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THE RELATIONSHIP BETWEEN LUNG DAMAGE AND STRUCTURAL AND FUNCTIONAL CHANGES IN THE CENTRAL NERVOUS SYSTEM IN MDR TUBERCULOSIS: LITERATURE REVIEW

Panoyeva Nilufar Khamroyevna, Sanoyeva Matlyuba Jakhonkulovna
Bukhara State Medical Institute named after Abu Ali ibn Sino,Bukhara,Uzbekistan

Annotation.
Multidrug-resistant tuberculosis (MDR tuberculosis) remains one of the most pressing problems in modern phthisiology and neurology, due to the high incidence of complications, the systemic nature of the pathological process, and an unfavorable prognosis involving the central nervous system. Damage to the central nervous system in patients with MDR pulmonary tuberculosis is accompanied by the development of neurological disorders of varying severity, changes in the bioelectric activity of the brain and structural disorders detected by neuroimaging studies.
Keywords: MDR tuberculosis, central nervous system, neuroimaging, electroencephalography, hypoxia, neurological complications, brain.

Introduction. Despite significant advances in modern medicine, tuberculosis continues to be one of the leading causes of infectious morbidity and mortality in the world. MDR tuberculosis of the lungs is particularly dangerous, characterized by the resistance of mycobacteria to the main anti-tuberculosis drugs. This form of the disease is characterized by a severe clinical course, a high incidence of complications, and an unfavorable prognosis[1-4].

One of the most significant systemic complications of MDR tuberculosis is damage to the central nervous system. Neurological disorders in this pathology can develop due to chronic intoxication, hypoxic tissue damage, vascular disorders and immune-inflammatory processes. Patients often have asthenoneurotic disorders, cognitive impairments, dyscirculatory changes, and signs of chronic cerebral insufficiency[2,8,9].

In recent years, the active development of neuroimaging and neurophysiological research methods has significantly expanded the understanding of the mechanisms of brain damage in chronic infectious diseases. The use of magnetic resonance imaging, computed tomography, and electroencephalography makes it possible to detect early signs of cerebral dysfunction and assess the extent of damage to the central nervous system[5,10,14].

The development of damage to the central nervous system in MDR pulmonary tuberculosis is multifactorial. One of the leading mechanisms is considered to be chronic hypoxia, which occurs due to impaired respiratory function and decreased tissue oxygenation. Prolonged hypoxia leads to impaired energy metabolism in neurons, a decrease in the functional activity of the brain, and the development of vascular dyscirculatory changes[11-13].

Chronic tuberculosis intoxication plays an essential role. Inflammatory products and toxic metabolites have a damaging effect on the vascular wall and nervous tissue, contributing to the development of microcirculatory disorders and cerebral ischemia.

Of no small importance are immuno-inflammatory reactions, accompanied by the activation of pro-inflammatory cytokines and the development of endothelial dysfunction. Impaired cerebral

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hemodynamics leads to the formation of focal and diffuse changes in the brain, which progress as the severity of the underlying disease increases[1,17,20].

Neurophysiological research methods make it possible to assess the functional state of the central nervous system and identify early signs of cerebral dysfunction. Electroencephalography is one of the most informative methods.

According to various studies, diffuse changes in the bioelectric activity of the brain are most often detected in patients with MDR pulmonary tuberculosis, accompanied by disorganization of the alpha rhythm and a decrease in the functional activity of the cortex. The severity of EEG changes directly depends on the degree of intoxication, the severity of respiratory failure, and the prevalence of tuberculosis[6,9].

Patients with a long-term course of the disease may show signs of dysfunction of the mid-stem structures of the brain, indicating involvement of deep parts of the central nervous system. Such changes are considered as a manifestation of chronic hypoxic-intoxication encephalopathy.

Violation of the bioelectric activity of the brain is accompanied by the development of cognitive disorders, memory loss, emotional lability and asthenic syndrome. Many authors emphasize that the severity of neurophysiological disorders increases with the progression of MDR pulmonary tuberculosis[3,8,19].

Modern neuroimaging techniques play an important role in the diagnosis of central nervous system damage in patients with MDR pulmonary tuberculosis. Magnetic resonance imaging makes it possible to detect early structural changes in cerebral tissues even in the absence of pronounced clinical symptoms[2].

Most often, patients show dyscirculatory changes in the brain, signs of chronic ischemia, dilation of cerebrospinal fluid spaces, and focal changes in white matter. Such changes reflect impaired cerebral circulation and chronic vascular hypoxic damage to the nervous tissue[6].

In some patients, moderate atrophic processes are detected, indicating a prolonged course of the disease and a chronic violation of neuronal metabolism. Atrophic changes are more common in patients with common forms of tuberculosis and severe respiratory failure[10].

Many researchers note a direct relationship between the degree of lung damage and the severity of neuroimaging changes in the brain. As respiratory failure progresses, the frequency of vascular hypoxic and degenerative changes in the central nervous system increases.

The development of neurological complications in MDR pulmonary tuberculosis depends on a number of factors. The most significant of these are considered to be the duration of the disease, the prevalence of the tuberculosis process, the severity of respiratory failure and the degree of chronic intoxication.

Patients with severe forms of MDR tuberculosis are significantly more likely to experience cognitive impairment, emotional instability, and signs of chronic encephalopathy. An additional unfavorable factor is the presence of concomitant somatic diseases that worsen cerebral hemodynamics and reduce the compensatory capabilities of the body[5].

Chronic hypoxia plays a special role in the development of cerebral disorders, leading to progressive damage to neurons and vascular structures of the brain. In this regard, timely diagnosis of respiratory failure and assessment of the state of the central nervous system are important for predicting the course of the disease[9].

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The results of modern research confirm the existence of a close relationship between the degree of lung damage and the severity of cerebral disorders in MDR tuberculosis. Structural and functional changes of the central nervous system are much more often detected in patients with widespread tuberculosis.

Increased respiratory failure is accompanied by the progression of chronic hypoxia, impaired cerebral circulation and the development of vascular degenerative processes. At the same time, neuroimaging changes in the brain often correlate with the severity of changes in the electroencephalogram and clinical manifestations of neurological disorders[1,13].

The integrated use of neuroimaging and neurophysiological research methods makes it possible to assess the degree of damage to the central nervous system and predict the risk of neurological complications in patients with MDR pulmonary tuberculosis.

Conclusion. Damage to the central nervous system in MDR pulmonary tuberculosis is an important clinical problem associated with the development of chronic hypoxia, intoxication, and vascular disorders. Modern methods of neuroimaging and neurophysiological diagnostics make it possible to identify early functional and structural changes in the brain, determine the degree of cerebral damage and assess the prognosis of the disease.

The most significant risk factors for neurological complications are the prevalence of tuberculosis, the severity of respiratory failure, and the duration of the disease. Detection of early signs of damage to the central nervous system is important for the timely prevention of the progression of neurological disorders and for improving the effectiveness of treatment of patients with MDR pulmonary tuberculosis.

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