

## Waist Circumference in Metabolic Syndrome in the Egyptian Population

Fared F. Abd El Hafez, Khaled M. Hadhoud, Mohamed S.S. Saad and Hatem M. Salem

Department Of Internal Medicine, Faculty of Medicine, Zagazig University  
[drhatem55@hotmail.com](mailto:drhatem55@hotmail.com)

**Abstract: Background:** Metabolic syndrome has received increased attention in the past few years. It consists of multiple interrelated risk factors of metabolic origin that appear to directly promote the development of atherosclerotic cardiovascular disease. Waist circumference is a stronger indicator for the development of these cardiovascular events than generalized obesity defined by elevated body mass index (BMI). **Objective:** This study was planned to determine waist circumference cut-off points diagnostic of abdominal obesity and metabolic syndrome among Egyptians and to detect the relationship between waist circumference, as the most important parameter of metabolic syndrome, and the occurrence of diabetes and prediabetes among Egyptians. **Subjects and methods :** the study included 300 subjects , 100 of them prediabetics , 45 males and 55 females (group 1) and 100 type-2 diabetic patients, 44 males and 56 females (group 2), as well as 100 healthy subjects, 50 males and 50 females served as control group ( group 3 ). All subjects in the three groups aged from 30 – 50 years. All subjects were subjected to thorough history taking, proper clinical examination including waist circumference measurement and BMI and proper investigations with special stress on lipid profile, fasting, 2h postprandial blood glucose, HbA1c, CBC, liver function tests, blood urea and serum creatinine and uric acid levels. **Results:** The incidence of metabolic syndrome was 75% in diabetics, 38% in prediabetics and 28% in the control group. The cut-off **points** for waist circumference were 115cm for males and 105cm in females. There were highly significant difference between diabetics and prediabetics when compared with the control group as regards lipid profile with the cholesterol, triglycerides and LDL-c levels were higher in both groups than the control group ( $p < 0.001$ ) . Also, significant positive correlation was detected between waist circumference and each of age, systolic and diastolic blood pressure, fasting and 2h postprandial blood glucose and triglycerides levels in diabetics as well as in prediabetics ( $p < 0.001$ ). **Conclusion:** cut-off points of waist circumference diagnostic of metabolic syndrome in our Egyptian population are higher than those advocated in the guidelines to be confirmed by further studies because the size of waist circumference as an estimate of visceral obesity still has a matter of controversy. Also, it is recommended that patients with an elevated waist circumference with one or more of cardiometabolic risk factors require aggressive treatment because of increased health risk.

[Fared F. Abd El Hafez, Khaled M. Hadhoud, Mohamed S.S. Saad and Hatem M. Salem. **Waist Circumference in Metabolic Syndrome in the Egyptian Population.** Journal of American Science 2011; 7(12):1257-1265]. (ISSN: 1545-1003). <http://www.americanscience.org>. 156

**Keywords:** waist circumference and metabolic syndrome

### 1. Introduction

Metabolic syndrome is a common condition that goes by many names (dysmetabolic syndrome, syndrome X, insulin resistance syndrome, obesity syndrome and Reaven's syndrome)<sup>(1)</sup>

The National Heart Lung and Blood institute (NHLBI) estimates that in the U.S. about 47 million adults have metabolic syndrome. It can affect anyone at any age, but it is most frequently seen in those who are significantly overweight with most of their excess fat in the abdominal area and inactive.<sup>(1)</sup>

The metabolic syndrome has received increased attention in the past few years. It consists of multiple, interrelated risk factors of metabolic origin that appear to directly promote the development of atherosclerotic cardiovascular disease (ASCVD). This constellation of metabolic risk factors is strongly associated with type 2 diabetes mellitus or the risk for this condition. The metabolic risk factors consist of atherogenic dyslipidemia (elevated triglycerides and apolipoprotein B, small LDL particles, and low HDL cholesterol [HDL-C]

concentrations), elevated blood pressure, elevated plasma glucose, a prothrombotic state, a proinflammatory state, hyperuricemia and microalbuminuria<sup>(2)</sup>.

At present, it is not clear whether the metabolic syndrome has a single cause, and it appears that it can be precipitated by multiple underlying risk factors. The most important of these underlying risk factors are abdominal obesity and insulin resistance. Other associated conditions include physical inactivity, aging, hormonal imbalance and genetic or ethnic predisposition<sup>(2)</sup>. Obesity has genetic as well as environmental causes. It has a strong effect on the development of type 2 DM as it is found in western countries<sup>(3)</sup> and some ethnic groups such as Pima Indians<sup>(4)</sup>. Obesity is more than just a risk factor, it has a causal effect in the development of type 2 DM against a genetic background<sup>(2)</sup>.

Several adipokines, secreted by fat cells, can affect insulin action in obesity. Of these, adiponectin and leptin seem to increase sensitivity to insulin, presumably by increasing hepatic responsiveness. On

the other hand, tumor necrosis factor inactivates insulin receptors, and the newly discovered peptide resistin interferes with insulin action on glucose metabolism and had been reported to be elevated in obese animal models. Mutations or abnormal levels of these adipokines may contribute to the development of insulin resistance in human obesity<sup>(5)</sup>.

The hypertriglyceridemia seen with abdominal obesity and insulin resistance is related to the oversecretion of triglyceride-rich VLDL particles. An increased rate of hepatic FFA uptake stimulates the secretion of apo B-100, leading to increased numbers of apo B-containing particles and possibly hypertriglyceridemia<sup>(6)</sup>.

