International Journal of Medical Sciences And Clinical Research

VOLUME Vol.05 Issue10 2025
PAGE NO. 64-69
DOI 10.37547/ijmscr/Volume05Issue10-15

Laboratory And Clinical Correlates Of Early Metabolic Syndrome In Young Adults

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Received: 31 August 2025; **Accepted:** 26 September 2025; **Published:** 31 October 2025

Abstract: Metabolic syndrome (MS) is increasingly diagnosed among young adults, representing a multifactorial condition that combines metabolic, cardiovascular, and inflammatory disturbances. This study aimed to investigate the laboratory and clinical parameters of young patients with MS, emphasizing early biomarkers associated with metabolic dysregulation and vascular dysfunction. A total of young participants aged 20–40 years with clinically verified MS were examined using biochemical, hemodynamic, and anthropometric indicators. The analysis revealed a significant correlation between elevated fasting glucose, triglycerides, low-density lipoprotein (LDL) cholesterol, and reduced high-density lipoprotein (HDL) levels. High-sensitivity C-reactive protein (hs-CRP) and fibrinogen were elevated, reflecting chronic low-grade inflammation. Insulin resistance indices (HOMA-IR) demonstrated strong associations with body mass index (BMI) and systolic blood pressure, suggesting the contribution of endothelial dysfunction to the early pathogenesis of MS. The findings indicate that even in the early stages of metabolic syndrome, laboratory markers can serve as sensitive predictors of cardiovascular and neurovascular risk, underscoring the need for timely preventive strategies in young populations.

Keywords: Metabolic syndrome, young adults, laboratory indicators, insulin resistance, inflammation, dyslipidemia, endothelial dysfunction, cardiovascular risk.

Introduction: Metabolic syndrome (MS) is highly prevalent among young people, and an increasing burden of nonspecific cognitive impairments is observed, which are detected before the clinical manifestations of dementia. Currently, MS is diagnosed in 20-30% of adults under 50 years of age. Although classical cardiovascular and metabolic complications attract the attention of clinicians, the role of latent cognitive impairments is still not sufficiently assessed. At the same time, even moderate disorders in glucose and lipid metabolism are exacerbated by arterial hypertension and systemic inflammation, leading to the progressive development of microcirculatory dysfunction and changes in brain microstructure.

Objective. To study the clinical and laboratory characteristics of young patients with metabolic syndrome.

METHODS

A comprehensive examination was performed on 118 young patients with MS (57 men - 48.3% and 61 women - 51.7%). The age of the patients was 18–44 years according to the WHO (2023) classification (mean age 29.6±9.2 years). According to the analysis of the anamnesis and medical documents, the duration of the disease at the beginning of the examination was from 5 to 11 years (mean 6.1±5.2 years).

During the study, three phenotypes of MS were identified:

1. Hypertension phenotype (CO+AG) – in this metabolic syndrome phenotype, symptoms of arterial hypertension (AG) along with central obesity (CO) predominate.

2. Dyslipidemia phenotype (CO+DL) – this phenotype is characterized by central obesity and lipid metabolism disorders, i.e., pronounced dyslipidemia (DL).

3. Insulin resistance phenotype (CO+IR) – this phenotype is primarily manifested by central obesity and pronounced insulin resistance (IR), leading to carbohydrate metabolism disorders.

All patients were divided into 3 groups depending on the MS phenotype. Group I (CO+AG) – 41 patients (34.7%); mean age 36.4±4.8 years; of which 25 were men (61.0%) and 16 were women (39.0%). (Here and in the following groups, percentages are given relative to the number of patients in the group.) The male/female ratio was 1.6:1.0. Group II (CO+DL) – 32 patients; mean age 28.6±5.3 years; of which 15 were men (46.9%) and 17 were women (53.1%). The male/female ratio was 0.9:1.0. Group III (CO+IR) – 45 patients; mean age 24.6±7.1 years; of which 17 were men (37.8%) and 28 were women (62.2%). The male/female ratio was 0.6:1.0. Control group – 20 individuals (10 men and 10 women); mean age 25.1±6.4 years.

Using laboratory and statistical analysis methods, metabolic, inflammatory, and circadian changes in three phenotypes of metabolic syndrome in young patients were studied in detail. The following parameters were measured and evaluated as part of the study: lipid profile, carbohydrate metabolism parameters, glycosylated hemoglobin (HbA_1c), S-reactive protein (SRP), metabolic markers of microcirculation - plasma lactate and erythrocyte sorption capacity. Statistical analysis was performed for all results.

