

## STUDY OF MODERN APPROACHES TO THE PREVENTION AND TREATMENT OF METABOLIC SYNDROME AMONG MILITARY PERSONNEL.

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### Abstract

Research into metabolic syndrome and its impact on military health is developing as a broad area of research, even within the field of general medicine. Authoritative organizations such as the World Health Organization, the European Society of Cardiology, and the American Diabetes Association have extensively studied the diagnostic criteria for metabolic syndrome, risk factors, and prevention methods, and regularly update their guidelines. These guidelines also serve as an important methodological foundation for research in military medicine.

### Keywords

Physical and mental processes, arterial hypertension, dyslipidemia, type 2 diabetes mellitus, overweight, high physical activity, stress, eating disorders, lack of sleep and extreme environmental conditions.

Currently, abdominal obesity and type 2 diabetes mellitus (DM) are recognized by the World Health Organization (WHO) as non-communicable pandemics due to their widespread prevalence, high risk of developing cardiovascular diseases, early disability, and premature mortality [1–5]. According to the WHO, approximately 30% of the world's population is overweight, and one in 20 people is diagnosed with carbohydrate metabolism disorders, with their incidence steadily increasing [1, 3, 6–8].

Metabolic syndrome (MS) is diagnosed in 30% of cases in individuals over 30 years of age, with the number of patients steadily increasing [2, 9–11]. According to our study, the prevalence of MS among patients in the therapeutic departments of

the N.N. Burdenko Main Military Clinical Hospital reaches 16% [12]. MS develops through two stages: the stage of polymetabolic disorders and the stage of the formation of nosological forms of the disease. In clinical practice, methods of mathematical modeling are increasingly used [13,15].

An integrated approach to the analysis of pathological processes makes it possible to identify the leading symptom complexes of the disease [13,14]. Assessing the factor load of the leading symptom complexes of the disease allows us to identify new relationships between the various manifestations of the disease [14]. In our opinion, studying the evolution of various variants of MS as a sequence of interrelated events, ranging from risk factors to the formation of nosological forms, should contribute to the development of targeted approaches to the prevention of certain complications of diseases, the development of which is induced by metabolic disorders included in the concept of MS.

The study materials were obtained as a result of a comprehensive clinical and instrumental examination and a prospective 7-year observation of 634 patients with MS. We assessed changes in the clinical significance of individual components of the syndrome and their combination during prospective observation and created a mathematical model of the various variants of the syndrome based on factor analysis. The selection criteria for patients for inclusion in the study were the recommendations of the All-Russian Scientific Society of Cardiologists (VNOK) in the absence of clinically significant somatic pathology at the beginning of the study [7]. Among those examined there were 457 men and 177 women[1,5,7].

The average age at the beginning of the study was  $50.8 \pm 3.3$  years; blood pressure (BP)  $140.3 \pm 4.5 / 89.4 \pm 3.8$  mmHg; body mass index (BMI) was  $32.0 \pm 2.9$  kg/m<sup>2</sup>, fasting glycemia level  $6.0 \pm 0.5$  mmol/l, postprandial glycemia  $7.4 \pm 0.4$  mmol/l; triglycerides  $1.8 \pm 0.03$  mmol/l, total cholesterol (TC)  $5.9 \pm 0.7$  mmol/l, high-density lipoprotein cholesterol (HDL-C)  $1.0 \pm 0.03$  mmol/l, low-density lipoprotein cholesterol (LDL-C)  $3.1 \pm 0.02$  mmol/l. All patients, along with the standard examination, underwent echocardiography, which assessed the end-systolic (ESV) and end-diastolic volumes (EDV), diastolic thickness of the posterior wall of the left ventricle (DPWLV) and interventricular septum (DPIVS), myocardial mass (MMLV), size of the left atrium (LA); bicycle ergometry (BEM); Holter ECG monitoring with counting the number of atrial (ExS) and ventricular (ExV) extrasystoles; 24-hour blood pressure monitoring (ABPM); ultrasound Doppler study of the main arteries of the head (USDG MAH) with assessment of the velocity (V) of blood flow and the presence of initial atherosclerotic changes in the middle (MCA), anterior (ACA), posterior (PCA) cerebral, common carotid (CCA), internal carotid (ICA) arteries. Serum hormone levels (cortisol, aldosterone) were

also measured. The results were incorporated into a formal protocol, which subsequently allowed for the creation of a database for computer processing [3,4].

