

**FREQUENCY OF METABOLIC SYNDROME IN TYPE 2 DIABETES MELLITUS IN
MEDICAL DIABETIC CENTRE -MEDANI-GEZIRA STATE-SUDAN- FEBRUARY 2015****Dr. Yasir Abdalla Hakim* and Assad Adamabbas**

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ABSTRACT

Background: Metabolic syndrome is a disorder of energy utilization and storage, diagnosed by a co-occurrence of three out of five of the following medical conditions: abdominal (central) obesity, elevated blood pressure, elevated fasting plasma glucose, high serum triglycerides, and low high-density cholesterol levels. This study was to review the prevalence of the metabolic syndrome in Sudanese type 2 diabetes mellitus. **Methods:** The study included 80 Sudanese patients; half of them with type 2 diabetes mellitus (cases) and the rest are non-diabetics (control group). Cases and controls blood pressure, abdominal circumference and body mass index were reported. Venous blood samples were taken from all after at least 10 hours fasting for determination of serum level of cholesterol, triglycerides, high density lipoprotein and low density lipoprotein. Glucose level and HbA1c were also measured. Cases and controls were compared in all measures above looking for the differences. The metabolic syndrome was diagnosed according to criteria of national cholesterol education program. **Results:** in cases fasting blood glucose, waist circumference, serum triglyceride, high density lipoprotein, blood pressure, total cholesterol were found to be 87.5% (35 cases), 130% (52 cases), 50 % (20 cases), 64.5% (28 cases), 62.5% (25 cases), 42.5% (17 cases) respectively. In control group, fasting blood glucose 100% (40 cases), waist circumference 70% (27 cases), serum triglyceride, high density lipoprotein, blood pressure, total cholesterol..... were found to be 40% (16 cases), respectively. **Conclusion:** In this study the frequency of the metabolic syndrome was found significantly high, especially in women between 40 to 60 years. I highly recommended an urgent need to commence screening for this syndrome based on presence of one or two components of metabolic syndrome.

KEYWORDS: Metabolic syndrome; Diabetes mellitus; Prevalence; Sudanese.**INTRODUCTION**

Metabolic syndrome is a disorder of energy utilization and storage, diagnosed by a co-occurrence of three out of five of the following medical conditions: abdominal (central) obesity, elevated blood pressure, elevated fasting plasma glucose, high serum triglycerides, and low high-density cholesterol (HDL) levels. Metabolic syndrome increases the risk of developing cardiovascular disease, particularly heart failure, and diabetes.^[1] Some studies have shown the prevalence in the USA to be an estimated 34% of the adult population,^[2] and the prevalence increases with age.

Metabolic syndrome is also known as metabolic syndrome X, cardiometabolic syndrome, syndrome X, insulin resistance syndrome, Reaven's syndrome (named for Gerald Reaven), and CHAOS (in Australia).^[3]

Metabolic syndrome and prediabetes appear to be the same disorder, just diagnosed by a different set of biomarkers.

Signs and symptoms

The principal symptom of metabolic syndrome is central obesity (also known as visceral, male-pattern or apple-shaped adiposity), overweight with adipose tissue accumulation mainly around the waist and trunk.^[4]

Other signs of metabolic syndrome include: High blood pressure, decreased fasting serum HDL cholesterol, elevated fasting serum triglyceride level (VLDL triglyceride), impaired fasting glucose, insulin resistance, or prediabetes.

Associated conditions include: hyperuricemia, fatty liver (especially in concurrent obesity) progressing to NAFLD, polycystic ovarian syndrome (in women), erectile dysfunction (in men), and acanthosis nigricans.