HDL and VLDL metabolism are closely linked, which explains why increased plasma triglyceride is almost always associated with reduced HDL levels. Cholesterol ester transfer protein mediates the exchange of triglyceride in VLDL for cholesterol ester in LDL and HDL, leading to the production of triglyceride-rich LDL and HDL particles. Subsequent hepatic lipase-mediated hydrolysis of these particles leads to the generation of small, dense LDL particles and a decrease in HDL cholesterol<sup>(7)</sup>.

An increased serum urate concentration (hyperuricemia) has long been recognized as a common feature in patients with the metabolic syndrome<sup>(8)</sup>.

Although, in most studies, all metabolic syndrome components correlated with urate levels, the strongest correlation was with waist circumference<sup>(9)</sup>.

Diminished uric acid excretion is reported in patients with the metabolic syndrome<sup>(10)</sup> and appears to reflect impaired renal uric acid excretion mediated by hyperinsulinemia-enhanced proximal tubular sodium reabsorption<sup>(11)</sup>.

Reduced uric acid excretion due to enhanced sodium reabsorption has also been reported in conditions such as obesity and hypertension, the two most common diseases associated with the metabolic syndrome<sup>(12)</sup>.

Microalbuminuria is an important clinical marker in patients with diabetes because of its well-established association with progressive renal disease<sup>(13)</sup>. It is also becoming increasingly recognized as an independent risk factor for cardiovascular disease in patients with hypertension and diabetes,<sup>(14)</sup> and also in the general population<sup>(15)</sup>.

Microalbuminuria occurs in 11% to 40% of persons with hypertension, the prevalence increasing with age and the duration of hypertension<sup>(16)</sup>.

The prothrombotic and proinflammatory states are not included in the proposed criteria for metabolic syndrome due to the belief that these states are consequences of other risk factors. A prothrombotic state is characterized by abnormalities, specifically elevations, in procoagulant factors, antifibrinolytic factors, platelet alterations, and endothelial dysfunction<sup>(17)</sup>. A proinflammatory state is characterized by

elevations of circulating inflammatory molecules such as C-reactive protein (CRP), tumor necrosis factor- $\alpha$ , plasma resistin, interleukin (IL)-6, and IL-18<sup>(18)</sup>. CRP is a general marker of inflammation that has been linked to cardiovascular disease (CVD) in patients with metabolic syndrome<sup>(19)</sup>. Routine assessment is not encouraged; however, elevated levels of CRP are associated with many of the clinical features of the syndrome (i.e., increased waist circumference). Although a prothrombotic or a proinflammatory state is not involved with the clinical recognition of metabolic syndrome, both are found to aggregate with the diagnostic criteria<sup>(20)</sup>.

Also, hyperinsulinemia increases GnRH pulse frequency, LH over FSH dominance, increased ovarian androgen production, decreased follicular maturation, and decreased sex hormone binding globulin (SHBG); all these steps lead to the development of polycystic ovary syndrome (PCOS). Insulin resistance is a common finding among patients of normal weight as well as those overweight patients. PCOS may be associated with chronic inflammation, with several investigators correlating inflammatory mediators with anovulation and other PCOS symptoms<sup>(21)</sup>.

Abdominal obesity characterized by high waist circumference is a stronger predictor than generalized obesity defined by elevated BMI (Body mass index) of subsequent development of major coronary events, vascular mortality, diabetes and metabolic syndrome. Men and women who have waist circumference greater than 102 cm and 88 cm, respectively, are considered to be at increased risk for cardio-metabolic disease<sup>(22)</sup>. Furthermore, increased waist circumference is a central component of the metabolic syndrome. Recent IDF (International Diabetes Federation) guidelines for definition of metabolic syndrome identify an increased waist circumference as a prerequisite for diagnosis ( $\geq 94$  cm in males and  $\geq 80$  cm in females in Europeans)<sup>(23)</sup>. Indeed, although an elevated waist circumference per se alerts the clinician to the need for further clinical assessment, it has been shown that only patients with an elevated waist circumference in combination with elevations in one or more cardiometabolic risk factors represent those who are at substantially increased health risk and thus require aggressive treatment<sup>(24)</sup>.

A very high rate of obesity was reported among Egyptians. Recently estimates of waist circumference were gaining increasing importance as a more useful tool in the assessment of body fat distribution and in the diagnosis of abdominal obesity.

In Egypt, other Arab and Middle Eastern countries, the thresholds of waist circumference diagnostic of abdominal obesity are derived from European data<sup>(25)</sup>.

There is a need to develop national guidelines for definition of abdominal obesity.

So, this study was planned to determine waist circumference cut-off points diagnostic of abdominal obesity and metabolic syndrome among Egyptians and to detect the relationship between waist circumference and occurrence of diabetes and prediabetes among Egyptians.

## 2. Subjects and Methods

This study was carried out in the department of Internal Medicine, faculty of Medicine, Zagazig University. The study was conducted on 300 subjects (139 male and 161 female).

### They were divided into the following groups:

#### Group 1:-

It included 100 prediabetics, 45 males and 55 females, 3% had hypertension and 1% had ischemic heart disease.

#### Group 2:-

It comprised 100 type 2 diabetic patients, 44 males and 56 females. The duration of diabetes was between 5-10 years, 23% had hypertension and 7% with Ischaemic heart disease; 67% of patients were on oral therapy, 28% on Insulin therapy and 5% on combination of insulin and oral therapy.

#### Group 3:-

It included 100 healthy subjects, 50 males and 50 females.

Subjects aged from 30 to 50 years old with a mean value  $\pm$  SD of (41.36 $\pm$ 6.0) for prediabetics, (43.9  $\pm$  5.6) for diabetics and (41.1  $\pm$  6.1) for the Control group.

Patients were randomly recruited from those attending the diabetes out-patient clinic of zagazig university hospitals.

