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The assessment of nutritional status, insulin resistance, oxidative status and inflammatory markers of Algerian women with metabolic syndrome

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ABSTRACT

The aim of the study was to assess the nutritional status and metabolic parameters of women with metabolic syndrome. 178 patients and 171 controls were selected during general medicine examinations. The collection of nutritional data concerned the measurement of some anthropometric parameters, Food ration, glycemic and lipid panel parameters, also, renal function tests, oxidant status and inflammatory markers were determined. The results shows that the insulin resistance index, the LDL-TBARS, the triglycerides, the fibrinogen and CRP levels were increased, but the antioxidant activity was decreased and the nutritional status was Unbalanced, all these anomalies could facilitate the appearance of serious complications in future.

Key words: Antioxidant Status, C-Reactive Protein, Insulin Levels, Metabolic Syndrome, Nutritional Status.

INTRODUCTION

Metabolic syndrome is a combination of abdominal obesity, hyperglycaemia, dyslipidaemia and high blood pressure [2]. This metabolic dysfunction increases the risk of diabetes and cardiovascular disease [22].

Metabolic syndrome affects 64 million Americans [16], 30 million European [20], in Algeria, this disease affects 26.33% of the population and 32.7% of Algerian women [4].

The emergence of the metabolic syndrome is linked to a lifestyle characterized by a diet high in simple carbohydrates, saturated fat and sodium [42], also smoking, drink, physical inactivity and work stress [14].

Visceral obesity increases the flow of free fatty acids to the liver and affects the action of insuluine and increases blood sugar. Also, adipocytes affect lipoprotein metabolism, increase the secretion of VLDL and reduced HDL synthesis [21]. Additionally, abdominal adipose tissue favors the production of angiotensinogen (factor of hypertension), cytokine synthesis (pro-inflammatory) and the production of free radicals (pro-oxidant) [1].

The objective of this study was to compare the change in nutritional status, blood glucose, lipids, inflammation, renal function, oxidative and antioxidant status, vitamins and trace elements in Algerian women with metabolic syndrome compared to control subjects.

EXPERIMENTAL SECTION

The study lasted three years between July-2011 and May-2014 in the Department of General Medicine of Youcef Damardji hospital (Tiaret, Algeria) and Meslem Tayeb hospital (Mascara, Algeria). The purpose of the study was explained to all participants and investigation was carried out with their written consent. The investigated cohort was selected during general medicine examinations. Study subjects were 178 women with metabolic syndrome of

median age 55 ± 7.1 years and 171 control women of median age 51± 6.3 years. The inclusion criteria were women aged between 40 and 60 years. The presence of metabolic syndrome was determined by using definition proposed by the International Diabetes Federation (IDF) [2]: A women to be defined as having metabolic syndrome they must have: Waist circumference: ≥ 80 cm, plus any two of the following four factors: Fasting glycemia : ≥ 5,6 mmol/l ; Blood pressure : ≥ 130/85 mm Hg ; Triglycerides : ≥ 1,7 mmol/l and HDL-Colesterol : < 1,29 mmol/l.

Anthropometric parameters and basic metabolic rate (BMR)

The body weight was measured with a minimum of clothing using an electronic scale (SECA, Germany) with an accuracy of ± 50 g. The height was determined in a vertical position, without shoes and heels together with a stadiometer for wall mounting. The body mass index (BMI) was calculated as the weight (Kg) divided by the square of the height (m). The waist circumference was measured at level midway between the lowest rib and the iliac crest. The hip circumference was determined around the widest of the buttocks portion. Waist-hip ratio was calculated as the waist circumference divided by hip measurement. The basal metabolic rate was evaluated according to the formula of Black [8]:

BMR (in Megajoule) = $0{,}963 \times W^{0,48} \times H^{0,5} \times A^{-0,13}$;

W: body weight (kg); H: height (m); A: age in years.

Food ration

Food consumption was determined by the method of seven day food record, the principle is based on recording the food consumed in a food diary and the duration of the investigation was 7 consecutive days. Diet is converted into nutrients by using the food composition tables [51].

