneous compared to visceral fat cells. When the hormone effects are taken together it appears that elevation of the concentration of any of the lipolysis-regulating hormones will result in a more rapid lipid mobilization from visceral as compared to subcutaneous fat .

EFFECTS OF VISCERAL OBESITY-"THE PORTAL THEORY"

The mechanisms linking visceral fat to metabolic and cardiovascular disorders are not elucidated. (Bjørntorp et al 1994) A common theory is that visceral obesity leads to accelerated mobilization of fatty acids to the portal system because of increased rate of

lipolysis in visceral fat cells in combination with an enlargement of the visceral fat depot (mass effect). Elevated ‘portal’ free fatty acids concentrations can have a number of undesirable effects on the liver. Many of the adverse metabolic consequences of obesity could be mediated through increased delivery of non-esterified fatty acids (NEFA) to the liver. These would include excessive VLDL-triacylglycerol secretion, stimulation of hepatic glucose production and impaired hepatic insulin clearance (hence, systemic hyperinsulinaemia, dyslipidemia and glucose intolerance).

Direct studies of visceral fat cells in upper-obese subjects have demonstrated a number of abnormalities in the hormonal regulation of lipolysis. These changes are, above all located at the level of receptor–signal transduction. Decreased functions off insulin receptors and alpha2-adrenoceptors plus increased action of beta3-adrenoceptors have been demonstrated in visceral fat cells of upper-body obese subjects.

OBJECTIVE THEORIES

Several theories has been proposed to object the ”portal theory” of the danger of visceral fat due to its close localization to portal vein and higher blood flow in visceral fat than in subcutaneous. First, it is important to consider carefully, the idea that omental fat (whose lipolytic products are liberated into the portal vein) is more lipolytically active than subcutaneous fat. If this fat depot is consistently exporting fatty acids at a high rate, how is it supposed that it ever `accumulated' in the first place? It is believe that the only consistent interpretation is that this depot has a high rate of lipid turnover, with a high lipolytic capacity in times of stress or fasting, matched or exceeded at other times by a high capacity for fatty acid uptake and storage. Fatty acid uptake is likely to involve the lipoprotein lipase pathway. In that case, omental fat could be envisaged as protecting the liver from an influx of triacylglycerol- fatty acids in the postprandial period. A direct test of this requires measurement of NEFA or glycerol concentrations in the hepatic portal vein. Such measurements have appeared from time to time in the literature. Hagenfeldt et al took samples of hepatic portal blood at cholecystectomy in five subjects. The mean arterial and portal vein NEFA concentrations were 561 and 580 mmol/l, respectively, and the authors concluded that the release of NEFA from omental adipose tissue was `of minor importance' (although

surgical stress is one condition in which, on the above arguments, augmented omental lipolysis might be expected).

For comparison, in studies looking at the venous drainage from subcutaneous abdominal adipose tissue, typical NEFA concentrations after overnight fast are: arterial 595, adipose venous 1284 mmol/l, although, of course, blood flow is less in subcutaneous adipose tissue, than through the portal vein. Such direct and indirect tests of the portal theory as are available are, therefore, far from conclusive. They do not obviously support the idea of a large additional influx of fatty acids into the portal vein from visceral fat.

Concluding that visceral fat, even though due to its close localization to the portal vein, higher lipolytic activity and higher blood flow than subcutaneous fat, does not cause higher concentrations of non esterified fatty acids (NEFA) in portal blood than what can be measured in arterial blood. It is from these theories therefore difficult to conclude that that visceral fat is more dangerous than the subcutaneous fat.

Another hypothesis that doesn't find the release of free fatty acids from deep abdominal fat stores to be the true mediator of the disease, propose that it is the combination of physical inactivity with high-fat, refined sugar diet consumed in the industrialized countries that is the major underlying cause in genetically susceptible individuals. (R.James Barnard et al)

Further support for the suggestion that abdominal fat is not the real underlying factor for the metabolic syndrome comes from the intervention studies, which have shown that most of the metabolic factors (including hyperinsulinemia, hypertriglycideremia and hypertension) can be significantly reduced with little or no change in bodyweight. (R.James Barnard et al) Believing that diet has a more significant role.

TREATMENT OF OBESITY

Dietary interventions

Dietary interventions for obesity are designed to create a negative energy balance (i.e., calories ingested < calories expended) by reducing daily energy intake below energy requirements. Energy requirements vary by sex, weight, and level of physical activity such that men, heavier individuals, and more active individuals have greater

energy needs. However, greater energy deficits result in greater weight losses.