**After being informed on the purpose and procedures of the study, all subjects signed an informed consent form.**

Type 2 DM and prediabetes were diagnosed according to American Diabetes Association Guidelines for diagnosis and classification of DM<sup>(26)</sup>.

Subjects with ischemic heart disease refers to those with self-reported and confirmed history of angina or myocardial infarction documented by ECG<sup>(27)</sup>.

The following criteria were considered as exclusion Criteria: all conditions affect blood glucose level and lipid profile as subjects on dietary regimen, patients with chronic liver disease, chronic kidney disease or autoimmune disease and patients on antihyperlipidaemic drugs.

### All patients and control subjects were submitted to:

\* Thorough history taking with special stress on age, sex, duration of diabetes and type of treatment.

\* proper clinical examination with special stress on body mass index (BMI), blood pressures determination, signs of diabetic Complications and waist circumference measurement by simple tape at the part of the trunk located midway between the lower costal margin (bottom of lower rib) and the iliac crest (top of pelvic bone) while the person is standing, with feet about 25-30 cm (10-12 inches). The measurer stands beside the individual and fit the tape snugly, without compressing any underlying soft tissues. The circumference should be measured to the nearest 0.5 cm (1/4 inches), at the end of a normal expiration<sup>(28)</sup>.

\* Laboratory investigations including:

- CBC
- Liver functions tests
- Blood urea and serum creatinine and uric acid levels.
- Lipid profile (HDL, LDL, Triglycerides and total cholesterol)
- Fasting and 2 hour postprandial blood glucose.
- Glycosylated hemoglobin (HbA1c).

### We diagnosed the metabolic syndrome by:

The US National Cholesterol Education Program Adult Treatment Panel III (29) requires at least three of the following (including waist circumference):

\* central obesity: waist circumference  $\geq$ 102 cm or 40 inches (male),  $\geq$  88 cm or 36 inches (female)

\* dyslipidaemia: TG  $\geq$  1.7 mmol/L (150 mg/dl)

\* dyslipidaemia: HDL-C < 40 mg/dL (male), <50 mg/dL (female)

\* blood pressure  $\geq$  130/85 mmHg

\* fasting plasma glucose  $\geq$  6.1 mmol/L (110 mg/dl)

### Statistical analysis

The statistical analysis of data was done by using SPSS program (Statistical package for social science) version 16 on windows XP.

The description of data was done as:

1- Frequency and proportion for qualitative data.

2- Mean  $\pm$  SD for normally distributed quantitative data.

3- ROC curve to detect cutoff point for waist circumference.

The analysis of data was done to test statistical significant difference between groups:

1- for qualitative data (frequency & proportion) Chi-square test was used.

2- for quantitative data normally distributed (mean  $\pm$  SD)

\* student t-test was used to compare 2 groups.

\* One way Anova test was used to compare more than 2 groups.

3- Correlation coefficients to detect relationships between more than one item.

**N.B P** is significant if < 0.05.

## 3. Results

### Table (1): Clinical characteristics of the studied Subjects:

The Incidence of metabolic syndrome was 75% in diabetics, 38% in prediabetics and 28% in the control group. The Cutoff points for waist circumference were investigated by ROC curve, the optional point 115 cm for males and 105 cm for females. As regards lipid profile, there were highly significant difference between diabetics and prediabetics when compared with the control group, the cholesterol level, triglycerides level and LDL Level were higher in both groups than control group, on the other hand non significant difference was found between the three groups as regards HDL level. The Cardiometabolic risk increased with diabetics more than other groups [as regards HTN, IHD].

### Table (2): Relationship between waist circumference and all parameters in all parameters in all subjects:

There was a positive Correlation between all parameters and waist circumference except HDL-C which showed a negative correlation with waist circumference.

### Table (3) : Correlation between waist Circumference and other parameters in each group in the study :-

Significant positive correlation was detected between waist circumference and each of age, systolic and diastolic blood pressure, fasting and 2h postprandial blood glucose and triglycerides levels in diabetics as well as in prediabetics .

In the control group, a significant correlation was found between waist circumference and each of triglycerides level and blood pressure.