Glycemic parameters and blood pressure

The glycemia was determined by the enzymatic technique with glucose oxidase-peroxidase (GOD-PAP, Allemagne). The insulin level was measured by method Elisa, insulin resistance index is calculated by using homeostasis model assessment (HOMA = glucose (mmol/L) x insulin (µU/mL)/22,5) [37]. Glycosylated hemoglobin was determined by ion exchange chromatography (Spinreact, Allmagne). The blood pressure was measured by a Sphygmomanometer artery (PIC, Italie).

Lipid panel parameters

Total cholesterol level (T-Chol) was determined by the colorimetric enzymatic method with cholesterol esterase, cholesterol oxidase and peroxidase (Biocon, Allmagne). The Triglyceride level (TG) was quantified by colorimetric enzymatic method with lipase, glycerokinase and glycerophosphate oxidase (Biocon, Allmagne). The phospholipid level (PL) was determined by colorimetric enzymatic method with phospholipase D, choline oxidase and peroxidase (Biocon, Allmagne). Very low density lipoprotein (VLDL) and low density lipoprotein (LDL) are precipitated by phosphotungstic acid in the presence of magnesium ions ($MgCl_2$). High density lipoprotein (HDL_2 and HDL_3) are precipitated by dextran sulfate in the presence of $MgCl_2$. The LDL-cholesterol (LDL-Chol), the HDL_2-cholesterol (HDL_2-Chol), the HDL_3-cholesterol (HDL_3-Chol), the VLDL-triglyceride (VLDL-TG) was determined by the same method as that used for the total cholesterol and triglyceride. The apolipoprotein A1 and apolipoprotein B level were quantified by immunoturbidimetric method (Randox, Antrim, UK).

Renal function tests and inflammatory markers

The fibrinogen level was determined by the enzymatic method with thrombin (Cypress, Belgique). The C protein reactive level (CRP) was quantified by the immunoturbidimetric method (Biolabo, France). The urea level was determined by the kinetic enzymatic technique with urease-GLDH (Biocon, Allmagne). The uric acid level was determined by the enzymatic technique with uricase-peroxidase (Biocon, Allmagne). Albumin was determined by a colorimetric method with bromocresol green (Biolabo, France). The creatinine level was quantified by the reaction of Jaffé with alkaline picrate (Biocon, Allmagne). The creatinine clearance (CC) is calculated according to the formula of Cockcroft: CC (ml/min) = 0,85 x (140–A) x W / 0,814 x serum creatinine (µmol/L) ; W : body weight (kg) ; A: age in years.

Oxidant status (plasma TBARS and LDL-TBARS)

The oxidatif stress is determined by reaction of chromogenic reagent (2-thiobarbituric acid) with malondialdehyde MDA at 95 °C, knoevenagel-type condensation to yield a chromophore whith absorbance at 532 nm [32]. LDL was isolated from humen plasma by selective precipitation with amphipatic polymers; the ability of LDL to form peroxides was assessed by mesuring thiobarbituric acid reactive substances (TBARS) after incubation with Ca^{2+} and H_2O_2.

Antioxidant status

Antioxidant enzymes

The total antioxidant status (TAS) in plasma was determined by a colorimetric enzymatic method with peroxidase (Randox, Antrim, UK). Erythrocyte superoxide dismutase activity (SOD) was measured by a colorimetric enzymatic method with xanthine oxidase (Ransod, Randox, Antrim, UK). The catalase activity and the glutathion peroxidase (GPX) were quantified by a colorimetric method (Ransel, Randox, Antrim, UK).

Vitamins and trace elements

Plasma α-tocopherol level was measured by high performance liquid chromatography (HPLC) using Kontron system, after hexane extraction, the chromatographic conditions are: water symmetry C_{18} column (3,9mm × 150 mm) (Waters, Milford, MA, États-Unis), the injection volume was 20 µl, the mobile phase was methanol 100 %, the flow rate was 0,9 ml/min, the retention time was 25 min and the wavelength was equal to 294nm. The plasma ascorbic acid level was determined by colorimetric method after stabilization of plasma with metaphosphoric acid, wavelength was equal to 465 nm. Plasma zinc and copper level were quantified by a colorimetric method; wavelength was equal to 560 and 580 nm respectively (Randox, Antrim, UK). plasma selenium (Se^{2+}) was measured by high performance liquid chromatography (HPLC) using Kontron system, after cyclohexane extraction, the chromatographic conditions are: water symmetry C_{18} column (3,9 mm × 150 mm) (Waters, Milford, MA, États-Unis), the injection volume was 2 ml, the mobile phase (90 % cyclohéxane and 10 % ethyl acetate), the flow rate was 0,5 ml/min, the retention time was 2 min.

