амплітуда рухів в усіх суглобах зросла на $3{,}65^0 \pm 1{,}04$ в порівнянні з вихідними значеннями).

Отже, використання тейпів у реабілітаційних програмах, супроводжувалось достовірним покращенням показників амплітуди рухів в плечовому, ліктьовому, колінному та гомілковому суглобах, що супроводжується зменшенням негативного впливу контрактур у суглобах та гіпертонусу м'язів.

Список літератури

1. Cerebral Palsy Alliance launches 2023. *Research Report* https://cerebralpalsy.org.au/news-stories/cerebral-palsy-alliance-launches-2023-research-report/
2. Global prevalence of cerebral palsy: A systematic analysis https://www.ncbi.nlm.nih.gov/pmc/articles/PMC9804547/.
3. Jackman M., Sakzewski L., Morgan C., et al. Interventions to improve physical function for children and young people with cerebral palsy: international clinical practice guideline. *Dev Med Child Neurol.* 2022. May;64(5):536-549.
4. Tanochni M., Sibi D. J., Kumar P. G., et al. Predictors of Risk for Cerebral Palsy: A Review. *Pediatr Phys. Ther*. 2023 1;35(3):347-357 DOI: 10.1097/PEP.0000000000001020

УДК 616.831.4-092

PATHOPHYSIOLOGICAL ASPECTS OF SPINOCEREBELLAR ATAXIA TYPE 1

Dereka T. H.

Alexander Dubček University of Trenčín, Slovak Republic
E-mail: tetiana.dereka@tnuni.sk

Hereditary ataxias are one of the most complex sections of clinical neurology, neurogenetics, and have attracted the close attention of neurologists for more than a century and a half. Epidemiological indicators indicate that ataxias are the most common group of hereditary diseases of the nervous system, second in frequency only to hereditary diseases of the neuromuscular system. Spinocerebellar ataxia type 1 (SCA1) is a rare autosomal dominant disorder characterized by damage to the cortex, pathways, cerebellar nuclei and brainstem, and other parts of the brain. The disease is caused by the expansion of

tandem trinucleotide CAG repeats and is associated with a mutation in the SCA1 gene, which is located on the arm of the 6th pair of chromosomes, encoding the synthesis of the abnormal protein ataxin 1 [1, 2].

Aim: The aim of our study is to examine, analyze and summarize data concerning scientific developments at the level of gene and cell therapy of hereditary ataxias.

Methods: The methodological basis was: analysis and synthesis of current medical literature; systematization and generalization of practical experience data on issues of symptomatic therapy and physical rehabilitation of patients with SCA1.

Results: At the present stage, scientists are actively conducting scientific research at the level of gene and cell therapy of hereditary ataxias. At this stage, symptomatic therapy is indicated for patients with spinocerebellar ataxia type-1. Special complexes of "cerebellar" and "sensory" exercises, as well as procedures with biological feedback and stabilography are recommended. One of the methods aimed at reducing the spread of ataxias of various types is screening tests. In the future, genetic methods for correcting ataxic manifestations may appear in service.

Conclusion: Currently, the only way to slow the progression of symptoms and maintain the patient's functional status remains physical therapy programs and symptomatic support.

References

1. Bhandari J., Thada P. K. Spinocerebellar Ataxia. StatPearls Publ. 2024. https://www.ncbi.nlm.nih.gov/books/NBK557816/
2. Kerkhof L. M., Van de Warrenburg B. C., Van Roon-Mom W. C., Buijsen R. A. M. (2023). Therapeutic strategies for Spinocerebellar Ataxia Type 1. *Biomolecules.* No 13(5), 788. https://doi.org/10.3390/biom13050788.