**Table 1: Clinical Characteristics of the studied subjects**

|  | Group I<br>prediabetics     | Group II<br>Diabetics          | Group III<br>control       |                              |
|--|-----------------------------|--------------------------------|----------------------------|------------------------------|
| <b>Age (years)</b>                         |                             |                                |                            |                              |
| (±SD)                                      | 41.36 ±6.0                  | 34.9 ± 5.6                     | 41.1 ±6.1                  | F = 6.735                    |
| Range                                      | (30 - 50)                   | (30 - 50)                      | (30 - 53)                  | P = 0.00137                  |
| <b>Waist circumference (c.m)</b>           |                             |                                |                            |                              |
| Male                                       | 104.31 ± 8.6<br>(87-125)    | 104.5 ± 13.2<br>(80-130)       | 97.7 ± 9.6<br>(80 -120)    | F 5.94<br>P 0.1003           |
| Female                                     | 96.7 ± 10.2<br>(80 - 120)   | 108.5 ± 10.5<br>(88-135)       | 97.8 ±9.5<br>(82 -127)     | F 22.19<br>P <0.001          |
|  | T 4.4 P<0.01                | T 1.66<br>P 0.01               | T 0.04<br>P 0.96           |                              |
| <b>BL. P(mmHg)</b>                         |                             |                                |                            |                              |
| S.BL.P                                     | 123.5 ± 13.95<br>(100- 180) | 129 ± 15.3<br>(100 - 170)      | 122.8 ±16.6 (90<br>-160)   | F 4.89<br>P 0.001            |
| D.BL.P                                     | 80.1 ± 7.8 (60 -<br>110)    | 82.75 ± 9.4<br>(60-110)        | 79.1 ± 10.7 (60-<br>110)   | F 4.04<br>P 0.018            |
| <b>Blood Glucose level (ml/dl)</b>         |                             |                                |                            |                              |
| FBG  | 106.7 ± 14.3<br>(75 -125)   | 187.8 ± 67.2<br>(70 - 378)     | 81.85 ± 11.0<br>(65 -103)  | F 190<br>P <0.01             |
| PP (post<br>prandial)                      | 161.3 ± 25.1<br>(105 - 205) | 279.5 ± 91.8<br>(98 - 550)     | 117.0± 13.7 (80-<br>150)   | F 190 P<br><0.001            |
| <b>Lipid profile (ml/dl)</b>               |                             |                                |                            |                              |
| Cholesterol<br>X±SD                        | 203.5 ± 46.5<br>(120 - 300) | 195.8 ±<br>36.7 (125 -<br>300) | 165.0 ± 37.8<br>(100-265)  | F 25.5<br>P 39<br>P < 0.001  |
| Triglycerides<br>X±SD                      | 163.3 + 41.0 (90<br>- 250)  | 176.9 ±<br>38.4 (104-<br>290)  | 145.2 ± 39.7 (61<br>- 340) | F 16.07<br>P 33<br>P < 0.001 |
| LDL<br>X±SD                                | 118.2 ±38.3 (42-185)        | 108.1 ± 29.2 (46 -<br>182)     | 93.9 ± 27.5 (41 -193)      | F 14.5<br>P 14.5<br>P <0.001 |
| HDL<br>X±SD                                | 53.6 ±14.8 (19-<br>95)      | 50.2 ±22.3<br>(15-102)         | 50.4 ± 12.3 (21 -<br>84)   | F 1.24<br>P 0.29<br>P <0.001 |
| <b>Presence of other diseases</b>          |                             |                                |                            |                              |
| - ve                                       | 95%                         | 67%                            | 91%                        | X2 34.72<br>P 34.72          |
| +ve  | 5%                          | 33%                            | 9%                         | P 3 < 5.001                  |
| <b>Incidence of metabolic syndrome (%)</b> |                             |                                |                            |                              |
|  | 38%                         | 75%                            | 28%                        |                              |

**Table 2: Relationship between waist circumference and all parameters in all subjects**

| All parameters          | r     | P      | sig |
|-------------------------|-------|--------|-----|
| Age                     | 0.47  | <0.001 | HS  |
| Systolic Bl.P           | 0.41  | <0.001 | HS  |
| Diastolic Bl.P          | 0.38  | <0.001 | HS  |
| F.B.G                   | 0.15  | <0.01  | SIG |
| p.p                     | 0.16  | <0.01  | SIG |
| T.C (total cholesterol) | 0.19  | <0.001 | HS  |
| T.G                     | 0.32  | <0.001 | HS  |
| H.D.L                   | -0.04 | >0.05  | NS  |
| L.D.L                   | 0.14  | <0.01  | SIG |

**Table 3: Correlation between waist circumference and other parameters in each group.**

| <b>a- prediabetic group</b> |              |                   |            |
|-----------------------------|--------------|-------------------|------------|
| I                           | r            | P                 | Sig        |
| Age                         | <b>0.6</b>   | <b>&lt; 0.001</b> | <b>HS</b>  |
| Bl.p                        |              |                   |            |
| Syst.                       | <b>0.38</b>  | <b>&lt;0.001</b>  | <b>HS</b>  |
| Diast.                      | <b>0.47</b>  | <b>&lt; 0.001</b> | <b>HS</b>  |
| FBG                         | <b>0.4</b>   | <b>&lt;0.001</b>  | <b>HS</b>  |
| PP                          | <b>0.45</b>  | <b>&lt;0.001</b>  | <b>HS</b>  |
| TC                          | <b>0.27</b>  | <b>&lt;0.01</b>   | <b>HS</b>  |
| TG                          | <b>0.31</b>  | <b>&lt; 0.001</b> | <b>HS</b>  |
| HDL                         | <b>-0.13</b> | <b>&gt;0.05</b>   | <b>NS</b>  |
| LDL                         | <b>0.23</b>  | <b>&lt;0.05</b>   | <b>Sig</b> |

| <b>b- Diabetic group</b> |       |         |     |
|--------------------------|-------|---------|-----|
|                          | r     | P       | Sig |
| Age                      | 0.37  | <0.001  | HS  |
| Bl. p                    | 0.45  | <0.001  | HS  |
| Syst.                    | 0.35  | < 0.001 | HS  |
| Dias.                    |       |         |     |
| FBG                      | 0.43  | <0.001  | HS  |
| PP                       | 0.41  | <0.001  | HS  |
| TC                       | 0.08  | >0.05   | NS  |
| TG                       | 0.23  | <0.05   | Sig |
| HDL                      | -0.05 | >0.05   | NS  |
| LDL                      | 0.05  | >0.05   | NS  |

| <b>c. Control group.</b> |      |         |     |
|--------------------------|------|---------|-----|
|                          | r    | P       | Sig |
| Age                      | 0.37 | <0.001  | HS  |
| Bl.P                     |      |         |     |
| Syst.                    | 0.32 | <0.001  | HS  |
| Diast.                   | 0.29 | <0.001  | HS  |
| FBG                      | 0.06 | >0.05   | NS  |
| PP                       | 0.08 | <0.05   | NS  |
| TC                       | 0.16 | > 0.05  | NS  |
| TG                       | 0.3  | < 0.001 | HS  |
| HDL                      | 0.12 | >0.05   | NS  |
| LDL                      | 0.13 | > 0.05  | NS  |

#### 4. Discussion

A quarter of the world's adults have metabolic syndrome. People with metabolic syndrome are twice as likely to die from, and three times as likely to have a heart attack or stroke compared with people without the syndrome. People with metabolic syndrome have a five-fold greater risk of developing type 2 diabetes. Up to 80% of the 200 million people with diabetes globally will die because of cardiovascular disease<sup>(30)</sup>.