Statistical Analysis of Research Results: The Student's t-test was used as a statistical criterion for the significance of differences. Differences were considered significant at  $p \leq 0.05$ . After obtaining the correlation coefficient matrix, factor analysis was performed to reduce the amount of information about the medical object under study. This allows for the creation of mathematical models for the syndromological approach in medicine [2, 7].

By factorizing the correlation coefficient matrix, clinical features were grouped into groups identical to the concept of "disease syndrome," with the first factor reflecting the maximum information about the relationships between the phenomenon under study, and each subsequent factor adding additional information about the relationships between features. Results of our own research By the end point of the study (7-year observation), all 634 people had developed clinically defined conditions: persistent arterial hypertension in 188 subjects (1st group), type 2 diabetes in 141 patients (2nd group) and their combination in 305 patients (3rd group), which allowed us to conditionally talk about the variants of the outcome of MS - hypertensive, diabetic, combined [4,7].

To study the factors determining the various MS outcomes, all patients were divided into three groups based on the study endpoint (conversion to a specific nosological entity). It turned out that these groups already differed in the pre-clinical stage of the syndrome. We conducted a comparative analysis of the main symptom complexes characterizing MS in its various course variants. For this purpose, using factor analysis, we identified factors for each patient group. These factors combined 71.9% of the system's variance in Group 1, 72.1% in Group 2, and 77.3% in Group 3, respectively. Each of the variance systems included five symptom complexes [3,5].

A summary assessment of the symptom complexes for each MS course variant revealed certain differences in the initial characteristics of the patient groups, whose subsequent evolution occurred along one or another course (see table). The first factor (symptom complex) carries the most information about the phenomenon under study, as determined by the percentage of variance used. Each subsequent factor carries less information, but it is orthogonal to the previous one, i.e., the factors are not correlated with each other. As noted above, using multivariate statistics methods, five factors (symptom complexes) were identified in patients with the hypertensive variant of MS, allowing us to represent the clinical characteristics of this group of patients [4,6,8].

The first factor, which explains 20% of the system's variance and is called "arterial hypertension," encompasses features unique to arterial hypertension syndrome—the presence of mean BP (MAP), directly related to both mean systolic (SBP) and mean diastolic (DBP) pressures. An increase in MAP in patients in this group leads to a simultaneous increase in both SBP and DBP, directly indicating a disruption in the physiological mechanisms of blood pressure regulation in the earliest stages of MS, as under normal conditions, an increase in SBP compensatorily leads to a decrease in DBP.

It is understandable why patients in this group subsequently develop persistent arterial hypertension. The second symptom complex in patients with the hypertensive variant of the syndrome (accounting for 18% of the variance) follows logically from the first. Since the overwhelming majority of it included signs reflecting the state of the myocardium itself, we designated it as a factor of "myocardial hypertrophy" [3].

