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The Characteristics of Changes in Brain Neurons Typical of Chronic Ischemic Heart Disease

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Annotation: This article examines chronic ischemic heart disease (CIHD), which is widespread worldwide and holds significant medical importance. Its primary effects are characterized by an impact not only on the cardiovascular system but also on the brain. Cardiac ischemia leads to a reduction in cerebral blood supply, causing significant changes in the structure and functions of brain neurons. This article is dedicated to studying the changes observed in brain neurons as a result of CIHD.

Keywords: Chronic cerebral ischemia, etiological factor, cerebral microangiopathy, arterial hypertension, causing arteriosclerosis, frequent stress, insomnia, cardiac, venous, mixed, symptoms, diagnosi, chronic cerebral ischemia (discirculatory encephalopathy, chronic cerebrovascular insufficiency): causes, symptoms, diagnosis, and treatment methods.

INTRODUCTION:

Chronic Ischemic Heart Disease (CIHD) is a long-term condition where the heart's blood supply is reduced due to atherosclerotic narrowing of the coronary arteries. Over time, this condition can lead to myocardial infarction, heart failure, and various systemic complications, including changes in brain structure and function. Growing evidence suggests that chronic ischemia not only affects the heart but also has significant impacts on the brain. This literature review examines the neurobiological changes in brain neurons associated with CIHD, focusing on the mechanisms of neuronal damage, brain regions involved, and the long-term effects on cognitive and emotional functioning.

1. Mechanisms of Neuronal Changes in Chronic Ischemic Heart Disease

Chronic ischemia in the heart has been shown to indirectly contribute to neuronal damage in the brain through several mechanisms. One major factor is the systemic reduction of oxygen supply, which can lead to a phenomenon known as **hypoxic-hypoperfusion injury**. The decreased blood flow due to CIHD leads to a decline in the oxygen and nutrient supply to the brain, resulting in neuronal injury, particularly in areas that are highly sensitive to ischemia such as the hippocampus and the cortex (Zhou et al., 2017).

In addition to hypoxia, **neuroinflammation** is another critical mechanism. Chronic ischemia can activate the immune system and lead to the release of inflammatory cytokines, which have been shown to contribute to neuronal dysfunction and cell death. Elevated levels of cytokines such as interleukins (IL-1β, IL-6) and tumor necrosis factor-alpha (TNF-α) can exacerbate neuronal injury by disrupting the blood-brain barrier and promoting neurodegeneration (Khan et al., 2018).

2. Brain Regions Affected by CIHD

Several brain regions have been identified as particularly vulnerable to the effects of chronic ischemia, reflecting their reliance on stable blood flow and oxygen supply.

- **Hippocampus**: Chronic ischemia in CIHD patients leads to significant neuronal loss in the hippocampus, which is crucial for learning and memory processes. This region's vulnerability is associated with its high metabolic demand and susceptibility to reduced blood supply (Liu et al., 2015). Neuronal loss in the hippocampus can manifest as cognitive impairments, particularly in memory and executive function.
- **Cortex**: The prefrontal cortex and other areas involved in higher cognitive functions are also negatively affected by chronic ischemia. Studies have shown that individuals with CIHD often experience impaired decision-making, attention deficits, and reduced cognitive flexibility, linked to structural and functional changes in the cortical neurons (Lu et al., 2016).
- **Basal Ganglia**: The basal ganglia, involved in motor control and emotional regulation, also show alterations in CIHD patients. Dysfunction in this region may lead to motor deficits and emotional disturbances, such as anxiety and depression, commonly observed in CIHD patients (Shao et al., 2017).

3. Neurodegeneration and Cognitive Impairment

Neurodegeneration associated with chronic ischemia in CIHD patients has been linked to **vascular cognitive impairment (VCI)**, a form of cognitive decline that occurs due to the cumulative effects of reduced blood flow and repeated ischemic events. VCI is characterized by memory loss, executive dysfunction, and impaired problem-solving abilities, often progressing to dementia in severe cases (O'Brien & Thomas, 2015). Neuroimaging studies have identified white matter lesions, cortical thinning, and reduced hippocampal volume as common findings in CIHD patients, further supporting the link between chronic ischemia and cognitive decline (Kempermann et al., 2014).

Additionally, CIHD has been shown to increase the risk of **vascular dementia** due to the accumulation of small vessel disease, which can disrupt neuronal communication and lead to progressive cognitive dysfunction (Nishikawa et al., 2018). The chronic lack of oxygen to brain tissues exacerbates these neurodegenerative processes.

