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NEUROSPECIFIC ENOLASE – EARLY DIAGNOSTICAL MARKER OF CNS DAMAGE

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Mechanisms of hypoxic damage of brain cells are characterized by a complex cascade of pathophysiological processes. Final result of this mechanism is the death of neurons due to necrosis and apoptosis. In newborn babies is not always possible to objectively assess the condition of the CNS defeat, because very often the severity of lesions does not correspond to clinical symptoms, especially in premature newborns. So far determination the severity of hypoxic-ischemic CNS lesions is still very actually in modern medicine. More objective method of such an assessment is determining the activity of neurospecific enolase (NSE).

Research purpose: to increase the efficiency of diagnosis of hypoxic-ischemic CNS lesions in premature infants by determining the activity of NSE in early neonatal period.

The concentration of NSE was determined in 15 conventionally healthy preterm infants (CHPI), which made the comparison group and 64 premature babies with hypoxic-ischemic CNS lesions on the 1-7 day of life. They were divided into three groups: I group – 26 premature children with mild CNS lesions; II group – 20 premature children with severe hypoxic-ischemic lesions and low birth weight; III group – 18 premature newborns with severe damage of central nervous system and extremely low birth weight.

Determining the level of NSE in serum of premature infants found that at the end of the early neonatal period in brain cells of children with hypoxic-ischemic CNS lesions showed destructive changes of neuronal membranes. About this evidenced significant increase the level of enzyme. So, if perinatal CNS lesions of mild degree occur, NSE content in the blood of children of I group increased by 45% relative to the comparison group ($p < 0,05$). Thus, even mild hypoxia caused a significant alteration of neuronal membranes and damage brain tissue. In the second group of infants with low birth weight on the base of severe hypoxia there was further increase activity of this enzyme in the blood, which manifested by increased serum concentrations of NSE in 2,2 times relative to the children of I group ($p < 0,001$). It should also be noted that its activity in case of severe hypoxia was almost 3,3 times higher relative to the comparison group ($p < 0,001$).

Maximum concentration of enolase reached in premature infants with very low birth weight and severe hypoxic-ischemic injury of the CNS. Its contents in serum of premature neonates of III group was 4 times greater than in comparison group ($p < 0,001$), increased 2,9 ($p < 0,001$), and 1,3 ($p < 0,05$) times relative to infants of I and II groups, respectively.

Thus, hypoxic-ischemic injury of the nervous tissue causes increased permeability of cell membranes and leave into the blood such neurospecific protein as NSE. The high rates of NSE in serum of premature infants on a base of hypoxic injury describe breach of the functional condition of cell membranes of neurons and correspond to the severity of brain damage due to hypoxia. Therefore, to assess the severity of hypoxia is necessary to determine the level of NSE in serum in the early neonatal period in premature infants.

EVALUATING THE EFFECTIVENESS OF INHALED STEROIDS IN CHILDREN WITH BRONCHIAL ASTHMA

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Properties of inhaled steroids are fundamentally different from the system hormones: they work mainly locally on the bronchial mucosa; they are in low quantity getting into the bloodstream and minimally affect the whole body. Patients don’t get used to inhaled hormones, on the contrary from systemic. More common steroids for inhalation are budesonide, beclomethasone, fluticasone, ciclesonide, flunisolide etc.

The aim of the work is analyzed literature data about effectiveness of inhaled steroids in children with bronchial asthma.

According to receptor affinity more active fluticasone and ciclesonide (22,0), than budesonide (9,4), flunisolide (1,8), and beclomethasone (0,4).

As for local anti-inflammatory action, first place occupies fluticasone and ciclesonide (1,7), than budesonide (1,0), flunisolide (0,7), and beclomethasone (0,4).

Systemic activity and potential possibility of formation systemic side effects more specific for flunisolide (12,8), and beclomethasone (3,5), less typical for budesonide (1,0) and fluticasone (0,07).

The relative bioavailability is high for budesonide (100%), also for beclomethasone (90%) and fluticasone (80-90%), but low for ciclesonide (63%) and flunisolide (21%).

Clinical efficiency of inhaled steroids was investigated in some trials. At equivalent doses all inhaled corticosteroids are equally effective (level of evidence A).

An improvement in asthma symptoms, exacerbations and side effects of different inhaled corticosteroids at equivalent doses could be neither demonstrated nor refuted and the trade-off between benefits and harms of using is unclear. The resource use or costs of different ICS should therefore also be considered in final decision making. Longer-term superiority trials are needed to identify the usefulness and safety of different inhaled corticosteroids. Additionally these studies should be powered for patient relevant outcomes (exacerbations, asthma symptoms, quality of life and side effects).

CASE OF ACUTE RHEUMATIC FEVER (ARF) THAT OCCURRED AFTER PRESUMED MACROLIDE FAILURE.

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Group A Streptococcus (GAS), also known as Streptococcus pyogenic, is one of the most common pathogenic bacteria in children and causes a broad range of infections and disease states. Standard therapy for GAS pharyngitis is penicillin or amoxicillin. Alternative therapy with significant penicillin allergy is macrolide antibiotics or possibly clindamycin. Macrolide resistance (MR) in GAS remains an increasing worldwide concern. An 11-year-oldpreviously healthy boy presented with fever, rash, joint pain, and swelling. His initial complaints were fever, sore throat, ear pain, and rash. The patient was reported to have a significant penicillin allergy. After 7 days of illness, he was empirically treated with azithromycin and diphenhydramine for 10 days. While still taking azithromycin and ∼2 weeks after initial symptoms, he developed fever, ankle pain, and swelling. Upon presentation to hospital at 21 days after initial symptom onset, he was afebrile with normal vital signs. On examination, he had notable features of a faded, evanescent, erythematous patchy rash on upper and lower extremities and splotchy truncal rash. He had pain and swelling of his left ankle, and bilateral second to fourth metacarpal phalangeal joints. His leukocyte count was 10.6 G/l , hemoglobin 106 g/l, hematocrit 30%, platelet count 409 G/l , erythrocyte sedimentation rate 51 mm/h (normal 0–20 mm/h), C-reactive protein 5.8 mg/dL (normal, 0.00–0.8 mg/dL), antistreptolysin O titer 700 IU/mL (normal 0–200 IU/mL). The results of an echocardiogram, electrocardiogram, and chest radiograph were normal. A throat culture revealed an erythromycin-resistant, clindamycin sensitive strain of GAS. He was treated with clindamycin and aspirin. He showed prompt resolution of his arthritis with resolution of the rash within 2 weeks and no recurrence. The results of a repeat throat culture were negative, and he received erythromycin as secondary prophylaxis.

Group A Streptococcal pharyngitis is a common illness in the pediatric population. Penicillin or amoxicillin remain the standard therapy. In nonanaphylactic cases of penicillin allergy, a first-generation cephalosporin may be used.