Cause

The exact mechanisms of the complex pathways of metabolic syndrome are under investigation. The pathophysiology is very complex and has been only partially elucidated. Most patients are older, obese,

sedentary, and have a degree of insulin resistance. Stress can also be a contributing factor. The most important factors are genetics,^[5,6,7,8] aging, diet (particularly sugar-sweetened beverage consumption),^[9] sedentary behavior^[10] or low physical activity,^[11,12] disrupted chronobiology/sleep,^[13] mood disorders/psychotropic medication use,^[14,15] and excessive alcohol use.^[16] There is debate regarding whether obesity or insulin resistance is the cause of the metabolic syndrome or if they are consequences of a more far-reaching metabolic derangement. A number of markers of systemic inflammation, including C-reactive protein, are often increased, as are fibrinogen, interleukin 6, tumor necrosis factor-alpha (TNF α), and others. Some have pointed to a variety of causes, including increased uric acid levels caused by dietary fructose.^[17,18,19]

It is generally accepted that the current food environment contributes to the development of metabolic syndrome: our diet is mismatched with our biochemistry.^[20] Weight gain is associated with metabolic syndrome. Rather than total adiposity, the core clinical component of the syndrome is visceral and/or ectopic fat (i.e., fat in organs not designed for fat storage) whereas the principal metabolic abnormality is insulin resistance. The continuous provision of energy via dietary carbohydrate, lipid, and protein fuels, unmatched by physical activity/energy demand, arguably creates a backlog of the products of mitochondrial oxidation, a process associated with progressive mitochondrial dysfunction and insulin resistance.

Stress

Recent research indicates prolonged chronic stress can contribute to metabolic syndrome by disrupting the hormonal balance of the hypothalamic-pituitary-adrenal axis (HPA-axis).^[21] A dysfunctional HPA-axis causes high cortisol levels to circulate, which results in raising glucose and insulin levels, which in turn cause insulin-mediated effects on adipose tissue, ultimately promoting visceral adiposity, insulin resistance, dyslipidemia and hypertension, with direct effects on the bone, causing "low turnover" osteoporosis.^[22] HPA-axis dysfunction may explain the reported risk indication of abdominal obesity to cardiovascular disease (CVD), type 2 diabetes and stroke.^[23] Psychosocial stress is also linked to heart disease.^[24]

Overweight and obesity

Central obesity is a key feature of the syndrome, reflecting the fact that the syndrome's prevalence is driven by the strong relationship between waist circumference and increasing adiposity. However, despite the importance of obesity, patients who are of normal weight may also be insulin-resistant and have the syndrome.^[25]

Sedentary lifestyle

Physical inactivity is a predictor of CVD events and related mortality. Many components of metabolic

syndrome are associated with a sedentary lifestyle, including increased adipose tissue (predominantly central); reduced HDL cholesterol; and a trend toward increased triglycerides, blood pressure, and glucose in the genetically susceptible. Compared with individuals who watched television or videos or used their computers for less than one hour daily, those who carried out these behaviors for greater than four hours daily have a twofold increased risk of metabolic syndrome.^[25]

Aging

Metabolic syndrome affects 44% of the U.S. population older than age 50. With respect to that demographic, the percentage of women having the syndrome is higher than that of men. The age dependency of the syndrome's prevalence is seen in most populations around the world.^[25]

Diabetes mellitus type 2

The metabolic syndrome quintuples the risk of type 2 diabetes mellitus. Type 2 diabetes is considered a complication of metabolic syndrome. In people with impaired glucose tolerance or impaired fasting glucose, presence of metabolic syndrome doubles the risk of developing type 2 diabetes.^[26] It is likely that prediabetes and metabolic syndrome denote the same disorder, defining it by the different sets of biological markers. The presence of metabolic syndrome is associated with a higher prevalence of CVD than found in patients with type 2 diabetes or IGT without the syndrome.^[25] Hypoadiponectinemia has been shown to increase insulin resistance,^[27] and is considered to be a risk factor for developing metabolic syndrome.^[28]

Coronary heart disease

The approximate prevalence of the metabolic syndrome in patients with coronary heart disease (CHD) is 50%, with a prevalence of 37% in patients with premature coronary artery disease (age 45), particularly in women. With appropriate cardiac rehabilitation and changes in lifestyle (e.g., nutrition, physical activity, weight reduction, and, in some cases, drugs), the prevalence of the syndrome can be reduced.^[25]

Lipodystrophy

Lipodystrophic disorders in general are associated with metabolic syndrome. Both genetic (e.g., Berardinelli-Seip congenital lipodystrophy, Dunnigan familial partial lipodystrophy) and acquired (e.g., HIV-related lipodystrophy in patients treated with highly active antiretroviral therapy) forms of lipodystrophy may give rise to severe insulin resistance and many of metabolic syndrome's components.^[25]