A low calorie diet (LCD) is designed to create an energy deficit of 500–1,000 kcal/day and induce a weight loss of 0.5–1 kg/week Careful self-monitoring of calorie intake is crucial to the success of LCDs. Obese individuals underestimate their intake by ~30–50%. Thus, patients must be instructed in reading food intake below energy requirements. .

The more self-monitoring records patients complete each week, the more weight they lose. There are several options for facilitating adherence to an LCD, including the use of structured meal plans. Finding suggests that the provision of structure induces greater weight loss than does behaviour therapy alone with a self-selected diet. Indicating that providing detailed menus is sufficient to structure patients' dietary adherence.

Specific dietary changes that are appropriate for addressing different aspects of the syndrome include reducing saturated fat intake to lower insulin resistance, reducing sodium intake to lower blood pressure, and reducing high-glycemic–index carbohydrate intake to lower triglyceride levels. A diet that includes more fruits, vegetables, whole grains, monounsaturated fats, and low-fat dairy products will benefit most patients with metabolic syndrome.

Physical activity

Physical activity is the second component of lifestyle modification. The benefits of physical activity include inducing negative energy balance (by increasing calorie expenditure), sparing fat-free mass during weight loss, and improving cardiovascular fitness. Physical activity, however, produces minimal weight loss in the absence of caloric restriction.

The greatest benefit of physical activity is in facilitating the maintenance of weight loss. Case studies have shown that people who exercise regularly are more successful in maintaining weight losses than are those who do not exercise. Additional evidence comes from randomised trials. Participants who receive diet plus exercise maintain greater weight losses 1 year after treatment than do those who receive diet alone, although the differences are not always statistically significant. Physical activity can be divided into two types: programmed

and lifestyle. Programmed activity is typically planned, aerobic, and completed in a single bout (e.g., walking, biking, aerobics classes). Lifestyle activity involves increasing energy expenditure throughout the day by methods such as using stairs rather than escalators or choosing a distant parking spot.

EFFECT OF EXERCISE ON FAT METABOLISM

Exercise is frequently advocated in treatment of obesity as a means of increasing energy expenditure and potentially countertracing the negative effects of dietary restriction. Aerobic exercise, in particular, is advantageous because it may preserve fat-free mass and increases fat oxidation, thereby helping to maintain the Resting metabolic rate (RMR) during weight reduction, so the main task is to reduce adipose tissue mass while preserving lean body mass. A clinically drop in body fat will require long-term exercise participation. Important things to consider when evaluating the effect of exercise are the length of the study and the type, intensity and duration.

Carbohydrates and fat are the main energy sources during physical activity. Energy used depends on the intensity (% of VO_2 max) and the duration of the exercise. Lipids in form of Free fatty acids (FFA), one of the primary energy sources at rest, are oxidized in progressively increasing amount as total energy expenditure increases with exercise. FFA remains a significant fuel source during sub maximal steady state exercise (<50-60% VO_2 max). At high intensity work (greater than 70% of VO_2 max), fat is used in decreasing amount for energy expenditure and glycogen becomes the predominantly energy source (T.Eriksen 2002). These facts are important to consider when physical activity is prescribed for treatment of obesity.

The oxidation of FFA is determined by the availability of FFA and the capacity of tissues to oxidize FFA. Both cardiac and skeletal muscle is metabolically oxidative. However mitochondria are few in number in fast-twitch glycolytic muscle fibers. It is the slow-twitch oxidative muscle fibers that are responsible for most skeletal muscle oxidation of FFA. Slow-twitch fibers have a large number of mitochondria, are well vascularized, contain myoglobin and are primarily activated during exercise of moderate intensity. The relatively amount of

fast-twitch versus slow-twitch muscle fiber contributes to an individuals ability to utilize fat metabolism (T.Eriksen 2002)

Endurance training enhances the capacity of skeletal muscle to utilize FFA as fuel. Thus, if food consumption remains constant, regular long-term exercise would be expected to diminish fat stores and loose weight.

RQ (the volume of carbon dioxide expired per minute divided by the volume of oxygen consumed during the same time interval is referred to as the respiratory quotient) tends to decrease overtime with prolonged aerobic exercise, reflecting increased fat utilization.