Statistical analysis

The comparison between the two groups (patients *vs* controls) was done using paired Student's-*t* test, the results are expressed on average ± standard deviation and a significance level of $p < 0.05$ was used.

RESULTS AND DISCUSSION

Anthropometric parameters and basic metabolic rate

The anthropometric parameters and basic metabolic rate are reported in Table 1. Significant differences were noted between patients and controls for body weight (80.03 ± 8.64 vs 67.18 ± 4.71 Kg), BMI (30.87 ± 4.97 vs 24.98 ± 2.76 Kg/m^2), waist circumference (104.16 ± 10.58 vs 79.61 ± 4.74 cm) and waist-hip ratio (0.95 ± 0.03 vs 0.84 ± 0.04). But, no significant difference was found for basic metabolic rate.

Table 1:Anthropometric parameters and basic metabolic rate of the studied population

Population Parameters	MetS *X* ± SD	Controls *X* ± SD
Body weight (Kg)	80.03 ± 8.64*	67.18 ± 4.71
Height (m)	1.61 ± 0.09	1.64 ± 0.07
BMI (Kg/m^2)	30.87 ± 4.97*	24.98 ± 2.76
Waist circumference (cm)	104.16 ± 10.58*	79.61 ± 4.74
Hip circumference (cm)	109.64 ± 8.01*	94.77 ± 8.52
Waist-hip ratio	0.95 ± 0.03*	0.84 ± 0.04
Basic metabolic rate (MJ)	6.32 ± 0.39	5.95 ± 0.21

*MetS, metabolic syndrome ; BMI, body mass index ; MJ, Megajoule ; X, average ; SD, standard deviation ; *, p < 0.05.*

The results of anthropometric parameters show that patients have android obesity (BMI > 30 Kg/m2 and waist-hip ratio ≥ 0,95) [42, 47], also, the increase in body weight of patients is the result of accumulation of abdominal fat [30] and white adipose tissue presents only 2% of resting energy expenditure, That is why, there is no difference between the two populations concerning the BMR [7].

Daily food ration

Table 2 shows daily food ration. The daily total energy intake of patients (9.14 ± 0.79 MJ) is significantly higher compared to controls (8.11 ± 0.81 MJ), also, food consumption assessment shows a significant increase of: simple carbohydrates by 50%, fats by 24%, saturated fatty acids (SFA) by 131%, polyunsaturated fatty acids (PUFA) by 34%, trans fatty acids (TFA) by 131%, dietary cholesterol by 54%, sodium by 39%, iron intake by 50% and n-6/n-3 fatty acid ratio by 152% in patients compared with controls. However, the diet study indicates a significant decrease of: animal protein by 33%, Monounsaturated fatty acids (MUFA) by 23%, calcium by 23%, vitamin C by 25% and vitamin E intake by 41% in patients compared with controls. No significant difference was evident in complex carbohydrates, proteins and vegetable proteins between the two groups.

