

# Hormonal and Biochemical Parameters of Metabolic Syndrome in Male Patients with Body Weight Excess and Obesity

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Developed form of metabolic syndrome was revealed in 64.3% men with grade 1 obesity and in 81.8% men with grade 2-3 obesity (patients of the therapeutic clinic). The major components were dyslipidemia, fatty hepatosis, arterial hypertension, and hyperinsulinemia. In patients with excess body weight, changes in some biochemical parameters also attested to the presence of some components of the metabolic syndrome, but purine metabolism disorders were more frequently observed in this group than hyperinsulinemia.

**Key Words:** *hormonal and biochemical parameters; metabolic syndrome components*

Abdominal obesity is accompanied by dyslipoproteinemia, hyperglycemia, hyperinsulinemia, insulin resistance, hyperuricemia, and other metabolic disturbances that contribute to fat accumulation [5,6, 8,12]. Carbohydrate metabolism disorders, arterial hypertension, abdominal obesity, dyslipidemia, and other pathological changes are components of the metabolic syndrome (MS). MS significantly increases the risk of coronary heart disease and cerebrovascular disorders [1,2,4,11]. The liver is one of the major targets of atherogenic dyslipidemia. Some authors believe that nonalcoholic fatty liver disease (*e.g.*, fatty hepatosis) is a component of MS [3,10].

A close pathogenetic relationship exists between obesity and MS. Here we studied hormonal and biochemical criteria of MS and several components of this syndrome in male patients with excess body weight and obesity of various degrees.

## MATERIALS AND METHODS

We examined 105 male patients (average age  $46.0 \pm 11.1$  years). The body mass index (BMI, the weight in kilograms divided by the square of the height in meters) was  $25 \text{ kg/m}^2$  or more. They were admitted to the therapeutic clinic for cardiovascular abnormalities (61%), gastrointestinal disturbances (16%), musculoskeletal disorders (9%), and other diseases (14%). Informed consent for participation in the trial was obtained from each patient.

The patients were divided into 3 groups by BMI: group 1, patients with excess body weight ( $\text{BMI}=25.0\text{--}29.9 \text{ kg/m}^2$ ,  $n=27$ ); group 2, patients with grade 1 obesity ( $\text{BMI}=30.0\text{--}34.9 \text{ kg/m}^2$ ,  $n=56$ ); and group 3, patients with grade 2-3 obesity ( $\text{BMI}>35 \text{ kg/m}^2$ ,  $n=22$ ). The absolute content of adipose tissue was measured by impedancemetry using an OMRON device. The distribution of the adipose tissue was estimated from the waist/hip ratio.

Blood samples were obtained from fasting patients in the morning. Biochemical study included measurement of glucose, total cholesterol (CH), triglycerides, high-density lipoprotein (HDL) CH, and uric acid in blood serum. Activities of aspartate transaminase (AST) and alanine transaminase (ALT)

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were estimated. Biochemical parameters of the serum were studied enzymatically on a Photometer 5010 device (Boehringer Mannheim). The concentrations of immunoreactive insulin and C-peptide in the serum were measured using radioimmune kits (IRMAZENco INSULIN and RIAZENco C-PEPTIDE, respectively; Zen Tech). The degree of insulin resistance was evaluated from the HOMA index. The HOMA index was calculated as follows:

fasting plasma insulin (U/ml) × fasting plasma glucose (mmol/liter) / 22.5 [7].

The results of hormonal and biochemical studies, clinical examination of patients, and liver ultrasonography were used to estimate the incidence of the following MS components [9]:

- hyperinsulinemia (insulin level > 16 U/ml);
- carbohydrate metabolism disorders (fasting hyperglycemia, glucose level 5.6–6.7 mmol/liter; impaired glucose tolerance in the standard glucose tolerance test or postprandial glycemia > 6.7 mmol/liter; type 2 diabetes mellitus);
- dyslipidemia (plasma triglyceride level ≥ 1.7 mmol/liter, HDL CH level < 1.0 mmol/liter);
- abdominal obesity (waist/hip ratio ≥ 1.0);
- arterial hypertension (systolic blood pressure ≥ 140 mm Hg and/or diastolic blood pressure ≥ 90 mm Hg in repeated measurement; hypotensive therapy);
- purine metabolism disorders (hyperuricemia, uric acid level ≥ 420 μmol/liter; gout);

- fatty hepatosis (increase in AST and ALT activity by more than 40 U/liter; ultrasonic characteristics).

The presence of developed MS was taken into account in patients with at least 4 components.

Intergroup differences were evaluated by parametric (analysis of variance, Student's *t* test) and nonparametric methods ( $\chi^2$  test). The differences were significant at  $p < 0.05$ .

