МІНІСТЕРСТВО ОХОРОНИ ЗДОРОВ'Я УКРАЇНИ
БУКОВИНСЬКИЙ ДЕРЖАВНИЙ МЕДИЧНИЙ УНІВЕРСИТЕТ

М А Т Е Р І А Л И

106-ї підсумкової науково-практичної конференції
з міжнародною участю
професорсько-викладацького колективу
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Матеріали підсумкової 106-ї науково-практичної конференції з міжнародною участю професорсько-викладацького колективу Буковинського державного медичного університету (м. Чернівці, 03, 05, 10 лютого 2025 р.) – Чернівці: Медуніверситет, 2025. – 450 с. іл.

У збірнику представлені матеріали 106-ї науково-практичної конференції з міжнародною участю професорсько-викладацького колективу Буковинського державного медичного університету (м. Чернівці, 03, 05, 10 лютого 2025 р.) зі стилістикою та орфографією у авторській редакції. Публікації присвячені актуальним проблемам фундаментальної, теоретичної та клінічної медицини.

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< 0.0001), Apgar score at the end of the first (r = -0. 46, p = 0.0002) and fifth (r = -0.49, p < 0.0001) minutes of life; positive correlation with intraventricular hemorrhage grade II-IV (r = 0.57, p < 0.0001) and duration of mechanical ventilation (r = 0.67, p < 0.0001). Direct correlations between the sleep-wake phases and the aEEG were demonstrated in preterm infants with gestational age (r = 0.43, p = 0.0005) and postconceptional age (r = 0.49, p < 0.0001) and their formation after 32 weeks of gestation. Electroencephalographic seizures were diagnosed in 12.9% of cases, allowing timely anticonvulsant therapy.

The criteria for pathological aEEG in preterm infants with perinatal pathology are proposed, in particular: Low voltage (LV) and inactive background pattern (flat trace, FT) in children of any gestational or postconceptional age; burst suppression (BS) pattern in children of gestational or postconceptional age greater than 32 weeks; discontinuous normal voltage (DNV) pattern in children of gestational or postconceptional age greater than 36 weeks; absence of sleep-wake phases in children of gestational or postconceptional age greater than 32 weeks; seizure graph elements in children of any gestational or postconceptional age.

Conclusions. The aEEG is a mandatory method of monitoring the bioelectrical activity of the brain in patients in neonatal intensive care units, the main tasks of which are to identify the main pattern, to determine the sleep-wake phases, and to detect electroencephalographic seizures. In premature infants, the evaluation of aEEG results should be performed with regard to the physiological characteristics of brain maturation depending on the gestational age at birth and postconceptional age at the time of examination, as well as the severity of somatic pathology and the complexity of therapeutic interventions. Premature infants meeting the above criteria should be included in the program of monitoring and early intervention for early diagnosis, prevention and treatment of the consequences of damage to the central nervous system.

Chernei N.Ya.

MODERN ASPECTS OF THE TREATMENT OF DUODENAL ULCER

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Introduction. Duodenal ulcer is one of the most serious pathologies of the gastrointestinal tract. The use of already-known schemes for the treatment of peptic ulcer disease is not always sufficiently effective, therefore it remains an extremely relevant issue, especially in childhood. To date, several experimental researches have confirmed the participation of endothelial dysfunction in the formation of acid-dependent diseases, in particular, duodenal ulcers. The proven multifactorial etiopathogenesis of this disease requires not only an individual approach to each patient but also determines the need to find new treatment approaches.

The aim of the study. To investigate the effectiveness of complex treatment of duodenal ulcers using nitrogen monoxide donors.

Material and methods. In total 35 children with duodenal ulcers (ages 11 to 18 years) were examined. During esophagogastroduodenoscopy, all children were diagnosed with an ulcer defect. Depending on the treatment scheme, the children were divided into two groups: I group (17 children) – children who received therapy according to generally accepted recommendations; II group (18 children) – children who, in addition to traditional therapy, received citrarginine 10 ml twice a day after previous dilution in 50 ml of distilled water for two weeks.

