INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCHERS
ISSN: 3030-332X Impact factor: 8,293 Volume 10, issue 2, March 2025
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METABOLIC SYNDROME IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS

Abstract :Goal. To characterize the metabolic syndrome (MT) in patients with SLE and clarify the contribution of immune inflammation to the development of the latter.
Materials and methods. 154 wives were examined. with SLE, MS. age 35 years, duration of the disease 99 months. The control group consisted of 69 people. without rheumatic diseases of comparable age. The APR III criteria were used to diagnose MS. The detection of atherosclerotic vascular lesions was carried out by ultrasound scanning of the carotid arteries. The concentration of cholesterol, TG, and HDL cholesterol in the blood serum was determined by colorimetric and photometric methods, the levels of hs-CRP is an immunonephelometric method.
Results. MS was diagnosed in 29/154 (19%) patients with SLE and in 5/69 (7%) in the control group (p=0.02). The incidence of MS components: hypertension, HTG, and hypoalphalipoproteinemia was significantly higher in patients with SLE than in the control group. In SLE, the levels of TG, HDL-C, and CRP exceeded the values of these indicators in healthy individuals. The thickness of the carotid artery CMM in SLE and in the control was the same, ATB and coronary artery disease were more often detected in SLE (15% and 14%) than in the control (4% and 2%), (p=0.01). Patients with SLE and MS were older in age, with higher the values of disease activity, IP, and the maximum dose of HC during the disease period ($p<0.05$). The concentration of CRP was significantly higher in patients with SLE and MS. Subclinical manifestations of atherosclerosis were more often diagnosed in patients SLE and MS compared with patients with SLE without MS ($p<0.05$). There were no differences in the frequency of detection of clinical manifestations of atherosclerosis between the groups.
Conclusion. Autoimmune inflammation in SLE plays an important role in the development of MS and cardiovascular diseases.

Key words:autoimmune inflammation, systemic lupus erythematosus, metabolic syndrome, atherosclerosis

INTRODUCTION

Systemic lupus erythematosus (SLE) is an autoimmune disease characterized by the hyperproduction of organ-specific autoantibodies to various components of the cell nucleus with the development of immune-inflammatory damage to tissues and internal organs. As life expectancy increases, cardiovascular diseases (CVD) caused by atherosclerosis and thrombotic complications occupy one of the first places in the mortality structure of patients with SLE. Clinical manifestations of atherosclerotic lesions of the coronary arteries (angina pectoris and/or infarction
myocardial infarction (MI)) affects from 6% to 15% of young women (35-44 years old) with SLE, subclinical forms of the disease (the presence of asymptomatic atherosclerotic plaques) they are registered in 40% of patients. The accelerated development of atherosclerotic vascular lesions in SLE may be due to both the accumulation of traditional cardiovascular risk factors (RFS) and a wide range of "non-traditional" RFS associated with autoimmune inflammation or

pharmacotherapy of the disease, primarily the use of glucocorticoids (GK). Recently, there have been studies on the problem of metabolic syndrome (MS) in SLE. The key factor in the development of this syndrome is considered to be insulin resistance (IR), and its components are impaired glucose tolerance, diabetes mellitus, hypertension, combined with abdominal obesity and atherogenic dyslipidemia (DLP) [increased levels of triglycerides (TG) and low-density lipoprotein cholesterol (LDL cholesterol), decreased concentrations of high-density lipoprotein cholesterol (HDL cholesterol). The combination of symptoms that make up MS, It leads to accelerated development of cardiovascular complications (CVD) caused by atherosclerosis. It is assumed that autoimmune inflammation
plays a leading role in the formation of MS in patients with SLE, since an increase in the concentration of acute-phase proteins, proinflammatory cytokines, and cellular adhesion molecules is associated with various components of MS: obesity, impaired glucose tolerance, DLP and arterial hypertension.

MATERIALS AND METHODS OF RESEARCH

156 women with a reliable diagnosis of SLE were examined. The average age of the patients at the time of the examination was 35 years old, the duration of the disease is 99 months. The control group consisted of 69 "conditionally healthy" wives. without rheumatic diseases comparable to patients in age. The activity of SLE was assessed by the SLEDAI2K index (Systemic Lupus Erythematosus Disease Activity Index), severity of irreversible damage
to internal organs (IP) – according to the SLICC index (Systematic Lupus International Collaborating Clinics/American College of Rheumatology Damage Index). To assess the risk of developing CVD, a family history of CVD (early onset of coronary artery disease in the immediate family: MI or sudden cardiac death in men under 55 years of age, in women under 65 years of age); increased body mass index body weight (BMI) ≥ 25 kg/m^2; DLP (abnormal levels of lipids and lipoproteins – total cholesterol (cholesterol) > 5.0 mmol/l, TG > 1.8 mmol/l,
LDL CHOLESTEROL > 3.0 mmol/L, HDL CHOLESTEROL < 1.3 mmol/L); arterial hypertension (AH) – increased blood pressure >140/90 mmHg or taking antihypertensive drugs; smoking, menopause. Criteria were used to diagnose MS
ATP III: waist size (FROM) > 88 cm, blood pressure (BP) ≥ 130/85 mmHg, hypertriglyceridemia (HTG) – increased triglyceride levels (TG) ≥ 1.7 mmol/L, decreased concentration of high-density lipoprotein cholesterol (HDL cholesterol) < 1.3 mmol/L, increased fasting glucose level > 6.1 mmol/l. The diagnosis of MS was established in the presence of 3 or more signs. IHD was determined based on a history of MI or a typical picture of angina pectoris and/or positive results of stress tests (treadmill test or bicycle ergometric test).