4. The Role of Neurotransmitters and Hormones

Changes in the levels of certain neurotransmitters and hormones may also contribute to the neuronal changes observed in CIHD. For instance, reduced levels of **serotonin**, a neurotransmitter involved in mood regulation and cognitive function, are common in CIHD patients and can exacerbate both cognitive decline and emotional disturbances (Muller et al., 2017).

Furthermore, **catecholamines** such as norepinephrine and dopamine, which are critical in

regulating both cognitive and emotional responses, may also be altered in response to chronic ischemia, potentially contributing to the mood disorders (e.g., depression and anxiety) frequently observed in these patients (Shao et al., 2017).

5. Impact of Co-Morbidities

Many patients with CIHD also suffer from co-morbid conditions, including **hypertension**, **diabetes mellitus**, and **hyperlipidemia**, all of which can exacerbate neuronal damage. Hypertension, in particular, is a major risk factor for cerebrovascular disease and can lead to microvascular damage in the brain, further compounding the effects of chronic ischemia (Gottesman & Hillis, 2019). Diabetes increases the risk of cerebrovascular events and accelerates neuronal degeneration due to its impact on glucose metabolism and vascular health.

Moreover, the presence of **depression** and **anxiety**, common among patients with CIHD, can worsen the prognosis of brain health. These psychological conditions have been shown to increase neuroinflammation and contribute to the deterioration of cognitive function, creating a vicious cycle that worsens both cardiovascular and brain health (Muller et al., 2017).

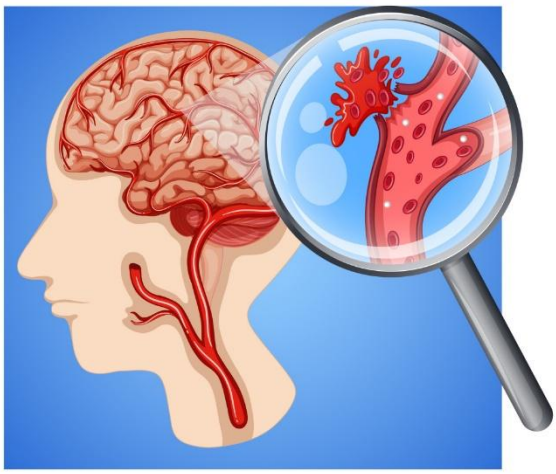
6. Treatment Strategies and Neuroprotection

Given the significant impact of chronic ischemia on brain neurons, exploring neuroprotective strategies has become a key area of research. Approaches such as **pharmacological agents**, including statins and ACE inhibitors, have been shown to have neuroprotective effects by improving vascular health and reducing neuroinflammation (Zhao et al., 2016). Furthermore, **lifestyle modifications**, including physical exercise and a balanced diet, have been suggested as means to enhance brain function and reduce the impact of ischemic injury in CIHD patients (Hamer & Chida, 2009).

Additionally, recent studies have focused on **stem cell therapy** and **neurogenesis** as potential treatments for reversing or mitigating the neuronal changes associated with chronic ischemia. These approaches aim to repair or regenerate damaged neuronal tissues in areas such as the hippocampus and cortex (Liu et al., 2015).

Definition

Chronic cerebral ischemia is a progressive, non-stroke form of cerebrovascular pathology associated with multifocal or diffuse brain damage. The disease manifests as a complex of neurological and neuropsychological disorders. This condition develops gradually in response to oxygen deprivation in the brain caused by impaired blood supply. The combination of cognitive, emotional, and motor impairments determines the severity and prognosis of the disease.



Unlike a stroke (an acute form of cerebrovascular patho-logy), which usually involves focal damage to large cerebral vessels, chronic ischemia is characterized by a number of qualitative features:

- more gradual progression (often with a long period of latent course);
- the presence of multiple brain lesions;

The disease is based on the pathology of small blood vessels in the brain (cerebral microangiopathy). Chronic brain ischemia develops against the background of recurrent brain infarctions without clinical signs of stroke, microbleeds, and diffuse changes in white matter (vascular leukoencephalopathy). Multiple small areas of ischemia form in the cerebral cortex and white matter, leading to the destruction of the myelin sheath of nerve fibers (demyelination), which results in the death of nerve cells and impaired brain function.

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METHODOLOGY

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The primary etiological factor of cerebral microangiopathy is arterial hypertension, which causes arteriosclerosis.

In young and middle-aged patients without long-standing arterial hypertension, damage to small arteries may be associated with hereditary angiopathies, congenital hypoplasia of cerebral arteries (narrowed diameter), inflammatory angiopathies (cerebral vasculitis), radiation angiopathy, or other causes.