The groups were representative in terms of age, sex, and place of residence. All children before the start of treatment probably did not differ in terms of clinical manifestations, endoscopic signs and indicators of endothelial dysfunction (p>0,05).

Results. After a course of complex treatment, a probably faster regression of disease symptoms was noted in children of the II group(p<0,05).In contrast to the children of the I group, in the children of the II group, the pain syndrome disappeared 4.4±0.3 days faster, the dyspeptic syndrome disappeared by 3.3±0.4 days, and the astheno-vegetative syndrome disappeared by 2.5±0.6 days (p<0,05).3 months after the treatment, a statistically significant increase in the level of NO in the blood plasma ((11.1±0.6) μmol/l, p<0.05) and a simultaneous decrease in the Et-1

content in the blood plasma were observed in the patients of the II group ((1.07±0.06) pmol/l, p<0.05).After 6 months, more pronounced changes in indicators of endothelial dysfunction were detected, Table.

Table

Indicators of endothelial dysfunction in children of the II group before and after the treatment

Indicators	Before treatment	After 6 months
NO, μmol/l	8,4±0,5	14,8±0,6*
Et-1,pmol/l	1,68±0,07	0,91±0,04*

Note.* - the difference is probable concerning the indicators before treatment(p<0,05)

Conclusions. The use of citrarginine in the complex treatment of children with duodenal ulcers accelerates the regression of the main symptoms of the disease and has a positive effect on the dynamics of endothelial dysfunction indicators.

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VULTO-VAN SILFHOUT-DE VRIES SYNDROME: CLINICAL CASE

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Introduction. Vulto-van Silfhout-de Vries syndrome (VSVS) — is an orphan disease with an autosomal dominant pattern of inheritance associated with speech and intellectual impairment, psychomotor retardation, and behavioral abnormalities, including autistic behavioral traits and poor vision. The literature describes 35 individuals with 28 different *denovo* pathogenic variants of VSVS associated with *DEAF1*. Most patients had additional nonspecific signs, including hypotension and gait disturbances, seizures, high pain threshold, and sleep disturbances.

The aim of the study. The purpose of this study was to demonstrate a clinical case of a rare hereditary Vulto-van Silfhout-De Vries Syndrome.

Material and methods. In this study, we present a boy with moderate intellectual disability, expressive language impairment, behavioral problems, autism spectrum disorder, and poor eye contact.

Results. Boy D. was born at 38 weeks of pregnancy from the III unplanned pregnancy, which was uneventful. He was born with a weight of 4500 and a length of 55 cm. He began to hold his head at 4 months, sit at 7 months, and walk at 1 year 4 months. In the history of digestive disorders were present vomiting and constipation.

Dysmorphic features included thin hair, straight eyebrows, a depressed bridge of the nose, a full tip of the nose, a drawn upper lip, a full lower lip, and a protruding upper jaw. The pads of the fingers protruded. Joint hypermobility according to the Beighton school was 6 points. In addition, there was an autism spectrum disorder, poor eye contact, sleep disorders, delayed language development, and behavioral disorders.

Chromosomal aberrations, fragile X syndrome, and aminoacidopathy/aminoaciduria associated with autistic spectrum disorders were excluded prior to whole-exome multi sequencing analysis. Whole-exome sequencing was carried out thanks to the grant Program in the 3-Billion laboratory. Result: a de novo heterozygous pathogenic missense variant was detected in the 5th exon of the *DEAF1* gene, variant NM_021008.4 c.646A>G (p. Lys216Glu) by whole exome sequencing.

Conclusions. The presented clinical case demonstrates the effectiveness of using whole-exome sequencing technology to identify the cause and verify the diagnosis of the disease. Mutations in the *DEAF1* gene should be considered in patients with a nonspecific phenotype, including intellectual disability, severe speech delay, and abnormal behavior, especially autism.