Obesity has been known to be positively related to insulin resistance. Increased secretion of free fatty acids, inflammatory cytokines and decreased secretion of adiponectin are molecules mediating obesity and insulin resistance<sup>(31)</sup>.

Insulin resistance is an obesity-related condition preceding the development of impaired glucose tolerance and type 2 diabetes. Insulin resistance, through suppression of glucose uptake in skeletal muscle and increase in hepatic glucose production, causes hyperglycemia<sup>(32)</sup>.

Visceral obesity is closely linked to insulin resistance, and is currently regarded as a principle component of the metabolic syndrome. It is well documented that insulin resistance is predictive of the risk of type 2 diabetes and cardiovascular disease<sup>(33)</sup>.

In conjunction with worldwide recognition of the metabolic syndrome, the size of waist circumference as an estimate of visceral obesity has been a matter of controversy. The International Diabetes Federation (IDF) has adopted different cutoffs for waist circumference in different ethnicities<sup>(34)</sup>; the cutoff points for Europeans are 94 cm in men and 80 cm in women while those for Chinese, South Asians and Japanese are 90 in men and 80 in women.<sup>(35)</sup> The Korean Society for the Study of Obesity (KSSO) defined the waist circumference cutoff value as 90 cm for men and 85 cm for women<sup>(36)</sup>. As regard our study we found that cutoff points are 115 cm in male and 105 cm in female which are far away from that obtained by study done in the past in which male cutoff point was 97.5 cm and female 92.3 cm<sup>(37)</sup>. Our study showed positive correlation between waist circumference and increasing risk of diabetes, hypertension, dyslipidaemia table 3a,b. These results were in agreement with that of **Esteghamati et al**<sup>(38)</sup> who found that there were +ve correlation between waist circumference and age, blood pressure, blood glucose level, and triglycerides in diabetics. And in normal group they reported positive correlation between waist circumference, Age, Blood pressure, Triglycerides in harmony with results of our study [table 3c].

In our study the mean waist circumference in diabetics (male 104.5±13.2 cm and in female 108.5±10.5 cm) and normal subjects (male 97.7±9.6 cm and female 97.8±9.5 cm) is greater than Iranian population diabetics (male 99.5±0.4 cm and female 97.9±0.4 cm) and normal subjects (male 93.2±0.6 cm and female 90.9±0.4 cm)<sup>(39)</sup>.

Also we have higher figures in waist circumference in comparison to the Framingham Heart Study<sup>(40)</sup> in which waist circumference in normal subjects was

33.6±5.1 inches (89.016±13.056 cm) and in diabetics 40.1±4.5 inches (102.656±11.52 cm), and incidence of metabolic syndrome was 40.6%.

The present study demonstrated a strong linear relationship between waist circumference and metabolic syndrome in Egyptian population. The increase was evident even in subjects with the average size of waist, i.e., 80-127 cm. It was also found that 115 cm in male and 105 cm in female of waist circumference were an optimal cutoff for predicting metabolic syndrome. The big difference between our figures and figures of the other studies in the world, may be due to genetic factors, level of activity, behaviour, endocrine factors, race, sex, and age factors, ethnic and cultural factors, socioeconomic status, dietary habits, smoking cessation, pregnancy, menopause and psychologic factors.

Also we found that mean of waist circumference in diabetics was significantly higher than prediabetics and normal subjects ( $p < 0.001$ ) (table 1). In agreement with our results are that of **Peter et al**<sup>(41)</sup> who found that the results of the logistic regression models in which waist circumference groups were used to predict the likelihood of having diabetes and cardiovascular disease (CVD). After controlling for age, sex, race, and smoking, participants in the medium and high waist circumference groups were more likely to have diabetes and CVD compared with participants in the low waist circumference group ( $P < 0.05$ ) in Canadian population. They have waist circumference less than our population in male (98.5±0.4) and in female (92.1±0.5).

In a Chinese study by **Ting Liu et al**<sup>(42)</sup> it was found that elevated blood pressure and central obesity were the most prevalent components of metabolic syndrome in men and women. These results go in harmony with our results as there were +ve correlation between waist circumference and blood pressure in all patients (table 2).

Our study showed a higher incidence of metabolic syndrome as regards the international figures according to I.D.F criteria for diagnosis, in diabetic groups the incidence was 75%, in prediabetic groups 38% and in normal individuals 28%. **Alireza Esteghamati et al**<sup>(43)</sup> found that the incidence in Iranian population in diabetics 69% and in normal individuals 32% but **Sharifi et al**<sup>(44)</sup> found that the incidence was 23.7% in Western part of Iran. As regards the Latin American incidence it differs according to locality; Mexico city 27%, Bogota 20% and Lima 18%<sup>(45)</sup>. Also in Framingham study in U.K in 2009 the incidence of metabolic syndrome in diabetics was 40.6%.

In Nigeria the incidence of the metabolic syndrome was 86% among diabetics. The frequency of occurrence of the metabolic syndrome was similar for men and women and increased with age in both sexes. The commonest occurring and least detected metabolic syndrome defining parameters are central obesity and elevated triglyceride levels respectively. The component of the metabolic syndrome that differed significantly in both sexes was HDL-C as found by

**Anthonia O Ogbera** <sup>(46)</sup>. But in our study the central obesity and elevated triglyceride levels were the commonest occurring parameters of metabolic syndrome and in addition to negative correlation with HDL-C.

