

Conclusions

Stroke is the third common cause of death after cancer and cardiovascular diseases and is the most common neurological disorder. Death and complications in hemorrhagic stroke are higher than ischemic stroke. The risk develops with age and occurs more common in men than women. The results of this study indicate, the most important risk factor in stroke is hypertension, which not only increases the incidence of this condition but also makes the recovery period longer. In the next places, diabetes and hyperlipidemia are the factors prolonging the recovery period. The effect of alcohol in this study did not show a significant trend, while not smoking and having physical activity made the initial recovery significantly shorter. Due to the impact of hypertension in a longer and harder recovery after strokes, it is recommended that more precise attention be given to hypertensive screening, especially in risk groups such as the elderly, and overall, a holistic approach to stroke care, which includes comprehensive medical management and rehabilitation support tailored to the individual's needs, is crucial for optimizing recovery in patients of all categories.

LITERATURE

1. Goldstein, L. B., Primary prevention of ischemic stroke: a guideline from the American Heart Association / L. B. Goldstein, R. Adams, M. J. Albert // American Stroke Association Stroke Council: Stroke 2006. – Vol. 37. – P. 1583–633.
2. Stroke risk factors, genetics, and prevention / A. K. Boehme, C. Esenwa C, M. S. Elkind // Front. Neuroendocrinol. – 2017. – Vol. 120, №. 3. – P. 472–474.

УДК 616.831-022-008.64:616.133.33

M. Saad

Scientific Supervisor: Ph.D., associate professor N. N. Usova

*Educational Establishment
«Gomel State Medical University»
Gomel, Republic of Belarus*

LEUKOENCEPHALOPATHY OF UNSPECIFIED GENESIS WITH MODERATE VESTIBULO-ATAXIC SYNDROME, DECOMPENSATION, PROBABLE CADASIL SYNDROME

Introduction

Leukoencephalopathy is a broad term encompassing disorders that affect the white matter of the brain. A subset of these conditions remains of unspecified origin, complicating diagnosis and treatment. Moderate vestibulo-ataxic syndrome with decompensation is a common manifestation in such cases, leading to progressive neurological impairment. The hereditary nature of CADASIL (Cerebral Autosomal Dominant Arteriopathy with Subcortical Infarcts and Leukoencephalopathy) makes it a prime suspect in unexplained cases of leukoencephalopathy. This study explores the possible connection between unspecified leukoencephalopathy and CADASIL, analyzing clinical presentations and diagnostic approaches [1].

Goal

To investigate the clinical features and diagnostic challenges of leukoencephalopathy of unspecified genesis, particularly in patients presenting with moderate vestibulo-ataxic syndrome, decompensation, and suspected CADASIL.

Material and methods of research

A retrospective analysis of ten patients (aged 45-60) presenting with progressive white matter degeneration, vestibulo-ataxic syndrome, and cognitive decline was conducted. All

subjects underwent neuroimaging (MRI), genetic testing for NOTCH3 mutations, and cerebrospinal fluid analysis. The severity of vestibulo-ataxic syndrome was assessed using the Scale for the Assessment and Rating of Ataxia (SARA), and cognitive function was evaluated through the Mini-Mental State Examination (MMSE).

The results of the research and their discussion

MRI findings demonstrated widespread white matter hyperintensities in all cases, predominantly in periventricular and subcortical regions. Five patients exhibited the characteristic anterior temporal lobe involvement associated with CADASIL, while three had deep lacunar infarcts suggestive of small-vessel disease. Genetic testing confirmed NOTCH3 mutations in four individuals, supporting a CADASIL diagnosis. The remaining six patients were categorized as having leukoencephalopathy of unknown origin. SARA scores correlated with disease progression, with decompensated cases showing significant gait disturbances. Cognitive decline was prominent in CADASIL-positive cases, with MMSE scores averaging 18/30. The findings highlight the challenge of diagnosing CADASIL solely based on clinical and imaging features, emphasizing the importance of genetic testing.

Conclusions

Leukoencephalopathy of unspecified genesis presents diagnostic difficulties, particularly when associated with vestibulo-ataxic syndrome and progressive neurological decline. CADASIL remains an important differential diagnosis, with genetic testing serving as a crucial tool for confirmation [2]. Early identification and supportive management are essential in mitigating disease progression and improving patient outcomes.

LITERATURE

1. Cadasil: a hereditary adult-onset condition causing stroke and dementia / Chabriat H, Joutel A, Dichgans M [et al] // Brain, 2009. – Vol. 132, №. 4. – P. 933–948.
2. The influence of genetic and cardiovascular risk factors on cerebral white matter integrity in CADASIL patients / S. Singhal, S. Bevan, T. Barrick [et al.] // Stroke, 2014. – Vol. 45, №. 5. – P. 1235–1240.

УДК 616.894-053.8

K. D. K. P. R. Perera, T. H. Hathagoda

Scientific Supervisor: Ph.D., associate professor N. N. Usova

*Educational Establishment
«Gomel State Medical University»
Gomel, Republic of Belarus*

COMPARING THE EFFECTS OF EARLY-ONSET VS. LATE-ONSET ALZHEIMER'S DISEASE, A PROGRESSIVE NEURODEGENERATIVE DISORDER WORLDWIDE

Introduction

Alzheimer's disease (AD), a degenerative neurological disorder characterized by behavioral problems, memory loss, and cognitive decline, is the most common type of dementia in the world. In this progressive neurodegenerative disease, beta-amyloid plaques and neurofibrillary tangles accumulate, compromising neuronal function and leading to widespread brain atrophy. Beta-amyloid, particularly the lethal A β 42 form, builds up between neurons to form plaques that interfere with synaptic transmission and cellular metabolism.

Meanwhile, abnormal tau protein breaks away from microtubules to form tangles inside neurons, which further disrupt synaptic function by obstructing nutrition transfer.