Table 2: Daily food ration of the studied population

Population Parameters	MetS $X \pm SD$	Controls $X \pm SD$
Total energy intake (MJ/d)	9.14 ± 0.79*	8.11 ± 0.81
Carbohydrates (g/d)	1006.02 ± 176.32*	873.45 ± 151.60
Simple carbohydrates (g/d)	342.05 ± 73.62*	227.10 ± 47.81
Complex carbohydrates (g/d)	663.10 ± 103.37	646.35 ± 119.90
Proteins (g/d)	284.31 ± 66.51	349.38 ± 59.36
Vegetable proteins (g/d)	184.80 ± 72.13	199.15 ± 69.33
Animal proteins (g/d)	99.48 ± 31.07*	150.23 ± 42.51
Fats (g/d)	896.67 ± 82.15*	718.17 ± 63.97
SFA (g/d)	349.70 ± 87.63*	150.82 ± 91.47
MUFA (g/d)	286.93 ± 66.00*	373.45 ± 89.09
PUFA (g/d)	260.03 ± 78.03*	193.90 ± 90.07
TFA (g/d)	76.54 ± 12.79*	38.82 ± 21.09
n-6/n-3 fatty acid ratio	9.28 ± 3.70*	3.68 ± 01.03
Dietary cholesterol (mg/d)	397.85 ± 77.03*	257.32 ± 51.86
Fibers (g/d)	33.02 ± 14.90	26.79 ± 08.55
Na^{+} (mg/d)	4461.32 ± 165.79*	3195.74 ± 179.06
Ca^{2+} (mg/d)	576.71 ± 173.81*	749.44 ± 87.3
Fe^{2+} (mg/d)	17.78 ± 5.57*	11.79 ± 2.38
Vitamin C (mg/d)	92.19 ± 25.59*	123.17 ± 2.12
Vitamin E (mg/d)	08.27 ± 4.33*	14.04 ± 03.78

*MetS, metabolic syndrome ; MJ, Megajoule ; d, day ; X, average ; SD, standard deviation ; *, $p < 0.05$.*

In patients, the total energy intake and the consumption of carbohydrate and fats were higher than controls, these results agree with those of Park et al. (2003) [42] equally, the high glycemic index of simple carbohydrates is the cause of hyperglycemia characteristic of the metabolic syndrome [5], but the animal protein intake of sick women was lower than a healthy women, several studies [10] have shown the benefits of fish protein (sardine) in lowering blood pressure. The diet study shows a increase in the intake of saturated fatty acids, trans fatty acids, n-6 / n-3 fatty acid ratio and dietary cholesterol in patients compared to controls, however a reduction of taking polyunsaturated fatty acids, according to Yki-Järvinen et al. (2005) [57] taking saturated fatty acids like myristic acid and trans fatty acids promotes the increase of triglyceridemia and according to Schaefer (2002) [48] a high ratio of dietary n-6/n-3 fatty acid induces decreased HDL-Chol, Also Lichtenstein et al. (2006) [34] showed that increasing dietary cholesterol intake induced increase in LDL-Chol [55]. The patients consume more sodium compared to controls, this cation that causes increased blood pressure [25]. Also, this study shows a significant increase in iron consumption while the intake of calcium, vitamin C and E is insufficient in patients compared to controls, according to Levesque (2006) [33], iron can become pro-oxidant (fenton reaction), the iron-salt-dependent decomposition of dihydrogen peroxide (H_2O_2), generating the highly reactive hydroxyl radical ($OH^{\cdot}$), Ascorbic acid is a scavenger of free radicals and the reduction in intake of vitamin C induces oxidative stress. Calcium regulates energy metabolism in adipocytes and the consumption of dairy products fight against the installation of abdominal obesity [38].

Glycemic parameters and blood pressure

Glycemic parameters and blood pressure are reported in Table 3. The results indicate significant increase of blood glucose by 144%, of glycosylated hemoglobin by 71%, of insulin by 75%, of insulin resistance index (HOMA) by 331% and of systolic blood pressure (SBP) by 25% in patients compared to controls. But, there was no significant difference in diastolic blood pressure (DBP) between the two groups.

Table 3: Glycemic parameters and blood pressure of the studied population

Population Parameters	MetS $X \pm SD$	Controls $X \pm SD$
Glycemia (mmol/l)	13.8 ± 5.83*	5.65 ± 0.53
Insulin (µU/mL)	4.93 ± 1.12*	2.81 ± 0.97
HOMA	3.02 ± 1.57*	0.7 ± 0.37
HbA1c (%)	10.51 ± 1.35*	6.12 ± 1.2
SBP (mm Hg)	13.93 ± 1.05*	11.09 ± 1.75
DBP (mm Hg)	7.53 ± 1.27	6,87 ± 0.94