Pre-existing arterial hypertension leads to an increase in the left ventricular septal diameter (LVSD), interventricular septal diameter (IVSD), and the total LVM. Already at this stage, we can speak of the appearance of the first signs of myocardial remodeling, since with an increase in LVM, signs of its systolic dysfunction appear. The third factor, explaining 13% of the system's variance, emphasizes that all the changes occurring in patients with the hypertensive variant of the course of MS are accompanied by lipid metabolism disorders - the "atherogenic dyslipidemia" factor. Arterial hypertension, reflected in the two previous symptom complexes, exists against the background of an increase in atherogenic lipids with a parallel decrease in HDL-C levels and an increase in BMI. The fourth factor - 11% of the variance - demonstrates the presence of cerebral artery remodeling in patients with the hypertensive variant of the evolution of MS already in its early stages - the "vascular remodeling" factor [4,6]. The signs included in the fourth factor are those reflecting the velocity characteristics of blood flow in the main arteries of the head (a direct correlation between blood flow velocity in the ACA, PCA, and CCA, with a simultaneous decrease in the CCA lumen according to ultrasound Doppler imaging). The fifth factor ("myocardial dysfunction") was, to some extent, unexpected. It turned out that even at the baseline of the study, before the final course of MS was determined, patients in this group had manifestations of myocardial dysfunction predisposing to the development of congestive heart failure. Signs characterizing LV ESV and LV EDV (a tendency toward a positive value, i.e., an increase) were accompanied by a decrease in LV pumping function. Moreover, glycemia, included in this symptom complex, completed the full set of MS characteristics in this group, but does not, in

the long term, determine its further development. The initial factor profile of patients with the diabetic variant of MS development turned out to be completely different. Only the first factor, which accounted for 28% of the variance in patients with the diabetic variant, showed some similarity with the above-described group of patients. As in the previous factor, the most significant symptom complex was average blood pressure [3,7].

Its magnitude directly determines the presence of individual signs of initial myocardial remodeling in the factor, namely, LVM and the thickness of its anterior and posterior walls. All this allowed us to designate the first factor of the second dispersion system as "hypertonic myocardial remodeling." A distinctive feature of hypertensive myocardial remodeling in the diabetic variant of MS is that already in its early stages, we observe a tendency for the development of extrasystoles, both supraventricular and ventricular. Moreover, the more pronounced the mean arterial pressure and the thickening of the heart walls, the more frequently these extrasystoles are detected. It is logical that the presence of hypertensive myocardial remodeling, described by the first factor in this group of patients, entails changes in central hemodynamics in these same patients. We designated the second factor as the "central hemodynamics" factor. In the early stages of MS, patients with the diabetic variant typically exhibit increases in both stroke volume (SV), minute volume (MV), and LV ejection fraction (EF) [3,8].

This means that, unlike patients with the hypertensive variant, they do not yet exhibit latent manifestations of congestive heart failure. However, initial manifestations of LV diastolic and systolic dysfunction are already evident in the form of positive LV ESV and LV EDV values. As expected, carbohydrate metabolism disorders are among the most significant symptom complexes in the diabetic variant of MS. The "carbohydrate metabolism disorder" factor, accounting for 10% of the system variance, ranked third. This factor was absent as a significant factor in the hypertensive variant [4,6].

Carbohydrate metabolism was then limited to a single indicator within the fifth factor. In this variant of MS, carbohydrate metabolism disturbances acquired independent significance and combined a genetic predisposition to diabetes (a first in our observations, emphasizing the importance of medical history collection!), the presence of fasting and postprandial glycemia, and the absence of any glycemic control measures. In the diabetic variant of MS, as in the hypertensive variant described above, vascular remodeling, detected by cerebral vascular ultrasound, plays a significant role in the clinical characteristics of patients. These factors, the fourth and fifth, each explain 10% of the system's variance, account for vascular remodeling. Both of these factors include blood flow velocity indicators through the

main cerebral vessels, but in different vascular beds, and signs of vascular stenosis within the same beds. Along with signs of cerebral vascular remodeling, the fourth factor included nonspecific changes in the T wave during exercise testing and the presence of extrasystoles during VEM. All of this gave us reason to designate the fourth factor as a factor of "cardiocerebral changes," in contrast to the fifth factor, in which the appearance of a significant sign—hyperlipidemia—allowed us to designate it as a factor of "initial atherosclerosis." At first glance, the symptom complexes characterizing the combined variant of the MS course were the most difficult to interpret. The initial characteristics of patients with this variant of the syndrome's evolution turned out to be more complex than those with the first two variants [2, 7, 11].