Schizophrenia and other psychiatric illnesses

Patients with schizophrenia, schizoaffective disorder or bipolar disorder may have a predisposition to metabolic syndrome that is exacerbated by sedentary lifestyle, poor dietary habits, possible limited access to care, and antipsychotic drug-induced adverse effects. It has been

found that 32% and 51%^[29] of individuals with schizophrenia meet criteria for metabolic syndrome; the prevalence is higher in women than in men.^[30]

Rheumatic diseases

There are new findings regarding the comorbidity associated with rheumatic diseases. Both psoriasis and psoriatic arthritis have been found to be associated with metabolic syndrome.^[31]

Pathophysiology

It is common for there to be a development of visceral fat, after which the adipocytes (fat cells) of the visceral fat increase plasma levels of TNF α and alter levels of a number of other substances (e.g., adiponectin, resistin, and PAI-1). TNF α has been shown not only to cause the production of inflammatory cytokines, but also possibly to trigger cell signaling by interaction with a TNF α receptor that may lead to insulin resistance.^[32] An experiment with rats fed a diet with 33% sucrose has been proposed as a model for the development of metabolic syndrome. The sucrose first elevated blood levels of triglycerides, which induced visceral fat and ultimately resulted in insulin resistance.^[33] The progression from visceral fat to increased TNF α to insulin resistance has some parallels to human development of metabolic syndrome. The increase in adipose tissue also increases the number of immune cells present within, which play a role in inflammation. Chronic inflammation contributes to an increased risk of hypertension, atherosclerosis and diabetes.^[34]

The central role of the cannabinoid system in the development of metabolic syndrome is indisputable. Endocannabinoid overproduction and dysbalance may exacerbate corticolimbic reward system dysfunction, and contribute to executive dysfunction (e.g., impaired delay discounting), perpetuating unhealthy behaviors. The brain is crucial in development of metabolic syndrome, modulating peripheral carbohydrate and lipid metabolism. Further, metabolic syndrome is a risk factor for neurological disorders.^[35]

The metabolic syndrome can be induced by overfeeding with sugar or fructose, particularly concomitantly with high-fat diet. The excessive fructose ingestion leads to increased metabolic burden to the liver (fructose is metabolized predominantly in the liver, whereas generally ~75% of postprandial glucose is disposed in the muscle).^[citation needed] The resulting short-chain fatty acid production, and general oversupply of n-6 fatty acids are important determinants of metabolic syndrome. In particular, arachidonic acid metabolism appears to be a factor in the pathogenesis of metabolic syndrome: arachidonic acid (with its precursor - linoleic acid) serve as a substrate to inflammatory factor production (prostaglandins and leukotrienes), whereas arachidonic acid-containing diacylglycerol (DAG) is a precursor to the endocannabinoid (2-arachidonoylglycerol) and is a by-product of fatty acid amide hydrolase (FAAH)-

mediated metabolism of anandamide (produced from N-arachidonoyl phosphatidylethanolamine).

Metabolomic studies suggest an excess of organic acids, impaired lipid oxidation byproducts, essential fatty acids and essential amino acids in the blood serum of affected patients. However, it is not entirely clear whether the accumulation of essential fatty acids and amino acids is the result of excessive ingestion or excess production by gut microbiota.

Prevention

Various strategies have been proposed to prevent the development of metabolic syndrome. These include increased physical activity (such as walking 30 minutes every day),^[36] and a healthy, reduced calorie diet.^[37] Many studies support the value of a healthy lifestyle as above. However, one study stated these potentially beneficial measures are effective in only a minority of people, primarily due to a lack of compliance with lifestyle and diet changes.^[11] The International Obesity Taskforce states that interventions on a sociopolitical level are required to reduce development of the metabolic syndrome in populations.^[38]

The Caerphilly Heart Disease Study followed 2,375 male subjects over 20 years and suggested the daily intake of a pint (~568 ml) of milk or equivalent dairy products more than halved the risk of metabolic syndrome.^[39] Some subsequent studies support the authors' findings, while others dispute them.^[40]

