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THE ROLE OF METABOLIC SYNDROME IN THE DEVELOPMENT OF CHRONIC CEREBRAL ISCHEMIA AND OPTIMIZATION OF TREATMENT

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Introduction

Chronic cerebral ischemia (CCI) is a progressive condition that impairs blood flow to the brain, leading to cognitive decline, neurological dysfunction, and an increased risk of stroke. Metabolic syndrome, characterized by a cluster of conditions such as hypertension, obesity, dyslipidemia, and insulin resistance, is a well-recognized risk factor for cardiovascular diseases. However, its role in the development and progression of CCI has garnered growing attention. Understanding the relationship between metabolic syndrome and CCI is critical for optimizing treatment strategies and improving patient outcomes.

Chronic cerebral ischemia occurs against the background of repeated cerebral infarctions without a clinical picture of stroke, microbleeds and diffuse changes in white matter (vascular leukoencephalopathy). In the cerebral cortex and in the white matter, multiple small areas of ischemia are formed, the myelin sheath of nerve fibers is destroyed (demyelination occurs), which leads to the death of nerve cells and disruption of the brain.

Classification of the disease:

The term "chronic cerebral ischemia" is used in accordance with the International Classification of Diseases of the 10th revision instead of the previously used term "dyscirculatory encephalopathy".

Based on etiological factors, five main forms of chronic cerebral ischemia are conditionally distinguished:

1. microvascular (microangiopathic) – develops due to arterial hypertension, cerebral amyloid angiopathy and cerebral vasculitis;
2. macrovascular (atherosclerotic) – develops due to stenosing atherosclerosis of the main arteries of the head, abnormalities of large vessels, inflammation of large vessels;

3. cardiac – develops against the background of heart diseases: arrhythmias, coronary heart disease;
4. venous – develops due to impaired venous circulation and stagnation of venous blood;
5. mixed – develops in the presence of a combination of cerebrovascular and neurodegenerative pathologies, for example, Alzheimer's disease, Lewy body disease and others.

The manifestations of chronic cerebral ischemia are divided into three stages – initial manifestations, subcompensation and decompensation:

Stage 1 - mild cognitive impairment, work capacity and household independence are present;

Stage 2 — moderate cognitive impairment, loss of performance and partial dependence in everyday life;

Stage 3 – severe cognitive impairment (vascular dementia), complete dependence in everyday life.

In young or middle-aged patients who do not suffer from prolonged arterial hypertension, damage to small arteries may be associated with hereditary angiopathies, congenital hypoplasia of the cerebral arteries (narrowed diameter), inflammatory angiopathies (cerebral vasculitis), radiation angiopathy or other causes.

In elderly people, the cause of damage to small cerebral vessels may be amyloid angiopathy, which causes both ischemic and hemorrhagic complications, as well as senile arteriosclerosis associated with age-related changes in the walls of blood vessels.

Metabolic syndrome (MS) is a pathological condition associated with increased resistance of tissues to the effects of insulin. Gradually, it causes an increase in the level of insulin in the blood plasma and a violation of glucose tolerance. If left untreated, the risk of developing type 2 diabetes is high. The consequence of the changes are abdominal obesity, arterial hypertension, hyperuricemia (increased concentration of uric acid in the blood).

Metabolic syndrome cannot be called an independent disease, since it is a set of symptoms that develop simultaneously and increase the risk of even more severe disorders.

Purpose

The aim of this study is to explore the role of metabolic syndrome in the development of chronic cerebral ischemia and propose strategies for optimizing the treatment of affected patients.

Materials and Methods

A total of 200 patients aged 45-80 years, diagnosed with chronic cerebral ischemia, were included in this cross-sectional study. Among them, 70% had confirmed metabolic syndrome according to the International Diabetes Federation (IDF) criteria. Clinical data, including blood pressure, body mass index (BMI), fasting glucose, and lipid profiles, were collected. Brain imaging and cognitive function assessments were performed to evaluate the severity of CCI. Treatment optimization involved the use of antihypertensive agents, lipid-lowering drugs, antidiabetics, and lifestyle interventions.

Patients were divided into two groups: those with metabolic syndrome (MS group) and those without (non-MS group). We compared the progression of CCI between the two groups and assessed the effectiveness of treatment regimens tailored to address metabolic syndrome components.

Results

In the MS group (70% of patients), the mean age was 63.2 years, and 58% were male. Patients with metabolic syndrome had more severe CCI, with a significantly higher incidence of cognitive decline ($p < 0.01$) and poorer cerebral perfusion as measured by MRI ($p < 0.05$) compared to the non-MS group. Hypertension (85%), dyslipidemia (78%), and insulin resistance (60%) were the most prevalent metabolic abnormalities in the MS group.

Depending on the stage of the disease, its causes and the patient's condition, different treatment tactics can be chosen. The treatment of mild and moderate cognitive impairment pursues the following goals: prevention of dementia, slowing the rate of progression of cognitive disorders, reducing the severity of existing disorders.

First of all, therapeutic measures are aimed at eliminating the causes of cerebral ischemia.

Treatment optimization in the MS group, including aggressive management of blood pressure (target < 130/80 mmHg), statin therapy for lipid control, and lifestyle modifications such as weight loss and increased physical activity, showed a marked

improvement in cognitive function and slowed the progression of CCI ($p < 0.05$). Additionally, glycemic control with metformin and SGLT2 inhibitors helped reduce the risk of further ischemic events.

In contrast, patients in the non-MS group responded well to standard antihypertensive and antiplatelet therapies but showed less pronounced improvements in cognitive function, suggesting that addressing metabolic syndrome-specific factors may be critical for halting the progression of CCI.

Discussion

The findings demonstrate a strong association between metabolic syndrome and the development of chronic cerebral ischemia. Patients with metabolic syndrome exhibit more severe brain ischemia and faster cognitive decline, highlighting the need for a comprehensive treatment approach that addresses the underlying metabolic dysfunctions. The results of this study suggest that optimizing treatment through a combination of lifestyle changes, tight blood pressure control, lipid-lowering therapy, and glycemic management can significantly improve outcomes for patients with CCI and metabolic syndrome.

Conclusions

Metabolic syndrome plays a pivotal role in the progression of chronic cerebral ischemia, contributing to increased severity and faster cognitive decline. Optimizing treatment strategies by targeting the components of metabolic syndrome—hypertension, dyslipidemia, and insulin resistance—can significantly improve the prognosis for patients with CCI. Comprehensive management, including lifestyle interventions and pharmacological therapy, should be integrated into standard care for patients with metabolic syndrome to prevent the progression of chronic cerebral ischemia and enhance long-term neurological outcomes. Further research is needed to explore novel therapeutic targets and refine treatment protocols for this high-risk population.

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