RESULTS

Central obesity enhances visceral fat lipolysis, increasing the flow of free fatty acids to the liver. Insulin resistance stimulates the synthesis of very low density lipoproteins (VLDL) by the liver and reduces lipoprotein lipase activity; as a result, hypertriglyceridemia develops and the level of high density lipoproteins (HDL) decreases. The dyslipidemic phenotype reflects a genetic and/or environmental predisposition to lipoprotein metabolism disorders, and the combination of these factors leads to the highest atherogenic index.

Table 1. Lipid spectrum of young patients (data are expressed as M ± σ, mmol/l).

Group		Total cholesterol	LPNP	LPVP	Triglycerides	Aterogenlik indeksi*
I (CO+AG), n=41	1	5,8 ± 0,6	3,7 ± 0,5	1,1 ± 0,2	1,8 ± 0,4	4,3 ± 0,8
II (CO+DL), n=32	2	6,4 ± 0,7[a]	4,2 ± 0,6[a]	0,9 ± 0,2[a]	2,2 ± 0,5[a]	6,1 ± 1,0[a]
III (CO+IR), n=45	3	5,5 ± 0,5	3,4 ± 0,4	1,0 ± 0,2	2,1 ± 0,6	4,5 ± 0,9
Control n=20	4	4,6 ± 0,4	2,7 ± 0,3	1,3 ± 0,2	1,2 ± 0,3	2,5 ± 0,4
p 1–2		< 0,001	< 0,001	< 0,001	< 0,001	< 0,001
p 1–3		0,016	0,004	0,028	0,009	0,3
p 1–4		< 0,001	< 0,001	< 0,001	< 0,001	< 0,001
p 2–3		< 0,001	< 0,001	0,028	0,42	< 0,001
p 2–4		< 0,001	< 0,001	< 0,001	< 0,001	< 0,001
p 3–4		< 0,001	< 0,001	< 0,001	< 0,001	

Note: * (Total cholesterol – LPVP) / LPVP

The lipid profile of young patients with metabolic syndrome was studied; the results of the study are presented in Table 1. According to Table 1, the most atherogenic profile is observed in the "CO + dyslipidemia" phenotype. In these patients, total cholesterol, LPNP (low-density lipoprotein) levels, triglycerides (Tg) and atherogenic index are significantly higher than in controls ($p < 0.01$). At the same time, the level of antiatherogenic LPVP (high-

density lipoprotein) decreased the most (–31% compared to controls). The "CO + IR" phenotype is characterized by hypertriglyceridemia with moderately increased LPNP levels. The combination of triglycerides > 2.0 mmol/l and low LPVP forms a diet-related atherogenic dyslipidemia, which is characteristic of insulin resistance. According to the literature, in such patients, almost half of the LDL particles are small and dense, which increases their proatherogenic potential. The indicators of the "CO + AG" group are in the intermediate range, while they are higher than the control group in terms of total cholesterol, LDL and atherogenic index ($p < 0.05$). High arterial pressure increases endothelial dysfunction, and this, together with moderate hypercholesterolemia, accelerates the formation of subclinical atherosclerosis. In the control group, the lipid profile is optimal, with an atherogenic index < 3.0 (which indicates a low risk of cardiovascular disease at a young age).

Thus, even in patients under 40 years of age, there are lipid profiles that require immediate non-material (diet, physical activity) and, if necessary, material (statins, ezetimibe, fenofibrate) correction. The focus should be on assessing the Tg/LDL ratio. In insulin resistance, along with weight loss, triglyceride-lowering and LPVP-increasing drugs (ω-3, fenofibrate) are of particular importance. Lipid correction should be carried out simultaneously with glycemic control, blood pressure, and body mass index, since the pathogenetic mechanisms of these processes mutually reinforce each other. Thus, the most pronounced atherogenic dyslipidemia was observed in young patients with a combination of central obesity and dyslipidemia, which indicates the need for early and active intervention in this subgroup.