Before coming to an end it is noteworthy to mention that the

Primary finding of this study is that waist circumference predicts the likelihood of diabetes beyond that explained by commonly evaluated cardiometabolic risk factors and BMI. Clinical guidelines for the assessment and/or management of obesity in the U.S. <sup>(47)</sup> and Canada <sup>(48)</sup> recommend that measurement of waist circumference should be used to identify the need for further assessment including measurement of cardiometabolic risk factors. The recent consensus statement of the ADA, the Obesity Society, and the American Society for Nutrition questions sequence of these clinical measures and, more importantly, the relevance of waist circumference measurement in clinical practice <sup>(49)</sup>.

**Katzmarzyk et al** <sup>(50)</sup> and **Despres et al.** <sup>(51)</sup> have shown that only patients with an elevated waist circumference in combination with elevations in one or more cardiometabolic risk factors represent those who are at substantially increased health risk and thus require aggressive treatment.

The mechanistic link that explains the association between waist circumference and diabetes risk independent of cardiometabolic risk factors is unclear and remains the focus of ongoing investigation <sup>(52)</sup>. Although the portal theory originally proposed a substrate-driven mechanism <sup>(53)</sup>, recent evidence suggests that the pathophysiology of abdominal adiposity may result from the augmented secretion of various prothrombotic and proinflammatory cytokines from an expanded abdominal fat depot <sup>(54)</sup>.

Accordingly, this finding does not indicate that a high waist circumference is not a risk factor for CVD but, rather, that waist circumference predicts CVD via its influence on cardiometabolic risk factors. Indeed, the utility of waist circumference to predict CVD risk will always be attenuated when metabolic risk factors that lie in the causal pathway between waist circumference and risk of CVD are included in the prediction model. This observation agrees with the findings of the INTERHEART study, in which the strong association between waist circumference and myocardial infarction was substantially attenuated after control for hypertension and the apolipoprotein B-to-A ratio <sup>(55)</sup>.

Numerous studies, however have shown that high waist circumference and BMI precede the onset of morbidity <sup>(56)</sup> and mortality <sup>(57)</sup>.

The demonstration that waist circumference predicts risk of diabetes beyond that explained by cardiometabolic risk factors routinely acquired in

clinical practice responds to prior criticism by Klein et al. <sup>(49)</sup> and lends critical support for the recommendation that waist circumference should be a routine measure for identification and management of the high-risk, abdominally obese patient <sup>(48)</sup>. Indeed, combined with the observation that waist circumference is associated with changes in abdominal obesity in response to treatment with or without weight loss <sup>(25)</sup>, it is difficult to imagine a cogent argument against inclusion of waist circumference in clinical practice.

From a clinical perspective, it is noteworthy that in addition to the utility of waist circumference measurement to identify the high-risk, abdominally obese patient, waist circumference is the single best anthropometric measure for detecting changes in abdominal obesity in response to treatment. It has repeatedly been demonstrated that although waist circumference is reduced consequent to weight loss, waist circumference can also be reduced in response to treatment in obese individuals who are resistant to weight loss or changes in BMI <sup>(25)</sup>. The implication is that when considering the efficacy of treatment strategies designed to manage abdominal obesity, practitioners are encouraged to look beyond body weight as the measure of benefit and measure waist circumference.

From the above mentioned discussion we can reach a conclusion that waist circumference cutoff points found in the current study in male 115 cm and in female 105cm, which were far away from those advocated in the guidelines ( $\geq 102$  and 88 in men and women respectively). Also 75% of diabetics, 38% of prediabetics and 28% of normal subjects have metabolic syndrome. In addition to that the mean cardiometabolic risk as fasting blood glucose, blood pressure and lipid profile was greater than that of international figures.

### Recommendation

Further studies on larger number of people are recommended to find exact figures for cutoff points of waist circumference diagnostic of metabolic syndrome in our Egyptian population.

Also, it is recommended that patients with an elevated waist circumference in combination with elevation in one or more cardiometabolic risk factors require aggressive treatment as they represent those who are at substantially increased health risk.

### Corresponding author

**Hatem M. Salem**

Department Of Internal Medicine, Faculty of Medicine, Zagazig University

[drhatem55@hotmail.com](mailto:drhatem55@hotmail.com)

### References

- 1- Rizzi, R(2006): opposing views on metabolic syndrome . Clinical laboratory news, American Association for Clinical Chemistry., 32:1, modified on March 31.2011,.