The first, leading symptom complex, which comprised almost half of the system's total variance (45%), revealed a clear association between mean arterial pressure (common with the two previously analyzed groups) and the spectrum of electrocardiographic and lipid-related changes, which had not been observed previously. For these patients, higher mean arterial pressure values were accompanied by more pronounced ECG changes (an increase in the QT interval—a sign of myocardial electrical instability, an increase in the frequency of ventricular and supraventricular extrasystoles) and an "atherogenic" lipid profile. Thus, the first symptom complex already outlined the factor of "hypertensive cardiopathy associated with dyslipidemia." All subsequent factors in patients with a combined course of MS had a significantly lower factor loading [10,13,14].

The second symptom complex, not only in its significance but also in its composition of features, was identical to that seen in patients in Group 2 (diabetic MS), differing only in the degree of significance of individual features but generally characterizing the state of "central hemodynamics."

The third factor (7% of the system's variance) included features indicating an interdependence between the severity of myocardial hypertrophy in patients with a combined MS variant and the patient's BMI and the severity of postprandial glycemia and dyslipidemia. Thus, the third factor demonstrates that not only arterial hypertension determines the presence of increased cardiac mass, but obesity itself (increased BMI), combined with glycemia and dyslipidemia, contributes to myocardial hypertrophy. Quite logically, we designated this factor as "dysmetabolic cardiopathy" [12, 13].

The fourth symptom complex in the combined variant of MS development included signs of narrowing of all major cerebral vessels detected by ultrasound Doppler imaging. This was sufficient to characterize 7% of the system's variance. Unlike the two previously discussed variants of MS evolution, the symptom

complex did not include velocity parameters, which can be explained by the predominance of organic changes in these vessels in this variant, sufficient to form an independent factor. This is precisely what justifies designating the fourth factor in this group of patients as the "initial cerebral atherosclerosis" factor. Finally, the fifth factor (5% of variance) directly correlates the presence of a narrowing of the lumen of the main cerebral arteries (the common and internal carotid arteries) with blood aldosterone levels (changes in the cerebral arteries do not exist in isolation, but suggest changes in other vascular beds, including those of the renin-angiotensin-aldosterone system), glycemia, and lipidemia [15].

This factor also included a fairly significant indicator, but with an inverse relationship from all the others - LVEF, which brings it closer to the fifth factor of patients with the hypertensive variant of MS development - "myocardial dysfunction." All of the above allowed us to designate the fifth factor of the last analyzed group of patients as the factor of "vascular-metabolic relationships." Conclusion Summarizing the results of the factor analysis of the initial data of patients with different variants of the MS course, we can conclude that different mathematical models are applicable to these patients [3, 7, 10].

While patients in the first group are initially characterized by significant arterial hypertension, hypertensive myocardial remodeling, concomitant severe atherogenic lipid metabolism disorder, and initial manifestations of myocardial dysfunction (a precursor to clinically significant circulatory failure), patients with the diabetic variant of MS have less severe arterial hypertension (its presence is not determined by any of the system's main factors), and metabolic disturbances, primarily carbohydrate metabolism, already contribute to both myocardial remodeling and vascular structural and functional changes. These patients do not yet exhibit myocardial dysfunction in the initial phase of MS. Finally, in the combined variant of MS, various combinations of features characteristic of the first two variants of the syndrome are observed in the initial phase [4,8].

Based on the above, another conclusion suggests itself. The onset of MS itself (the sum of its main components) is as uneven as its subsequent course. In some cases, it begins with arterial hypertension, gradually followed by weight gain and metabolic factors (lipidemia, impaired glucose tolerance). In others, metabolic disturbances (either obesity, dyslipidemia, or carbohydrate metabolism disorders) precede each other and arterial hypertension, but, when combined into a single metabolic syndrome, they subsequently form a specific nosology. In any case, this necessitates a differentiated approach to the prevention of conditions that have not yet emerged and the treatment of those that have already developed [6, 7, 13].

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