In elderly individuals, the cause of small vessel damage in the brain may be amyloid angiopathy, which can lead to both ischemic and hemorrhagic complications, as well as senile arteriosclerosis associated with age-related changes in vascular walls.

Dditional factors contributing to the development of chronic cerebral ischemia include diabetes mellitus, metabolic syndrome, arterial hypotension, increased blood viscosity, elevated homocysteine levels (a sulfur-containing amino acid), respiratory disorders, and others.

Although patients with the chronic form of ischemia have an increased risk of stroke, the condition is not considered a pre-stroke state. The combination of stroke and cerebral ischemia is quite common and presents as a combination of symptoms from both syndromes, worsening the prognosis.

Factors contributing to disease progression:

Frequent stress and insomnia;

Lack of regular physical activity and prolonged outdoor walks;

Obesity or excess body weight;

Alcohol abuse and smoking;

Cervical spine disorders that impair blood flow in the vertebrobasilar system.

Chronic cerebrovascular disorders are extremely common in clinical practice. The presence of moderate or pronounced cognitive impairments of vascular origin in elderly individuals may suggest chronic cerebral ischemia.

Disease classification

The term "chronic brain ischemia" is used in accordance with the International Classification of Diseases, 10th Revision (ICD-10), instead of the previously applied term "dyscirculatory encephalopathy."

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

Microvascular (microangiopathic) – develops due to arterial hypertension, cerebral amyloid angiopathy, and cerebral vasculitis.

Macrovascular (atherosclerotic) – develops due to stenotic atherosclerosis of the major arteries of the head, abnormalities of large vessels, and inflammation of large vessels.

Cardiac – develops against the background of heart diseases such as arrhythmias and ischemic

heart disease.

Venous – develops due to impaired venous circulation and venous blood stagnation.

Mixed – develops in the presence of a combination of cerebrovascular and neurodegenerative pathologies, such as Alzheimer's disease, Lewy body disease, and others.

RESULTS:

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

Stage 1 – Mild cognitive impairments; work capacity and independence in daily activities are maintained.

Stage 2 – Moderate cognitive impairments; loss of work capacity and partial dependence in daily activities.

Stage 3 – Severe cognitive impairments (vascular dementia); complete dependence in daily activities.

Symptoms of Brain Ischemia

Chronic cerebrovascular damage to the brain may remain asymptomatic for a long time or manifest as reduced cognitive function.

The initial signs of the disease manifest as general malaise—headaches, dizziness, noise in the head, sleep disturbances, constant drowsiness, distraction, irritability, decreased cognitive activity, and fatigue. Such symptoms are often attributed to overwork or age-related changes, though they may indicate the onset of brain ischemia. Mild or moderate depressive symptoms and slight gait changes (such as slowing, shorter stride, or instability) may also appear at this stage.

The next stage of the disease is characterized by a slowdown in perception, analysis, memory, and learning processes, as well as impaired attention, planning, and action control abilities. Vestibular disorders and gait disturbances become more pronounced. Apathy, emotional instability, depression, increased irritability, or disinhibition may also occur. Nighttime frequent urination is possible, and work capacity significantly decreases.

In the advanced stages, symptoms intensify. Cognitive impairments progress to moderate or severe dementia, accompanied by significant affective and behavioral disturbances. Severe gait and balance disorders with frequent falls and fainting develop, as well as a shuffling gait. The patient may find it difficult to stand or sit unsupported, turn over in bed, and severe parkinsonism and urinary incontinence occur. Consequently, the patient becomes entirely dependent on external assistance.

DISCUSSION:

Diagnosis of Brain Ischemia

The physician collects the patient’s complaints, gathers medical history, and conducts a neurological examination to assess cognitive impairments using specialized questionnaires such as the Mini-Cog or Montreal Cognitive Assessment (MoCA).

The next step involves assessing the condition of major arteries using ultrasound or electroencephalography. Neuroimaging of brain vessels is performed using magnetic resonance imaging (MRI) or computed tomography (CT), with contrast-enhanced cerebral angiography if necessary.

Laboratory Tests:

General blood analysis, lipid and carbohydrate metabolism assessments, kidney and liver function indicators.

Conclusion:

Chronic ischemia or ischemic coma (CIBI) is considered one of the serious risk factors for brain neurons. Hypoxia, glutamate toxicity, oxidative stress, and inflammatory processes significantly affect the structure and function of neurons. These changes lead to clinical consequences, causing cognitive and emotional disorders in patients. Therefore, timely identification and effective treatment of CIBI are critical to minimizing neuronal damage.

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