- 2- American Heart Association Science(2005) : Advisory and Coordinating Committee on August 10, 2005 , and by the National Heart , Lung , and Blood Institute in July.
- 3- Alberti KG, P. Zimmet, J.Shaw(2005): The metabolic syndrome a new world – wide definition . Lancet. 2005;366: 1059– 62.
- 4- Knowler WC , RG. Nelson, MF .Saad and Pettitt DJ(1993): Determinants of diabetes mellitus in the pima Indians. Diabetes Care.;16:216:227.
- 5- Current Medical Diagnosis & Treatment (2007 ) : (Diabetes Mellitus & Hypoglycemia ) by : Lawrence M.Terney, Stephen J McPhee, is the 46 th annual volume, copyright, The McGraw – Hill Companies ,2007;chapter 27, The page no 1221
- 6- Marsh JB(2003) : Lipoprotein metabolism in obesity and diabetes : insights from stable isotope kinetic studies in humans . Nutr Rev.; 61:363-375 .
- 7- Tribble DL M. Rizzo, A. Chait, et al. (2001): Enhanced oxidative susceptibility and reduced antioxidant content of metabolic precursors of small, dense low – density lipoproteins. Am J Med ; 110:103-110.
- 8- Sarafidis PA, PM .Nilsson.(2006): The metabolic syndrome : a glance at its history .J Hypertens.; 24:621-262. Excellent review of the metabolic syndrome history .
- 9- Puig JG, MA. Martinez, M. Mora, et al (2007): Serum urate, metabolic syndrome and cardiovascular risk factors : a population –based study. Nucleos Nucleot Nucl.;17:869-872.
- 10- Lopez – Suarez A, J .Elvira- Gonzalez , A. Bascunana-Quire, et al(2006).: Serum urate levels and urinary uric acid excretion in subjects with metabolic syndrome . Med Clin ( Barc).;126:321 - 324.
- 11- Strazzullo P, A .Barbota, F.Galletti, et al (2006): Abnormalities of renal sodium handling in the metabolic syndrome: results of the Olivetti Heart Study. J Hypertens.; 24:1633-1639.
- 12- Strazzullo P, JG. Puig(2007): Uric and oxidative stress: relative impact on cardiovascular risk. Nutr Metab Cardiovasc dis ; 17:409-414.
- 13- American Diabetes Association(2003): Diabetic nephropathy. Diabetes Care.;26:594-598.
- 14- Mogensen CE(2003): Microalbuminuria and hypertension with focus on type 1 and type 2 diabetes.J Intern Med.; 254:45-66.
- 15- Romundstad S, J .Holmen, K .Kvenild, et al(2003): Microalbuminuria and all-cause mortality in 2.089 apparently healthy individuals a 4.4-year follow-up study . Am J Kidney Dis.; 42:466-473.
- 16- Rossa TT, P. Palatini (2000): Clinical value of Microalbuminuria in hypertension. J Hypertens.; 18:645-654.
- 17- Grundy SM(2006): Metabolic syndrome: connecting and reconciling cardiovascular and diabetes worlds. J Am Coll Cardiol.; 47:1093-1100.
- 18- Bahia L, LG. Aguiar, N. Villela, et al.(2006): Relationship between adipokines, inflammation, and vascular reactivity in lean controls and obese subjects with metabolic syndrome. Clinics.; 61:433-440.
- 19- Clearfield MB(2005): C-reactive Protein: a new risk assessment tool for cardiovascular disease. J Am Osteopath Assoc.; 105:409-416.
- 20- Hansen B, SC .Smith, SM .Grundy, et al.(2004): Clinical management of metabolic syndrome: report of the American Heart Association/National Heart, Lung, and Blood Institute/American Diabetes Association Conference on scientific issues related to management. Circulation.; 109:551-556.
- 21- Gonzalez F, N .Rote, J. Minium, J .Kirwan(2006): "Reactive oxygen species-induced oxidative stress in the development of insulin resistance and hyperandrogenism in polycystic ovary syndrome.". J Clin Endocrinol metab.; 91 (1):336-40.
- 22- Kissebah AH, N .Vydelingum, R Nurray et al.(1982): Relation of body fat distribution to metabolic complications of obesity. J Clin Endocrinol Metab.; 54:254-260.
- 23- Waist Circumference and cardiometabolic risk(2007): a consensus statement from Shaping America's Health: Association for Weight Management and Obesity Prevention; NAASO, The Obesity Society, the American Society for Nutrition; and the American Diabetes Association. Diabetes care.; 30:1647-165.
- 24- Janiszewski(2008) : Themed Review: Lifestyle Treatment of the Metabolic Syndrome. American Journal of Lifestyle Medicine.; April 1, 2:99-108.
- 25- Ross R, J .Aru , J .Freeman, et al.(2001) : Abdominal adiposity and insulin resistance in obese men . Am J Physiol Endocrinol Metab.;282:657-63 .
- 26- Expert Committee of the Diagnosis and Classification of DM(1997): Report of the expert committee of diagnosis and classification of DM. Diabetes Care.; 20:1183- 1197.
- 27- Mittelmarke MB , BM. pasty, LP. Fried , et al .(1993):prevalence of cardiovascular disease among older adults. The cardiovascular Health study. Am. J Epidemiol ;137:311-317.
- 28- Reeder BA, A .Senthilvelan, Despres JP, H. Wang et al.(1997): The association of cardiovascular disease risk factors with abdominal obesity in Canada. Canadian Heart Health Surveys Research Group .CMAJ.;157 Suppl 1: S39-45 .
- 29- National Cholesterol Education program (NCEP)(2002): Adult Treatment panel III final report. Circulation ; 106:3143-3421.
- 30- The IDF Consensus worldwide definition of the metabolic syndrome (article on line), 2010, Available from <http://WWW.idf.org/webdata/docs/MetSdefupdate2010.pdf>
- 31- Matsuzawa Y(2006) : The metabolic syndrome and adipocytokines. FEBS Lett .;580:2917-2921.