*MetS, metabolic syndrome ; HOMA, Homestasic Model Assessment score ; HbA1c, glycosylated hemoglobin ; X, average ; SD, standard deviation ; SBP, systolic blood pressure ; DBP, diastolic blood pressure ; *, $p < 0.05$.*

The results indicate a significant increase in blood glucose and glycosylated hemoglobin in patients compared to controls, so the patients would be exposed to emergence of degenerative complications of hyperglycemia [46]. Also the analyzes show a significant increase in insulin and insulin resistance index (HOMA), so our population has insulin resistance, this results agree with those of Matsuzawa 2005. In fact, insulin resistance is an overabundance of circulating free fatty acids (FFA), released from an expanded adipose tissue mass. FFA reduces insulin sensitivity in muscle by inhibiting insulin-mediated glucose uptake. Increased level of circulating glucose increases pancreatic insulin secretion resulting in hyperinsulinemia [31]. On the other hand, we note a significant increase in systolic blood pressure, In the setting of insulin resistance, the vasodilatory effect of insulin can be lost and free fatty acids themselves can mediate relative vasoconstriction [56].

Lipid panel parameters

Table 4 shows lipid panel parameters. We note a significant increase of: triglycerides by 132%, VLDL-TG by 204%, T-Chol by 30%, LDL-Chol by 32%, HDL_3-Chol by 85%, total cholesterol/ HDL_2-cholesterol ratio (T-Chol / HDL_2-Chol ratio) by 214% and Apolipoprotein B levels by 50%. However, the results show a significant decrease of: HDL_2-Chol by 43%, phospholipids by 29% and Apolipoprotein A1 levels by 33% in patients compared to control subjects.

Table 4:Lipid panel parameters of the studied population

Population Parameters	MetS *X* ± SD	Controls *X* ± SD
Triglycerides (mmol/l)	1.81 ± 0.58*	0.78 ± 0.31
VLDL-TG (mmol/l)	0.7 ± 0.16*	0.23 ± 0.09
T-Chol (mmol/l)	4.36 ± 0.27*	3.34 ± 0.19
LDL-Chol (mmol/l)	2.02 ± 0.51*	1.53 ± 0.43
HDL_2-Chol (mmol/l)	0.32 ± 0.08*	0.57 ± 0.12
HDL_3-Chol (mmol/l)	0.63 ± 0.17*	0.34 ± 0.06
Chol-T/ HDL_2-Chol ratio	13.62 ± 2.23*	4.33 ± 1.72
Phospholipids (mmol/l)	2.75 ± 0.62*	3.92 ± 0.95
Apolipoprotein A1 (g/l)	0.87 ± 0.06*	1.31 ± 0.10
Apolipoprotein B (g/l)	0.96 ± 0.13*	0.64 ± 0.07

*MetS, metabolic syndrome ; X, average ; SD, standard deviation ; *, p < 0.05.*

We note a significant increase in triglycerides level and VLDL-TG in patients compared to controls [11]. Several studies Heidemann et al. (2011) [24], have shown that hypertriglyceridaemia is correlated with the consumption of simple carbohydrates, in the setting of metabolic syndrome, increased flux of free fatty acids to the liver increases hepatic triglyceride synthesis and production of very low-density lipoproteins (VLDL) occurs [19]. Hypercholesterolaemia observed in patients is related to the increase in LDL-Chol and HDL_3-Chol level [21], but the HDL_2-Chol level is decreased, according to Bouchard-Mercier et al. (2010) [9]; Huesca-Gum et al. (2004) [27] the increase in HDL_3-Chol level and the reduction in HDL_2-Chol level is explained by the lecithin cholesterol acyltransferase (LCAT) deficiency which is a disorder of the transformation of the free cholesterol of the HDL_3 into cholesterol esters of the HDL_2. The Increasing of atherogenic index (T-Chol / HDL_2-Chol ratio) in patients compared to controls, increases the risk of developing cardiovascular disease [54]. The results show the decrease in phospholipids level, according to Ji et al. (2006) [28] the reduction of phospholipid transfer protein (PLTP) activity and LCAT deficiency, decrease the use of phospholipids to esterify the cholesterol of the HDL_2. Apolipoprotein A1 level is significantly reduced in patients compared to control subjects, Apolipoprotein A1 has major role is centripetal movement of cholesterol from peripheral tissues including the arterial wall to the liver, low levels of this proteins have been identified as the risk factor in the development and progression of coronary damage. In our study, Apolipoprotein B level was found to be significantly high in patients compared to control subjects. These observations coincide with the findings several studies related to coronary heart disease [50].