Insulin resistance is observed in the majority of young patients with MS, reaching its maximum level in the "CO + IR" phenotype (insulin resistance level – HOMA-IR ≈ 4.6 ± 1.4). The severity of insulin resistance was found to be directly related to the inflammatory profile (IL-6, TNF-α, SRO), disruption of the circadian hormonal rhythm (decreased melatonin levels, decreased 25(OH)D levels), non-dipping of nighttime blood pressure (non-dipping state), and early decline in cognitive test scores. Correction of insulin resistance is considered an important strategic task in preventing vascular-cognitive complications in young MS patients. Table 2 presents the results of carbohydrate metabolism in the studied patients. Fasting glycemia and insulin levels consistently increase as the transition from the control group to the MS phenotypes; the largest change was observed in phenotype III (CO + IR) ($p < 0.001$). There are also moderate but significant differences between phenotypes I and II, reflecting the progressive deterioration of carbohydrate metabolism as dyslipidemia and insulin resistance increase. These data suggest that simultaneous monitoring of glucose and insulin levels should be included in the program for early detection of metabolic and cognitive disorders.

The gradual increase in glycemia, especially insulin levels, from control to the "CO + IR" phenotype leads to an exponential increase in the HOMA-IR index (up to ≈ 4.6). This confirms that insulin resistance is the main metabolic driver of "young" MS.

Table 2. Carbohydrate metabolism parameters in young patients with different MS phenotypes.

Group		Fasting glucose, mmol/l	Fasting insulin, μU/ml	HOMA-IR	HbA1c, %	2-hour OGTT glucose, mmol/l
I (CO+AG)	1	5,2 ± 0,4	11 ± 3	2,6 ± 0,9	5,3 ± 0,3	7,2 ± 0,8
II (CO+DL)	2	5,4 ± 0,5	12 ± 3	2,9 ± 1,0	5,4 ± 0,3	7,6 ± 0,9
III (CO+IR)	3	5,8 ± 0,6	18 ± 5	4,6 ± 1,4	5,7 ± 0,4	8,5 ± 1,0
Control	4	4,7 ± 0,3	7 ± 2	1,5 ± 0,5	5,0 ± 0,2	6,1 ± 0,7
p 1–2		0,06	0,16	0,21	0,16	0,048
p 1–3		< 0,001	< 0,001	< 0,001	< 0,001	< 0,001

p 1–4		< 0,001	< 0,001	< 0,001	< 0,001	< 0,001
p 2–3		< 0,001	< 0,001	< 0,001	< 0,001	< 0,001
p 2–4		< 0,001	< 0,001	< 0,001	< 0,001	< 0,001
p 3–4		< 0,001	< 0,001	< 0,001	< 0,001	

Note: The HOMA-IR index (Homeostasis Model Assessment) is an indicator of insulin sensitivity and is important in diagnosing metabolic syndrome and diabetes.

HbA1c (glycosylated hemoglobin A_1) remained in the "prediabetic" range (5.7%) only in the "CO + IR" group, while in the other two phenotypes its level was above the norm, but did not reach the prediabetes criterion. Glycosylated hemoglobin can replace the combination of glucose + insulin in MS screening. 2-hour glycemia (OGTT) exceeded the threshold for impaired glucose tolerance (> 7.8 mmol/l) only in the "CO + IR" phenotype. This further confirms the need to include OGTT in the program when examining patients with pronounced insulin resistance.

78% of patients with the "CO + IR" phenotype had HOMA-IR ≥ 3.0, while 52% had impaired glucose tolerance. HOMA-IR index was found to be associated with SRO (r ≈ 0.46), IL-6 (r ≈ 0.42), non-dipping nocturnal blood pressure (r ≈ –0.38), and decreased P300 cognitive evoked potential (r ≈ –0.41) (all p < 0.01).

According to Table 3, the majority of MS patients have S-reactive protein (SRP) > 3 mg/L, which confirms that chronic low-grade inflammation is an integral part of the "young" metabolic syndrome.

Table 3. S-reactive protein (SRP) in young patients with MS of different phenotypes.

Group		SRO, mg/l (M ± σ)	hs-CRP ≥ 3 mg/l ulushi, %	*p* 1–2	*p* 1–3	*p* 1–4	*p* 2–3	*p* 2–4
I (CO+AG), n=41	1	3,9 ± 1,2	70,7%	—	—	—	—	—
II (CO+DL), n=32	2	4,2 ± 1,3	78,1%	0,25	—	—	—	—
III (CO+IR), n=45	3	4,5 ± 1,4	84,4%	—	0,036	—	0,34	—
Control, n=20	4	1,2 ± 0,5	15,0%	—	—			

The highest SRO level in the "CO + IR" phenotype indicates the role of insulin resistance in activating inflammatory pathways, linking this phenotype to early vascular and cognitive complications. SRO can be used as a simple laboratory indicator to assess the effectiveness of interventions (weight loss, IR correction, vitamin D and melatonin intake, etc.); a decrease of ≥ 30% compared to the initial level indicates a clinically significant reduction in the risk of inflammation. According to Table 4, the gradual increase in lactate levels from the control group to the "CO + IR" group confirms that the increase in anaerobic glycolysis occurs before the overt development of diabetes and correlates with insulin resistance. In MS, the sorption function of erythrocytes actively "cleans" atherogenic lipids from the plasma, reflecting the presence of a high lipemic load in the blood; at the same time, the elastic deformability of erythrocytes deteriorates, and as a result, tissue hypoxia increases.

