CONCEPTS OF THE PATHOGENESIS OF CHILDHOOD TIC HYPERKINESIS

Ibragimova Nargiza Abzaldjanovna
14th City Children's Clinical Hospital
Tashkent, Uzbekistan
nargiz_2004@mail.ru
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ABSTRACT

Tic hyperkinesias are one of the most common forms of childhood movement disorders and are characterized by a complex structure of pathogenetic mechanisms. The observed clinical variability, high comorbidity, and tendency for symptoms to persist into adolescence necessitate an in-depth analysis of the causal factors. Available data indicate the combined influence of genetic predisposition, developmental disorders of brain pathways, immune responses, and cortical regulation disorders. This multi-layered nature of pathogenesis requires a systematic review of research findings to clarify the mechanisms of tics and identify areas for optimizing diagnostic and therapeutic approaches

Purpose of the study

To summarize current data on the pathogenesis of tic hyperkinesis in children, taking into account genetic, immunological, neuroimaging and neurophysiological factors.

Materials and methods

Data from clinical observations, cohort studies, diffusion MRI, cytokine level assessments, electrophysiological studies, and publications reflecting structural and functional changes in the brain in tic disorders were used. A comprehensive analysis of scientific literature, including international projects and studies on movement and immune disorders in children, was conducted.

Results

It has been established that tic hyperkinesis develops based on the interaction of several pathogenetic links. A significant role of genetic predisposition has been noted, confirmed by data on the prevalence of polygenic variants and structural genomic alterations in some patients. It has been revealed that children with tic disorders exhibit persistent changes in the cytokine profile, including increased levels of IL-1β, IL-6, and TNF-α, reflecting the involvement of inflammatory processes in neuronal regulation dysfunction. Neuroimaging data revealed white matter changes—decreased fractional anisotropy in the corpus callosum, corticospinal, and association tracts—indicating impaired interhemispheric integration and motor signal conduction. Electrophysiological studies revealed increased high-frequency cortical activity, an imbalance between excitatory and inhibitory processes, and incoherence in the interactions of cortical networks, which contributes to a decrease in the effectiveness of

suppressing involuntary motor impulses. The totality of the obtained data confirms the multifactorial nature of pathogenesis, including genetic, immune and neurophysiological interactions.

Conclusions

Tic hyperkinesias in children are influenced by a complex set of interrelated biological mechanisms. Hereditary predisposition, immune activity, white matter structural abnormalities, and altered cortical regulation determine the severity of clinical manifestations and the course of the disorder. Integrating data from various studies provides the basis for further refinement of diagnostic criteria and the development of targeted therapeutic strategies.

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