## RESULTS

Intergroup differences were revealed in BMI (Table 1). The increase in BMI was accompanied by an increase in the total amount of adipose tissue and waist/hip ratio. These changes reflect an increase in the content of visceral fat.

Increasing the body weight was accompanied by an increase in the contents of insulin and C-peptide in the blood. It should be emphasized that these parameters significantly differed between various groups. Fasting glucose level also increase, but intergroup differences were insignificant due to high scatter of individual values of this parameter in group 3. The NOMA index progressively increased from group 1 patients to group 3 patients, which attested to increasing severity of insulin resistance.

Serum activities of ALT and AST increased with increasing BMI. The increase in enzyme activities was most pronounced in patients with grade 2–3 obesity.

**TABLE 1.** Anthropometric, Hormonal, and Biochemical Parameters of Male Patients with Excess Body Weight and Obesity ( $M \pm m$ )

Parameter	Group		
	1	2	3
BMI, kg/m <sup>2</sup>	28.0 ± 0.2	32.4 ± 0.2**	39.5 ± 1.1***
Adipose tissue, kg	21.7 ± 0.7	30.2 ± 0.6**	41.5 ± 1.4***
Waist/hip ratio	0.95 ± 0.01	1.00 ± 0.01**	1.03 ± 0.02***
Immunoreactive insulin, U/ml	9.6 ± 0.8	15.3 ± 1.1**	24.0 ± 6.1**
C-peptide, pmol/liter	1.16 ± 0.07	1.61 ± 0.06**	2.12 ± 0.31**
Glucose, mmol/liter	5.09 ± 0.36	5.52 ± 0.23	6.17 ± 0.63
HOMA index	1.94 ± 0.20	3.70 ± 0.20*	4.83 ± 1.05**
AST, U/liter	28.7 ± 2.3	30.1 ± 1.9*	38.5 ± 4.6*
ALT, U/liter	37.7 ± 4.5	47.0 ± 3.9	62.6 ± 9.6**
Total CH, mmol/liter	6.33 ± 0.24	6.09 ± 0.18	6.04 ± 0.15
Triglycerides, mmol/liter	2.59 ± 0.29	3.05 ± 0.28	2.68 ± 0.34
HDL CH, mmol/liter	1.24 ± 0.07	1.11 ± 0.04	1.010 ± 0.053*
Uric acid, μmol/liter	364 ± 18	381 ± 15	389 ± 29

**Note.** \* $p < 0.05$  and \*\* $p < 0.01$  compared to group 1; \*\*\* $p < 0.01$  compared to group 2.

**TABLE 2.** Incidence of MS Components in Male Patients with Excess Body Weight and Obesity (%)

MS Component	Group		
	1	2	3
Hyperinsulinemia	0	68.4	71.4
Carbohydrate metabolism disorders	11.1	28.6	36.4
Dyslipidemia	77.8	80.4	90.9
Abdominal obesity	18.5	50.0*	72.7*
Arterial hypertension	59.3	76.8	81.8
Purine metabolism disorders	37.0	41.1	45.5
Fatty hepatosis	61.5	89.1*	90.5*
Developed MS	22.2	64.3**	81.8**

**Note.** \* $p < 0.01$  and \*\* $p < 0.001$  compared to group 1.

The content of total CH and triglycerides in blood serum remained unchanged with increasing BMI. HDL CH level in patients with grade 2-3 obesity was much lower than in group 1 patients. The concentration of uric acid in blood serum remained unchanged in patients of all groups.

Obesity is the major risk factor for MS. It could be expected that the increase in adipose tissue mass would result in a similar increase in major biochemical parameters of MS. However, some biochemical parameters underwent significant changes with transition from excess body weight to obesity. The greatest changes in other parameters were observed with the transition from grade 1 obesity to grade 2-3 obesity. Some parameters, including the concentrations of total CH and triglycerides in blood serum, remained unchanged under these conditions. It should be emphasized that the mean values of these parameters in patients of all groups were above normal.

The incidence of MS components was evaluated from the results of hormonal and biochemical studies and clinical and instrumental examination. Dyslipidemia, fatty hepatosis, and arterial hypertension were most frequently observed. The incidence of carbohydrate metabolism disorders was minimum (Table 2). Developed MS was diagnosed not only in individuals with obesity, but also in patients with excess body weight (22.2%). The patients with developed MS and excess body weight were primarily characterized by a combination of dyslipidemia, fatty hepatosis, arterial hypertension, and purine metabolism disorders. Dyslipidemia,

fatty hepatosis, arterial hypertension, and hyperinsulinemia were found in patients with various degrees of obesity. Our results indicate that hormonal and metabolic criteria of MS should be evaluated not only in obesity patients, but also in individuals with BMI of 25 kg/m<sup>2</sup>. This approach will decrease the risk for the development and progression of MS and fatal complications of this disorder, including cardiovascular and liver diseases.

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