- 32- Bonora E, S .Kiechl, J .Willeit, et al .(2007) : Insulin resistances as estimated by homeostasis model assessment predicts incident symptomatic cardiovascular disease in Caucasian subjects from the general population : the Bruneck study. *Diabetes Care.*;30:318-324.
- 33- Abdul – Ghani MA, K .Williams, RA .DeFronzo, M .Stern (2007):What is the best predictor of future type 2 diabetes.*Diabetes Care.*; 30:1544-1548.
- 34- Alberti KG, P .Zimmet, J. Shaw(2005):IDF Epidemiology Task Force Consensus Group. The metabolic syndrome-a new worldwide definition. *Lancet.*;366:1059-62.
- 35-Shinji Tabata, Shinichiro Yoshimitsu(2009): Tadamichi Hamachi Department of Preventive Medicine and Self-Defense Force Fukuoka Hospital , Faculty of Medical Sciences, Kyushu University, Fukuoka, Japan. *BMC Endocrine Disorders*: 1doi. 10.1186/1472-6823-9-1 . 9.
- 36-Lee JS, K .Kawakubo, K. Mori, A .Akabayashi(2007): Effective cut-off values of waist circumference to detect the clustering of cardiovascular risk factors of metabolic syndrome in Japanese men and women *Diab Vasc Dis Res.* 4:340-345.
- 37- Mohsen Ibrahim M.(1993): Abdominal obesity in Egypt: cut-off values of waist circumference and associated cardiovascular risk. In press..
- 38-Esteghamati et al.(2010): *Diabetology & Metabolic Syndrome*.2010 ;2:36.
- 39-Ali zandieh (2010) : Endocrinology and Metabolism Research Center (EMRC) ,Vali-Asr Hospital , school of Medicine, Tehran University of Medical Sciences, Tehran, Iran ..
- 40-Oscar H. M .Franco,Joseph. Massaro, Jacky Civil, R .Mark. Cobain(2009) : Form Unilever Corporate Research, Sharnbrook, UK (O.H.F., J.C., M.R.C., B.O.); University of Warwick, Warwick Medical School, Health Sciences Research Institute, Coventry, UK (O.H.F.); Department of Mathematics/Statistics and Biostatistics, Boston University, Boston, Mass (J.M.M.); and the National, Heart, Lung and Blood Institute's Framingham Heart Study, Framingham, Mass (R.B.D.).*Circulation.*; 120:1943-1950.
- 41- Peter M et al.(2008) : Themed Review. Lifestyle Treatment of the Metabolic Syndrome .*American Journal of Lifestyle Medicine* April 1, 2:99- 108
- 42- Ting liu et al.(2006): How to define prehypertension in Diabetes / Metabolic syndrome. *Am J Med.*; 119:133-141.
- 43-Alireza Esteghamati(2010): Endocrinology and Metabolism Research Center (EMRC), Vali-Asr Hospital,School of Medicine, Tehran University of Medical Sciences, Tehran, Iran. Esteghamati et al. *Diabetology & Metabolic Syndrome*;2:36
- 44- Sharifi F. (2009) : Prevalence of Metabolic Syndrome in an Adult Urban Population of the West of Iran. *Experimental Diabetes Research.*;Volume 2009 Article ID 136501, 5 pages
- 45- Jorge Escobedo(2009) : Medical Research Unit on Clinical Epidemiology, Mexican Social Security Institute, Mexico City, Mexico, Gabriel Mancera 222m Col. Del Valle, 03100 Mexico City, Mexico. *Cardiovascular Diabetology.*;52doi: 10.1186/1475-2840-8-52.
- 46- Anthonia O Ogbera (2010): Department of Medicine, Lagos State University Teaching Hospital, Ikeja,Lagos, Nigeria. *Ogbera Diabetology & Metabolic Syndrome.*; 2:1. <http://www.dmsjournal.com/content/2/>
- 47-Aronne LJ(2002): Classification of obesity and assessment of obesity- related health risks. *Obes Res.*;10(Suppl. 2): 105S-115S.
- 48- Lau DC, JD .Douketis, KM. Morrison, et al.(2007): Canadian clinical practice guidelines on the management and prevention of obesity in adults and children [summary], *Cmaj.*; 176: S1:S13.
- 49- Klein S, DB. Allison, SB. Heymsfield et al(2007).: Waist Circumference and cardiometabolic risk: a consensus statement from Shaping America's Health: Association for Weight Management and Obesity Prevention; NAASO, The Obesity Society; the American Society for Nutrition; and the American Diabetes Association. *Diabetes Care.*;30:1647-1652.
- 50- Katzmarzyk PT, I .Janssen, R. Ross et al.(2006) : The importance of waist circumference in the definition of metabolic syndrome: prospective analyses of mortality in men. *Diabetes Care.*;29:404-409.
- 51- Despres JP, I .Lemieux, D .Prud'homme (2001): Treatment of obesity: need to focus on high risk abdominally obese patients. *BMJ.*;322:716- 720.
- 52- Snijder MB, RM .van Dam, M. Visser et al.(2006): What aspects of body fat are particularly hazardous and how do we measure them ? *Int J Epidemiol.*; 35:83-92.
- 53- Bjorntorp P(1990): " Portal " adipose tissue as a generator of risk factors for cardiovascular disease and diabetes. *Arteriosclerosis.* 10:493 – 496.
- 54- Wajchenberg BL(2000): Subcutaneous and visceral adipose tissue : their relation to the metabolic ayndrome. *Endocr Rev.*; 21:697-738.
- 55- Yusuf S, S.Hawken, S.Ounpuu et al.(2005) : Obesity and the risk of myocardial infarction in 27,000 participants from 52 countries : a case- control study.*Lancet.*;366:1640-1649.
- 56- Wang Y, EB. Rimm , MJ .Stampher et al .(2005): Comparison of abdominal adiposity and overall obesity in predicting risk of type 2 diabetes among men . *Am J Clin Nutr.*; 81: 555- Adult Treatment Panel Definition of the metabolic syndrome to Asians . *Diabetes Care*
- 57- Bigaard J, A .Tjonneland, BL .Thomsen et al.(2003): Waist circumference , BMI Smoking , and mortality in middle-aged men and women . *Obes Res.*, 11: 895- 903.

11/12/2011