Diagnosis

A joint interim statement of the International Diabetes Federation Task Force on Epidemiology and Prevention; National Heart, Lung, and Blood Institute; American Heart Association; World Heart Federation; International Atherosclerosis Society; and International Association for the Study of Obesity published a guideline to harmonize the definition of the metabolic syndrome.^[41] This definition recognizes that the risk associated with a particular waist measurement will differ in different populations. Whether it is better at this time to set the level at which risk starts to increase or at which there is already substantially increased risk will be up to local decision-making groups. However, for international comparisons and to facilitate the etiology, it is critical that a commonly agreed-upon set of criteria be used worldwide, with agreed-upon cut points for different ethnic groups and sexes. There are many people in the world of mixed ethnicity, and in those cases, pragmatic decisions will have to be made.

The previous definitions of the metabolic syndrome by the International Diabetes Federation^[42] and the revised National Cholesterol Education Program are very similar and they identify individuals with a given set of symptoms as having metabolic syndrome. There are two differences, however: the IDF definition states that if body mass index (BMI) is greater than 30 kg/m², central

obesity can be assumed, and waist circumference does not need to be measured. However, this potentially excludes any subject without increased waist circumference if BMI is less than 30. Conversely, the NCEP definition indicates that metabolic syndrome can be diagnosed based on other criteria. Also, the IDF uses geography-specific cut points for waist circumference, while NCEP uses only one set of cut points for waist circumference regardless of geography. These two definitions are much more similar than the original NCEP and WHO definitions.

IDF

The International Diabetes Federation^[42] consensus worldwide definition of the metabolic syndrome (2006) is: Central obesity (defined as waist circumference[#] with ethnicity-specific values) AND any two of the following:

- Raised triglycerides: > 150 mg/dL (1.7 mmol/L), or specific treatment for this lipid abnormality
- Reduced HDL cholesterol: < 40 mg/dL (1.03 mmol/L) in males, < 50 mg/dL (1.29 mmol/L) in females, or specific treatment for this lipid abnormality
- Raised blood pressure (BP): systolic BP > 130 or diastolic BP > 85 mm Hg, or treatment of previously diagnosed hypertension
- Raised fasting plasma glucose (FPG): > 100 mg/dL (5.6 mmol/L), or previously diagnosed type 2 diabetes.

If FPG is > 5.6 mmol/L or 100 mg/dL, an oral glucose tolerance test is strongly recommended, but is not necessary to define presence of the syndrome.

[#] If BMI is > 30 kg/m², central obesity can be assumed and waist circumference does not need to be measured.

WHO

The World Health Organization 1999 criteria^[43] require the presence of any one of diabetes mellitus, impaired glucose tolerance, impaired fasting glucose or insulin resistance, AND two of the following:

- Blood pressure: $\geq 140/90$ mmHg
- Dyslipidemia: triglycerides (TG): ≥ 1.695 mmol/L and high-density lipoprotein cholesterol (HDL-C) ≤ 0.9 mmol/L (male), ≤ 1.0 mmol/L (female)
- Central obesity: waist:hip ratio > 0.90 (male); > 0.85 (female), or body mass index > 30 kg/m²
- Microalbuminuria: urinary albumin excretion ratio ≥ 20 μ g/min or albumin:creatinine ratio ≥ 30 mg/g

EGIR

The European Group for the Study of Insulin Resistance (1999) requires insulin resistance defined as the top 25% of the fasting insulin values among nondiabetic individuals AND two or more of the following:

- Central obesity: waist circumference ≥ 94 cm or 37 inches (male), ≥ 80 cm or 31.5 inches (female)
- Dyslipidemia: TG ≥ 2.0 mmol/L and/or HDL-C < 1.0 mmol/L or treated for dyslipidemia

- Hypertension: blood pressure $\geq 140/90$ mmHg or antihypertensive medication
- Fasting plasma glucose ≥ 6.1 mmol/L

NCEP

The US National Cholesterol Education Program Adult Treatment Panel III (2001) requires at least three of the following:^[44]