Table 5: Renal function tests and inflammatory markers of the studied population

Population Parameters	MetS *X* ± SD	Controls *X* ± SD
Urea (mmol/l)	10.11 ± 5.42*	4.91 ± 0.37
Uric acid (µmol/l)	341.6 ± 92.07*	229.8 ± 54.63
Creatinine (µmol/l)	159.01 ± 47.53*	71.11 ± 13.17
CC (ml/min)	38.00 ± 17.57*	147.39 ± 57.61
Albumin (g/l)	35.94 ± 7.13*	55.17 ± 4.83
Fibrinogen (g/l)	2.07 ± 0.44*	1,43 ± 0.29
CRP (g/l)	2.47 ± 1.17*	0.97 ± 0.02

*MetS, metabolic syndrome ; CC, creatinine clearance ; CRP, C reactive protein ; X, average ; SD, standard deviation. *, p < 0.05.*

Renal function tests and inflammatory markers

Renal function tests and inflammatory markers are reported in Table 5. Our results indicate an increase of: urea by 105%, uric acid by 48%, creatinine by 123%, fibrinogen by 44% and C reactive protein levels by 154%. But, the creatinine clearance and albumin level were 3.87-fold and 1.53-fold lower in patients compared to controls.

Our results show an increase in urea, creatinine and uric acid levels [18]. Also there is a decrease in creatinine clearance and albumin levels, these results show that the patients are at risk of kidney failure (nephropathy) [12], in fact, hyperglycemia play a role in the development of nephropathy include advanced glycosylation end products (AGEs) [53]. Statistical analysis of inflammatory markers, shows a significant increase in fibrinogen and C reactive protein (CRP) levels in patients compared to controls [53], according to Schneider (2005) [49] high levels of fibrinogen are associated with obesity, hypercholesterolemia and a risk of coronary ischemia, on the other hand, the increase of the CRP levels is correlated with atherosclerosis (Koenig 2005) [29], in fact, CRP can bind to LDL and the complex CRP / LDL activates the phagocytic function of macrophages, the origin of the foam cells during the development of atheroma [58].

Oxidant status

Table 6 shows the plasma TBARS and LDL-TBARS level. Our study shows that the plasma TBARS and LDL-TBARS levels were, respectively, 1.76-fold and 2.77-fold higher in patients compared to controls.

Table 6: Plasma TBARS and LDL-TBARS level of the studied population

Population Parameters	MetS $X \pm SD$	Controls $X \pm SD$
Plasma TBARS (µmol/l)	31.1 ± 8.12*	17.60 ± 6.08
LDL-TBARS (µmol/l)	13.2 ± 4.05*	4.76 ± 1.8

*MetS, metabolic syndrome ; LDL-TBARS, thiobarbituric acid reactive substances associated with LDL ; X, average ; SD, standard deviation ; *, $p < 0.05$.*

Plasma TBARS and LDL-TBARS level are increased in patients compared to controls [26], according to Pou et al. (2007) [44], plasma TBARS level increase with increase in BMI, effectively, the visceral adipose tissue contributes to the production of reactive oxygen species, these toxic elements induces endothelial dysfunction and inhibits the secretion of insulin [17], also, the increase in LDL-TBARS shows that peroxidation of LDL is important favoring their catabolism by macrophages, this would imply them in the formation of atherosclerotic plaque [13].

Antioxidant status

Antioxidant enzymes

The antioxidant enzymes activities are reported in Table 7. The results indicate a reduction by 30% of the total antioxidant status (TAS), by 23% of the superoxide dismutase activity (SOD), by 15% of the catalase activity and by 22% of the glutathion peroxidase activity (GPx) in patients compared to controls.

