To achieve this goal, we conducted a comprehensive examination of 102 children with acute tonsillopharyngitis, who were divided into two clinical groups depending on the isolation of beta-hemolytic streptococcus group A due to the culture of smears from the mucosa of the tonsils / pharynx. The first (I) clinical group included 68 patients in whom bacteriological examination did not reveal BGSA - ATP of non-streptococcal etiology (nATP). 34 children in whom the streptococcal etiology of the disease was determined formed the second (II) clinical group - streptococcal acute tonsillopharyngitis (sATP).

The presence of a subfebrile body temperature in the child increased its post-test probability by 27.4%, and higher fever figures reduced the probability of non-streptococcal ATP by only 4.3%. The absence of symptoms of intoxication syndrome in children with a high degree of specificity - 91.4% confirms the non-streptococcal nature of the disease. Expressive layers on the tonsils are more characteristic of streptococcal ATP, and the assessment of their severity <3 points was much more common in patients with non-streptococcal disease. Using the determination of the content of C-reactive protein <50.0 mg / l in the venous blood of children as a test, allowed to verify the non-streptococcal nature of acute tonsillopharyngitis with fairly high sensitivity - 73.5% (95% CI 63.7-81.8) , however, low specificity - 35.3% (95% CI 26.0-45.5).

Indicators of local inflammation and clinical manifestations of the general inflammatory reaction were less pronounced in patients with non-streptococcal ATP. Indicators of C-reactive protein <50.0 mg / l in venous blood with high sensitivity (73.5% (95% CI 63.7-81.8) and relative risk 1.2 (95% CI 1.0- 1,5) probably testified in favor of the nonstreptococcal nature of acute tonsillopharyngitis.

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CLINICAL CASE OF SPINAL MUSCULAR ATROPHY

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Spinal muscular atrophy is a group of hereditary neuromuscular diseases based on the primary damage to the motor neurons of the anterior horns of the spinal cord and the nuclei of the brainstem in the form of their progressive degeneration and death. There are 5 types of disease. Type I, or acute malignant infantile amyotrophy of Werdnig-Hoffmann, is the most severe and common form. Frequency - 1:10000-1:6000 infants, characterized by early onset (up to 6 months) and death from respiratory failure up to 2 years. The genetic basis of the disease is a mutation in the SMN1 gene (motoneuron survival factor gene). The diagnosis of Spinal muscular atrophy can be confirmed by detecting the deletion of the 7th and / or 8th exon of the SMN1 gene in a homozygous state.

Purpose of the study: verification of the diagnosis of Spinal muscular atrophy of the Ist type by molecular genetic diagnosis.

Material and methods: DNA isolation and studies of deletions of the 7th and 8th exons of the SMN1 and SMN2 genes by polymerase chain reaction.

The geneticist was invited to consult a one-month-old boy in the neonatal pathology department of the Chernivtsi Regional Children's Clinical Hospital, who was delivered by an ambulance crew at the age of 29 days with parents' complaints about the sudden decrease in extremities movements. From the anamnesis, it is known that the boy from the first pregnancy, which took place against the background of anemia, the threat of miscarriage, toxicosis, polyhydramnios. Childbirth at 40 weeks of pregnancy. Heredity through mother and father is burdened by oncopathology. At birth, the child weight was 3600, length - 55 cm. The child's condition on the admission is of moderate severity.

At inspection the child is weak, physiological reflexes are not caused, the tone of muscles is diffusely lowered, active movements in hands are minimum, in legs - are absent. When traction by the hands, the head sags back, the head does not hold. The skin is jaundiced, more on the face, torso and proximal limbs, the sclera were subicteric. The abdomen is enlarged, the liver is +3.5 cm. Type I Spinal muscular atrophy was suspected.

Electroneuromyography was performed, myopathic syndrome was detected. The level of creatine phosphokinasein the blood - 340 IU / L. DNA diagnostics was recommended, deletion of the 7th and 8th exons of the SMN1 gene in the homozygous state was revealed. The child was examined by a neurologist (hypoxic-ischemic encephalopathy, acute period, CNS depression syndrome. Spinal muscular atrophy type I. Differential diagnosis included Pompe disease, galactosemia and intrauterine cytomegalovirus infection. The family received recommendations for further treatment and diagnosis of the child.In Italian clinics, the boy received treatment with Zolgensma, which is FDA-approved, based on a viral vector to replace the defective SMN1 gene with a working copy.

So, timely verification of the clinical diagnosis of Spinal muscular atrophy allowed clinicians to correctly interpret the neurological symptoms in a patient with myopathic syndrome and to conduct timely treatment.

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DIAGNOSTIC VALUE OF SOME CLINICAL INDICATORS IN IDENTIFYING THE RISK OF BRONCHIAL REMODELING IN CHILDREN WITH BRONCHIAL ASTHMA

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Respiratory remodeling according to the Inflammatory Theory of the disease is considered to be a multifactorial process that is realized with the participation of many cytokines, chemokines and growth factors. Remodeling is initiated by damage to the epithelium in the process of chronic bronchitis with the subsequent development of such characteristic manifestations as (1) changes in the epithelium and loss of their integrity, (2) thickening of the basement membrane, (3) subepithelial fibrosis, (4) enlargement of the goblet and submucosal glands. (5) hypertrophy and hyperplasia of smooth muscle cells, (6) increased vascularization, especially around the large bronchi.

The study aimed to assess the value of clinical indicators in identifying the risk of bronchial remodeling in children with bronchial asthma. To achieve the goal of the method by simple random sampling, a cohort of 53 children was formed, in which the severe persistent course of asthma was determined. Depending on the characteristics of the rate of acetylation processes, patients were divided into 2 clinical groups: the first (I) group included 25 patients, the second (II) group was formed by 28 school-age children.

The birth-weight of a child less than 2500 g showed high specificity of 97.4 (95% CI 92.0-99.6)% and a moderate predicted value of a positive result of 75.7% (95% CI 41.1-95.7), however, low sensitivity of 8.1% (95% CI 3.5-15.3) showed the probability of bronchial remodeling. The mentioned above birth-weight of a child indicates increased the post-test probability of possible bronchial remodeling by 24.3%, and higher birth weight was associated with a decrease in the probability of this event by 1.5%. The risk ratio with a positive test result was 3.3 (95% CI 0.79-13.7), BP = 1.56 (95% CI 0.4-6.1) with an absolute risk of 0.27.

Indications for passive smoking of children had a moderate specificity 65.8 (95% CI 55.6-75.0)%, but low sensitivity 54.0 (95% CI 43.7-64.1)%, PCPR 61.2 (95% CI 50.2-71.4)%. The posttest probability of this test was + 11.2% and -8.8%. The ratio of the risks of bronchial remodeling with a positive test value reached 2.26 (95% CI 1.3-4.0) with a relative risk value of 1.5 (95% CI 1.1-2.1) and absolute risk - 0.2.

In cases where the source of the harmful effects of tobacco smoke on the child was the mother, the test had a high specificity of 86.8 (95% CI 78.5-92.8)%, but a low sensitivity of 27.0 (95% CI 18.6-36.8)%. The accuracy of this test in detecting bronchial remodeling was 56.9 (95% CI 49.7-69.4)%, relative risk + - 2.05, and probability- - 0.8.probability of positive result increased by 17.2% and decreased by 4.3%. Indications for maternal smoking were associated with a probable risk of formation of structural changes in the bronchi: chances ratio = 2.4 (95% CI 1.17-5.04) with absolute risk = 0.2 and relative risk = 1.5 (95% CI 0.8) -2.7).