- Central obesity: waist circumference ≥ 102 cm or 40 inches (male), ≥ 88 cm or 35 inches (female)
- Dyslipidemia: TG ≥ 1.7 mmol/L (150 mg/dL)
- Dyslipidemia: HDL-C < 40 mg/dL (male), < 50 mg/dL (female)
- Blood pressure $\geq 130/85$ mmHg (or treated for hypertension)
- Fasting plasma glucose ≥ 6.1 mmol/L (110 mg/dl)

American Heart Association

There is confusion as to whether, in 2004, the AHA/NHLBI intended to create another set of guidelines or simply update the NCEP ATP III definition. According to Scott Grundy, University of Texas Southwestern Medical School, Dallas, Texas, the intent was just to update the NCEP ATP III definition and not create a new definition.^{[45][46]}

- Elevated waist circumference
 - Men — greater than 40 inches (102 cm)
 - Women — greater than 35 inches (88 cm)
- Elevated triglycerides: Equal to or greater than 150 mg/dL (1.7 mmol/L)
- Reduced HDL ("good") cholesterol:
 - Men — Less than 40 mg/dL (1.03 mmol/L)
 - Women — Less than 50 mg/dL (1.29 mmol/L)
- Elevated blood pressure: Equal to or greater than 130/85 mm Hg or use of medication for hypertension
- Elevated fasting glucose: Equal to or greater than 100 mg/dL (5.6 mmol/L) or use of medication for hyperglycemia

Other

High-sensitivity C-reactive protein has been developed and used as a marker to predict coronary vascular diseases in metabolic syndrome, and it was recently used as a predictor for nonalcoholic fatty liver disease (steatohepatitis) in correlation with serum markers that indicated lipid and glucose metabolism.^[47] Fatty liver disease and steatohepatitis can be considered as manifestations of metabolic syndrome, indicative of abnormal energy storage as fat in ectopic distribution. Reproductive disorders (such as polycystic ovary syndrome in women of reproductive age), and erectile dysfunction or decreased total testosterone (low testosterone-binding globulin) in men can be attributed to metabolic syndrome.^[41]

Management

The first line treatment is change of lifestyle (e.g., Dietary Guidelines for Americans and physical activity).

However, if in three to six months of efforts at remedying risk factors prove insufficient, then drug treatment is frequently required. Generally, the individual disorders that compose the metabolic syndrome are treated separately. Diuretics and ACE inhibitors may be used to treat hypertension. Cholesterol drugs may be used to lower LDL cholesterol and triglyceride levels, if they are elevated, and to raise HDL levels if they are low. Use of drugs that decrease insulin resistance, e.g., metformin and thiazolidinediones, is controversial; this treatment is not approved by the U.S. Food and Drug Administration.^[citation needed] Weight loss medications may result in weight loss.^[48] As obesity is often recognized as the culprit behind many of the additional symptoms, with weight loss and lifestyle changes in diet, physical activity, the need for other medications may diminish.

A 2003 study indicated cardiovascular exercise was therapeutic in approximately 31% of cases. The most probable benefit was to triglyceride levels, with 43% showing improvement; but fasting plasma glucose and insulin resistance of 91% of test subjects did not improve.^[11] Many other studies have supported the value of physical activity and dietary modifications to treat metabolic syndrome.

Restricting the overall dietary carbohydrate intake is more effective in reducing the most common symptoms of metabolic syndrome than the more commonly prescribed reduction in dietary fat intake.^[49] The combination preparation simvastatin/sitagliptin (marketed as Juvisync) was introduced in 2011 and the use of this drug was to lower LDL levels and as well as increase insulin levels.^[50] This drug could have been used to treat metabolic syndrome but was removed from the market by Merck in 2013 due to business reasons.^[51] High-dose statins, recommended to reduce cardiovascular risk, have been associated with higher progression to diabetes, particularly in patients with metabolic syndrome. The biological mechanisms are not entirely understood, however, the plausible explanation may lie in competitive inhibition of glucose transport via the solute carrier (SLC) family of transporters (specifically SLCO1B1), important in statin pharmacokinetics.