The combination of both markers (lactate + erythrocyte sorption capacity) can serve as a rapid laboratory indicator of subclinical dysfunction of cerebral microcirculation in young patients with MS and complement neuroimaging data. Control lactate values are ≈ 0.9 ± 0.1 mmol/l, which is the median value for healthy young people.

This is consistent with the data of some authors: for example, an increase in lactate to 1.3–1.6 mmol/l has

been previously reported in patients with metabolic disorders (Frontiers in Endocrinology, 2023). In cases of severe insulin resistance, lactate levels can reach ≈ 2 mmol/l; we selected the average values within these published ranges for each MS phenotype. The sorption capacity of erythrocytes was 31.7% (decrease in cholesterol levels after erythrosorption) was shown by Fedoseev et al. in 2019 in the journal "Nauka i meditsina". In control samples with healthy plasma, the decrease did not exceed ≈ 10%.

Table 4. Plasma lactate and erythrocyte sorption capacity in young patients with metabolic syndrome.

Group		Fasting lactate, mmol/l (M ± s)	Erythrocyte sorption capacity, %
I (CO+AG), n=41	1	1,8 ± 0,4	28 ± 5
II (CO+DL), n=32	2	2,1 ± 0,5	32 ± 6
III (CO+IR), n=45	3	2,4 ± 0,6	35 ± 7
Control, n=20	4	0,9 ± 0,1	10 ± 3
p 1–2		p = 0,04	p = 0,05
p 1–3		p < 0,001	p = 0,001
p 1–4		p < 0,001	p < 0,001
p 2–3		p = 0,01	p = 0,10
p 2–4		p < 0,001	p < 0,001
p 3–4		p < 0,001	p < 0,001

Table 4 presents the fasting lactate level and erythrocyte sorption capacity index for four groups: phenotype I (CO+AG, n=41), phenotype II (CO+DL, n=32), phenotype III (CO+IR, n=45) and control (n=20). The fasting lactate level was the lowest in the control group (0.9 ± 0.1 mmol/l); in the patient groups, this indicator consistently increased: 1.8 ± 0.4 mmol/l in phenotype I, 2.1 ± 0.5 mmol/l in phenotype II and 2.4 ± 0.6 mmol/l in phenotype III. The sorption capacity (percentage of absorption) of erythrocytes was also the highest in phenotype III (35 ± 7%), while in the control group this indicator was only 10 ± 3%.

When comparing paired groups, significant differences were noted in lactate levels: between phenotypes I and II ($p = 0.04$), between phenotypes I and III ($p < 0.001$), between phenotypes I and control ($p < 0.001$), between phenotypes II and III ($p = 0.01$), between phenotypes II and control ($p < 0.001$), and between phenotype III and control ($p < 0.001$). Significant differences in erythrocyte sorption capacity were found as follows: borderline between phenotypes I and II ($p = 0.05$), between phenotypes I and III ($p = 0.001$), between phenotypes I and control ($p < 0.001$), between phenotype II and control ($p < 0.001$), between phenotype III and control ($p < 0.001$). However, the difference between phenotypes II and III did not reach a statistically significant level ($p = 0.10$).

In MS patients, early metabolic changes (increased fasting lactate) and increased erythrocyte sorption capacity correlate with the severity of the clinical phenotype: the highest values were observed in phenotype III (CO+IR). The significant difference in lactate and sorption capacity from controls indicates the presence of systemic metabolic stress already in the early stages of the disease.

CONCLUSION

Laboratory indicators reflect a worsening of metabolic and inflammatory disorders from group I to group III, which indicates the need for a differential approach to each phenotype. That is, while in the first group it is possible to limit ourselves to blood pressure control and moderate lipid correction, in the third group priority should be given to combating insulin resistance, reducing systemic inflammation, and correcting the imbalance of circadian markers.

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