Behaviour therapy

Behaviour therapy provides patients a set of principles and techniques to facilitate their adherence to the diet and activity goals described above. Common techniques include self-monitoring (of food and activity), stimulus control, slowing eating, cognitive restructuring, problem solving, and relapse prevention. Behaviour therapy typically is delivered to groups of 10–20 participants in 60- to 90-minute sessions for 20–26 weeks. Several reviews have shown that patients lose 9–10% of their starting weight28–30 but regain approximately one-third of the lost weight in the year following treatment.31 Perri et al.32, 33 have shown that continued patientprovider contact following treatment, in person or by mail, significantly improves the maintenance of weight loss. Longterm treatment recognizes that obesity is a chronic condition similar to hypertension or diabetes.

Pharmacological Interventions

As BMI or disease risk increase, more intensive options are available for the treatment of obesity. Pharmacotherapy is recommended for individuals with a BMI ≥30 kg/m2 or with a BMI ≥27 kg/m2 in the presence of two or more obesity-related co morbidities (e.g., coronary heart disease, type 2 diabetes, or sleep apnoea) and who cannot lose weight satisfactorily with more conservative approaches. Metformin, Sibutramine and Orlistat
are approved by the Food and Drug Administration for the induction and maintenance of weight loss. Metformin improves insulin resistance, and is the most widely used drug for this purpose. Sibutramine is a combined serotonin- norepinephrine reuptake inhibitor that is associated with reports of increased satiation (i.e., fullness). Orlistat is a gastric lipase

inhibitor that blocks the absorption of about one third of the fat contained in a meal, leading to the loss of about 150–180 kcal/day. Patients are negatively reinforced to eat a low-fat diet because the consumption of more than 20 g of fat per meal, or 70 g of fat per day, can induce adverse gastrointestinal events that include oily stools, flatus with discharge, and faecal urgency. (Anthony N. Fabricatore et al)

Patients suspected of having metabolic syndrome should have a fasting glucose level and a fasting lipid profile level obtained. A euglycemic clamp or homeostasis model assessment is used in research studies to accurately assess insulin resistance, but is impractical for use in the clinical setting. Fasting insulin levels and glucose challenge tests are indicators of insulin resistance but do not need to be measured in most situations because a fasting glucose level alone suffices for the definition of metabolic syndrome. (Darween Deen et al)

SUBJECTS AND METHOD

Sixty-three women in different age groups from 21 to 66 years with BMI ranging from 17.27 to 48.47 participated in the study. According to their BMI and presence of metabolic syndrome or not, various metabolic parameters were measured. Visceral fat divided on subcutaneous fat (VF/VS) gives us a more precise measure of the visceral fat in relation to total body fat. This main parameter correlated to the other variables is what this paper mainly focuses on.

The subjects were divided into three groups according to Obesity with or without metabolic syndrome. Here we define obesity as BMI >25. BMI>25 is defined as overweight, and BMI>30 obesity. But in this interpretation we have chosen to use obesity as BMI>25 to better see a correlation between the variables and visceral fat as visceral fat can be observed in a significant amount in overweight as well as in obese.

Division of groups:

1. Non-Obese (BM<25)
2. Obese and overweight without metabolic syndrome (BMI>25)
3. Obese and overweight with metabolic syndrome (BMI>25)

Subject number 59 was Non-Obese with metabolic syndrome. Since there was only one subject in this group it was not possible to calculate with this data. In a larger study with far more participants one can include such a deviant variable if there would be more subjects in this group. As far as to this study we had to exclude this subject. One can encounter these types of deviations in the population, and it should be something to be aware of, even though metabolic syndrome rarely is seen without obesity, but we couldn't use it in this type of statistical data. Therefore our group consisted of together 62 subjects.

Two parameters: HOMA and QUIKI are being compared with relative area of visceral fat/subcutaneous fat (VF/VS). HOMA is defined as Homeostasis model assessment. Which estimates steady state beta cell function (%B) and insulin sensitivity (%S).
QUICKI is a parameter of glucose/insulin. If QUICKI is high, than the value of insulin resistance is decreasing. The opposite can be seen for HOMA.

Pearson test was used to see if any correlation existed between different parameters. $P< 0.05$ was considered statistically significant. But in cases of higher value of significance we use $P<0.01$. Correlations were done with both Pearson and Spearman. It is useful to use both because Pearson can only do a linear aggregation, meaning that one deviation outside the mean range is not detected by this method. Spearman is a non-linear graph that uses monotonicity. Values outside the mean graph are better included.