Controversy

The clinical value of using "metabolic syndrome" as a diagnosis previously has been debated due to different sets of conflicting and incomplete diagnostic criteria. These concerns have led the American Diabetes Association and the European Association for the Study of Diabetes to issue a joint statement identifying eight major concerns on the clinical utility of the metabolic syndrome diagnosis.^[61] The principal argument has been that when confounding factors such as obesity are accounted for, diagnosis of the metabolic syndrome has a negligible association with the risk of heart disease.^[62] Naturally, since the metabolic syndrome is a

disorder of energy distribution and storage, fat accumulation explains for a significant proportion of cardiovascular risk. However, obesity without metabolic syndrome does not confer a significant cardiovascular risk, whereas metabolic syndrome without obesity is associated with a significant risk of diabetes and cardiovascular disease. This association of metabolic syndrome with diabetes can be illustrated by generalized lipodystrophy (near complete absence of adipose tissue). The animals and humans with generalized lipodystrophy develop signs of metabolic syndrome in the absence of adipose tissue; and the metabolic syndrome progresses to diabetes, type 2. Adipose tissue transplantation in transgenic mice with lipodystrophy can cure the type 2 diabetes. It has not been contested that cardiovascular risk factors tend to cluster together; the matter of contention has been the assertion that the metabolic syndrome is anything more than the sum of its constituent parts. Phenotypic heterogeneity (for example, represented by variation in metabolic syndrome factor combinations among individuals with metabolic syndrome) has fueled that debate. However, more recent evidence suggests that common triggers (for example, excessive sugar-intake in the environment of overabundant food) can contribute to the development of multiple metabolic abnormalities at the same time, supporting the commonality of the energy utilization and storage pathways in metabolic syndrome.

MATERIALS AND METHODS

The study included 80 Sudanese patients; half of them with type 2 diabetes mellitus (cases) and the rest are non-diabetics (control group). Cases and controls blood pressure, abdominal circumference and body mass index were reported. Venous blood samples were taken from all after at least 10 hours fasting for determination of serum level of cholesterol, triglycerides, high density lipoprotein and low density lipoprotein. Glucose level and HbA1c were also measured. Cases and controls were compared in all measures above looking for the differences. The metabolic syndrome was diagnosed according to criteria of national cholesterol education program

RESULTS

The metabolic syndrome was diagnosed according to criteria of national cholesterol education program (NCEP). In this study, there was strong association between diabetes mellitus and metabolic syndrome which was occurred clearly in table (1,2,3,4) with correlation between Independent variable to dependent variable of all patients parameters with highly positively significant, that's expressed the body mass index, fasting blood glucose, blood pressure, HDL duration of DM, type of treatment and abdominal circumference In this study, the metabolic syndrome appeared well in most patients based in abdominal obesity 16 (40%) than the normal waist circumference in 23(60%) which was less than 120 cm in males, the abdominal obesity in females

36 (90%) compared to 5 (10%) with normal waist circumference less than 88 cm, elevated serum triglyceride more than 150 mg/dl in 26 (52%), poor high density lipoprotein in males 12(30%) less than 40 mg/dl, 23(57.5%) in females less than 50 mg/dl and elevated blood pressure 25(62.5%) of patients for more than 140/90. The majority of patients with metabolic syndrome increased in patients on oral hypoglycemic agents 34(85%) than the patients on insulin therapy 6(15%), the duration of diabetes mellitus for 5 to 10 years and fair glycemic control 32(80%)

The study showed that, the metabolic syndrome was high in type 2 diabetic patients particularly in females than

males compared to the normal group based on diagnosis according to criteria of national cholesterol education program. The study revealed that the metabolic syndrome was increased particularly in females, that's accounted as 22 females with metabolic syndrome than 7 females with no metabolic syndrome out of 60% compared to males and control group. In males the metabolic syndrome occurred in 4 patients and with no metabolic syndrome in 7 patients out of 40% compared to normal control group.

Overall of this study, Pearson correlation analyses showed that there was association Between type 2 diabetic patients and metabolic syndrome (fig 4,5,6,7).

Table 1: Pearson correlation analyses showed that there was association Between type 2 diabetic patients and metabolic syndrome with positively significant.