Table 7: Antioxidant enzymes activities of the studied population

Population Parameters	MetS $X \pm SD$	Controls $X \pm SD$
TAS (mmol/l)	1.77 ± 0.78*	2.53 ± 0.51
SOD (UI/l Hb)	148.91 ± 55.10*	194.11 ± 64.78
Catalase (UI/l Hb)	2.86 ± 0.12*	3.40 ± 0.17
GPx (UI/l blood)	3267.27 ± 371.13*	4193.04 ± 409.65

*MetS, metabolic syndrome ; HB, hemoglobin ; X, average ; SD, standard deviation ; *, $p < 0.05$.*

The results shows a reduction in antioxidant enzymes activities in patients compared to controls, which confirms the presence of an intense oxidative stress in patients [3], according to Olusi (2002) [40] the superoxyde dismutase activity and the glutathion peroxydase activity is lower in people with BMI> 30 (Kg/m^2) and in this study all patients are obese, in the same way, the reduction in the antioxidant enzyme activity would be explained by the increase in their glycation by glucose especially when the patients have an hyperglycemia [15]. Also, the reduction in the activity of antioxydant would be explained by a deficiency of the availability in NADPH, H [39].

Vitamins and trace elements

Table 8 shows the vitamins and trace elements values. Our study indicates that the α-tocopherol, ascorbic acid, Zn^{2+}, Cu^{2+}, and Se^{2+} levels were, respectively, 1.83-fold, 1.32-fold, 1.33-fold, 1.30-fold and 1.25-fold lower in patients compared to controls.

Table 8: Vitamins and trace elements of the studied population

Population Parameters	MetS $X \pm SD$	Controls $X \pm SD$
α-tocopherol (µmol/l)	17.36 ± 6.54*	31.82 ± 5.93
Ascorbic acid (µmol/l)	41.07 ± 11.43*	54.59 ± 9.17
Zn^{2+} (µmol/l)	15.38 ± 3.12*	20.51 ± 5.27
Cu^{2+} (µmol/l)	13.94 ± 4.02*	18.20 ± 3.83
Se^{2+} (µmol/l)	0.84 ± 0.14*	1.05 ± 0.18

*MetS, metabolic syndrome ; X, average ; SD, standard deviation ; *, $p < 0.05$.*

We have noted a decrease in α-tocopherol and ascorbic acid levels, in patients compared to controls [38], according to Palmieri et al. (2006) [41] this will be caused by excessive consumption of vitamin E to participate against oxidative attack, also, Vitamin C plays a major role in the regeneration of vitamin E [15]. We have found a significant decrease in the Zn^{2+}, Cu^{2+} and Se^{2+}, according to Marcason (2008) [35] overweight, hypertension and insulin resistance are associated to a low plasma selenium levels, also, Quilliot et al. 2001 [45] showed the presence of a high urinary excretion of copper and zinc with a decrease in plasma concentration in patients with metabolic syndrome, causing reduction in the superoxyde dismutase activity.

CONCLUSION

This study has shown an imbalance in the nutritional status of Algerian women with metabolic syndrome. The food intake of patients was characterized by low dietary intake of polyunsaturated fatty acids, calcium, vitamin E and C, but higher in simple carbohydrates, saturated fatty acids, sodium and iron, this dietary imbalance is associated with elevated BMI, waist circumference and waist-hip ratio.

Our work has confirmed the presence of oxidative stress. While total antioxidant status, superoxyde dismutase activity, catalase activity and glutathion peroxydase activity and plasma level of vit E, vit C, zinc, copper, and selenium were significantly reduced. The insulin levels, the insulin resistance index (HOMA) and concentration of thiobarbituric acid reactive substances related to LDL were increased. Also this study indicated a significant increase in plasma fibrinogen and CRP levels.

At the end of this work, we propose to investigate new biomarkers of metabolic syndrome like adipokines (leptin, adiponectin, resistin and Visfatin) which represent indicators of appearance of serious complications, in future. For this, the adaptation of a healthy lifestyle by increasing physical activity, weight loss and maintaining a diet rich in plant foods, antioxidants and fiber as Mediterranean diet could provide for the installation of metabolic syndrome.

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