Statistical analysis

The task was to:
1) Compare the HOMA or QUICKI indices of insulin resistance between groups, and to compare the level of Triglycerides (TG) and High density lipoproteins (HDL).
2) Compare the area of visceral fat divided on subcutaneous fat (VF/SF) between the groups.
3) Make a correlation between (VF/SF) and HOMA or QUICKI and other indices.
The subjects in this study were divided into three groups according to their BMI and presence of metabolic syndrome or not. (Without metabolic syndrome, wo/MS)

Metabolic parameters	**1.Non-Obese**	**2.Obese and overweight wo/MS**	**3.Obese and overweight w/MS**
VF/SF	0.230(+)	0.280(#)	0.371
TG	0.772(+)	1.172(#)	2.327
HDL	1.549(+)	1.411(#)	1.267
Cholesterol	4.672	4.773	4.682
HOMA	0.943(*)(+)	2.006(#)	3.547
QUICKI	0.396	0.353	0.322

(MS=Metabolic syndrome)

*Difference between group 1 and 2

+Difference between group 1 and 3

#Difference between group 2 and 3

Evaluation of the VF/VS between the groups

There was no significant difference between group 1 and 2. But a significant difference between group 3 and the other two groups. Which is what was expected. Obese and overweight with metabolic syndrome has a higher significant correlation to visceral fat than the other two groups. Part of ANOVA analysis showed 0.001 significance between the groups. But in order to see which group that differed it was essential to use DUNCAN analysis for multiple comparison.

Evaluation of the Triglyceride (TG) level between the groups

Group1 and 2 did not differ in the level of Triacylglycerol. Group 3. Obese with metabolic syndrome has a significant higher TG level than the other groups.

Evaluation of the High density lipoprotein (HDL) level between the groups

There were no differences between group 1 and 2, or between group 2 and 3. But between non-obese and obese and overweight without metabolic syndrome; group 1 and 3 respectively, there were a significant difference.

Evaluation of the Cholesterol level between the groups

This parameter did not differ between in any of the subjects in the group. As seen with the DUNCAN analysis. All the subjects were in the same group, not divided into two groups as the other parameters were. Meaning that there were no differences between the groups.

Evaluation of HOMA and QUICKI between the groups

ANOVA analysis showed that there was a significant difference between the groups. Group 3 Obese and overweight with metabolic syndrome showed the highest difference in HOMA compared to the other groups. QUICKI did not show very high difference between the groups. Group 1 and 3 was the once that differed the most. Meaning that Group 1 Non-obese differed more from the Obese and overweight whether they had metabolic syndrome or not.

3) Correlation between VF/SF and HOMA and QUICKI and other indices.

Correlations

		VF_SF	HOMA	QUICKI	WHR	HDL	Glycemia	ins	cholesterol	TG
VF_SF	Pearson Correlation	1	,382(**)	-,375(**)	,410(**)	-,196	,410(**)	,241	,050	,352(**)
	Sig. (2-tailed)		,002	,003	,001	,134	,001	,064	,707	,006
	N	61	61	60	61	60	60	60	60	60

*P<0.05 **P<0.01

There was a significant correlation between VF/SF and most of these parameters. HOMA P<0.001 and QUICKI P<0.001. The only parameter that didn't correlate with VF/SF was cholesterol (sig = 0.707) Meaning that it is a 70.7% chance that we are wrong. The correlation between VF/SF and HDL was also week, sig =13.4%, which means a higher prosent of mistake than 10% (P<0.001), telling us that it has a weak correlation to VF/SF.

DISCUSSION

There were 62 women participating in this study, divided into three groups. After measuring all the parameters, comparing them between the groups and correlating the relative area of visceral fat divided on subcutaneous fat (VF/SF). We have found that there is a significant

correlation between (VF/SF) and HOMA and QUICKI. The significance of the different parameters was higher in the group of subjects that were obese with metabolic syndrome, telling us what we expected. The visceral fat is significant in metabolic syndrome, and has a high correlation with the other parameters which is increased in this syndrome. This conclusion is in correlation with the article of P. Arner. Regional adipocity in man, and the article from Hanh-Binh Nguyen-Duy et al. Visceral fat and liver fat are independent predictors of metabolic risk factors in men.

CONCLUSION

1. We have found differences in the amount of visceral fat in the three groups of subjects.

2. We have found differences in the metabolic variables (HOMA, TG, HDL) between the three groups.

3. In the entire group we found correlations between the relative amount of visceral fat and HOMA, QUICKI, TG, HDL.

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