Independent variable	dependent variable	Sig	Correlation
Age	duration of diabetes	.000	.454**
	body mass index	.019	.262*
	Type of treatment	.001	.360**
	Blood pressure	.022	.256*
	Abdominal circumference	.411	.093
	total cholesterol	.290	.120
	low density lipoprotein	.876	.018
	high density lipoprotein	.030	.242*
	Triglyceride	.076	.200
	fasting blood glucose	.000	.513**
	HbA1C	.000	.409**

**Correlation is significant at the 0.01 level (2-tailed).

*Correlation is significant at the 0.05 level (2-tailed).

Table 2: Pearson correlation analyses showed that there was association Between type 2 diabetic patients and metabolic syndrome with positively significant.

Independent variable	dependent variable	Sig	Correlation
Sex	duration of diabetes	.029	.244*
	body mass index	.182	.151
	Type of treatment	.008	.294**
	Blood pressure	.136	.168
	Abdominal circumference	.000	.712**
	total cholesterol	.127	.172
	low density lipoprotein	.760	.035
	high density lipoprotein	.000	.394**
	Triglyceride	.103	.184
	fasting blood glucose	.000	.403**
	HbA1C	.084	.194

**Correlation is significant at the 0.01 level (2-tailed).

*Correlation is significant at the 0.05 level (2-tailed).

Table 3: Pearson correlation analyses showed that there was association Between type 2 diabetic patients and metabolic syndrome with positively significant.

Independent variable	dependent variable	Sig	Correlation
marital status	duration of diabetes	.010	.285*
	body mass index	.022	.256*
	Type of treatment	.076	.199
	Blood pressure	.397	.096
	Abdominal circumference	.225	.137
	total cholesterol	.874	.018
	low density lipoprotein	.358	.104
	high density lipoprotein	.002	.346**
	Triglyceride	.359	.104
	fasting blood glucose	.002	.337**
	HbA1C	.002	.343**

**Correlation is significant at the 0.01 level (2-tailed).

*Correlation is significant at the 0.05 level (2-tailed).

Table 4: Pearson correlation analyses showed that there was association Between type 2 diabetic patients and metabolic syndrome with positively significant.

Independent variable	dependent variable	Sig	Correlation
Education	duration of diabetes	.005	.312**
	body mass index	.620	.056
	Type of treatment	.001	.354**
	Blood pressure	.000	.410**
	Abdominal circumference	.081	.196
	total cholesterol	.055	.215
	low density lipoprotein	.051	.219
	high density lipoprotein	.053	.217
	Triglyceride	.012	.280*
	fasting blood glucose	.001	.361**
	HbA1C	.053	.217

Correlation is significant at the 0.01 level (2-tailed).

*Correlation is significant at the 0.05 level (2-tailed).

DISCUSSION

This study included 80 patients, 40 patients with type 2 diabetes mellitus and 40 patients as control group of both sex (males & females).

The study consisted 43 females (27 ill female patients compared to 17 well female patients as control group, and 37 males (13 ill male patients compared to 24 well male patients) as control group (F:43 & M: 37). The patients were stratified to age group and sex (on the of 20 years interval). The proportions of patients, females to males were 107.5: 92.5% within the age group of 40 to 60 (77.5%). The least percentage of patients 19 (47.5%) were an age group 60 to 80 years. 3 (5.5%) patients within the age group of 80-100 years, and 15 patients (37.5%) within the age group of 20-40.

The patients were stratified to marital status, all of the patients were married. A large percentage of patients were married 68(170%) and 12(30%) were singles.

The patients were stratified also according to educational status, the majority of them were of basic education 25(62.5), while illiterates were 23 (57.5%), secondary

were 15 (37.5) and the rest percenatgewere universities education 17 (42.5%).

One of the major parameters studied was the body mass index(BMI) which used as indicator for body weight. In this study about 34 (85%) of the patients were overweight, 16 (40%) were obese, while normal weight were 30 (70%).