THE RESULTS AND THEIR DISCUSSION

MS was diagnosed in 29/154 (19%) patients with SLE and in 5/69 (7%) in the control group (p=0.02). The occurrence of such components of MS as hypertension, HTG and hypoalphalipoproteidemia in patients with SLE were significantly higher than in the control group. In SLE, the levels of TG, HDL cholesterol and hs-CRP also exceeded the values of these indicators in healthy individuals. The thickness of the carotid artery CMM in patients with SLE and in the control was the same, ATB and clinical manifestations of atherosclerosis (CHD) were more often detected in SLE (15% and 14%) than in the control (4% and 2%, respectively, p=0.01). Patients with SLE were divided into 2 groups: I
– with MS (29 people) and II – without MS (125 people). Patients with SLE and MS were older in age, with higher values of disease activity, IP, and the maximum dose of HA during the disease period (p<0, 05). Duration of administration, cumulative dose of HA, and the dose of HA currently taken The time was the same in the compared groups. The concentration of CRP was

significantly higher in group I than in group II ($p<0.005$). Subclinical manifestations of atherosclerosis (maximum value KIM and the frequency of ATB) were significantly more often diagnosed in patients of group I compared with group II ($p<0.05$). There were no differences in the frequency of detection of clinical manifestations of atherosclerosis (CHD and MI) between the groups. Studies on MS indicate its high prevalence not only in the general population, but also in rheumatic diseases (RH) based on systemic inflammation, such as SLE and rheumatoid arthritis. According to our data, the incidence of MS and its components in patients SLE turned out to be significantly higher than in the control, which coincides with the results of other authors. In patients with SLE, clinical (CHD) and subclinical (ATB) manifestations of atherosclerosis were detected more often than in the control group. This data confirm the connection of metabolic disorders with CVD caused by atherosclerosis. It has been shown that an increased risk of developing CVD

in SLE is associated not only with traditional RFS, but also with the immuno-inflammatory mechanisms underlying the pathogenesis of both SLE and atherosclerosis. Among the various immunological markers of atherosclerosis that determine the risk of developing severe CVD, special attention is paid to acute inflammatory phase proteins (CRP), proinflammatory cytokines [interleukin (IL)-6, IL-18, tumor necrosis factor (TNF)-α], cellular adhesion molecules (sVCAM-1), changes in the concentrations of which reflect the course of the autoimmune process in SLE. Previous studies have demonstrated that certain symptoms of MS ("atherogenic" lipid profile, IR, AH) may be the result of a subclinical ("low grade") inflammatory process or SLE therapy. For example, disorders of the blood lipid spectrum in patients with SLE are detected already at the onset of the disease and are associated with the inflammatory activity of the disease. It is believed that an increase in the level of TG and a decrease in HDL cholesterol concentration on the background of The high activity of the autoimmune process is associated with the ability of "pro-inflammatory" cytokines (TNF-α, IL-1, interferon-γ) and acute-phase proteins to suppress lipoprotein lipase activity. They found that insulin resistance is associated with systemic inflammation and changes in in the hypothalamic-pituitary system: increased concentrations of IL-6 and TNF-α lead to impaired neurosecretion in the ventromedial nuclei of the hypothalamus, causing hypercortisolemia and subsequently IR. The main mechanism of hypertension development The cause of obesity in hyperinsulinemia is the activation of the sympathetic-adrenal and renin-angiotensin-aldosterone systems, a certain role in

the development of which is played by subclinical inflammation. It is known that HA, the basic drugs in the treatment of SLE, negatively affect the formation of individual components of MS (increased blood pressure, hyperglycemia, obesity). With our data on the absence of GC influence on the development of MS The results of a number of authors are consistent with IR. Despite the association of the maximum dose of GK during the disease period with MS, there were no differences in the duration of administration and the cumulative dose of GK in patients with SLE. Thus, the results obtained indicate that autoimmune inflammation plays a major role in the development of MS and CVD.

CONCLUSIONS

Thus, the results obtained indicate that autoimmune inflammation plays a major role in the development of MS and CVD. Further study of the interrelationships of subclinical inflammation, MS and its components will allow us to determine their importance in the progression of atherosclerotic vascular damage and develop new approaches to prevention and treatment. MTR in patients with SLE.

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