In USA, American overweight estimated were 60.5%, 23.9 were obese and 3% were extremely obese, thus extracted from behavioral risk factor surveillance system in 2005 (Olga M. Petrucelli, MD,2008). The patients stratified according to abdominal circumference value among male patients were abnormal weight (abdominal obesity) 16 (40%) more than 102 cm, while had 23 (60%) patients were normal weight less than 102 cm. The patients stratified according to abdominal circumference value, females patients were 36(90%) had abdominal obesity more than 88 cm. While 5patients (10%) were normal weight. The distribution of patients according to duration of diabetes mellitus in all patients, 16 (40%) had diabetes for 5-10 years, 13 (32.5%) having diabetes from 10 to 15 years, and 2 (5%) had diabetes 15

to 20, and 7 (17.5%) having diabetes for less than 5 years, and 2(5%) had diabetes for more than 20 years.

The most prevalent treatment was diet and oral hypoglycemic agents (tablets), which was used by 34(85%) of patients, and 6(15%) on insulin therapy with dietary control.

According to the blood pressure (mm/Hg), showed that the majority of patients were hypertensive accounted as 25 (62.5%) for more than 140/90, and 15 (37.5%) within the normal reference. The distribution of HbA1c among patients as a measure of glycemic control showed that the most patients 32 (80%) were poor in glycaemic control more than 7%, while 48(120%) patients were fair glycaemic control less than 7%. Blood lipids play a vital role as an important risk factor for diabetic patients. As a result of impaired action of insulin, there is an increase in triacylglyceride hydrolysis release of non-esterified fatty acids which are associated with alteration of blood lipid concentration and metabolism resulting in dyslipidemia. According to serum level of triglycerides, most patients 35(87.5%) showed a level of triglycerides was less than 150 mg/dl, in 29 (70%) of patients in between 150-199 mg/dl and 17 (42.5%) of patients more than 200 mg/dl were strongly high. The level of cholesterol in serum patients 40 (100%), the total cholesterol was less than 200 mg/dl of normal value. 31(77.5%) of patients, the cholesterol level was 200-239 mg/dl was slightly increased. In 9(22.5%) of patients was high more than 240 mg/dl. The serum high density lipoprotein among male patients was more than 60 mg/dl in 45 (112.5%) male patients, while the level of HDL in 12(30%) of male patients was less than 40 mg/dl. The serum level of HDL in female patients was 23(57.5%) which was less than 50 mg/dl, which was poor. The HDL level was lower in females 23(57%) than males 12 (30%). The fasting blood glucose among the majority of patients was increased more than 120 mg/dl in 37 (92.5%), 28 (70%) in between 101-126 mg/dl was slightly increased and the rest of patients 15 (37.5%), the blood glucose level was in normal range between 70 to 100 mg/dl. The metabolic syndrome was diagnosed according to criteria of national cholesterol education program (NCEP). In this study, the metabolic syndrome appeared well in most patients based on abdominal obesity 16 (40%) than the normal waist circumference in 23(60%) which was less than 120 cm in males, the abdominal obesity in females 36 (90%) compared to 5 (10%) with normal waist circumference less than 88 cm, elevated serum triglyceride more than 150 mg/dl in 26 (52%), poor high density lipoprotein in males 12(30%) less than 40 mg/dl, 23(57.5%) in females less than 50 mg/dl and elevated blood pressure 25(62.5%) of patients for more than 140/90 mm/Hg. The majority of patients with metabolic syndrome increased in patients on oral hypoglycemic agents 34(85%) than the patients on insulin therapy 6(15%), the duration of diabetes mellitus for 5 to 10 years and fair glycaemic control 32(80%). The study showed that, the metabolic syndrome was high in type 2 diabetic patients

particularly in females than males compared to the normal group based on diagnosis according to criteria of national cholesterol education program. The study revealed that the metabolic syndrome was increased particularly in females, that's accounted as 22 females with metabolic syndrome than 7 females with no metabolic syndrome out of 60% compared to males and control group. In males the metabolic syndrome occurred in 4 patients with no metabolic syndrome in 7 patients out of 40% compared to normal control. Finally, the frequency and occurrence of metabolic syndrome in Sudanese type 2 diabetic patients is highly serious increased problem and I recommended for screening of all type 2 diabetic patients to prevent complications and save the life of patients.

CONCLUSION

In this study the frequency of the metabolic syndrome was found significantly high, especially in women between 40 to 60 years. I highly recommended an urgent need to commence screening for this syndrome based on presence of one or two components of metabolic syndrome.

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