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Febrile Epileptic Status in a Child

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Summary: FIRES (DESC) syndrome is a form of epileptic encephalopathy in school–age children caused by hyperthermia. The authors describe a 9-year-old child with a resistant epileptic status on the background of hyperthermia. Somatic examination of the patient revealed no pathology, however, the epileptic status and hyperthermia did not respond to antiepileptic and antipyretic drugs, as well as antibiotics, glucocorticoid hormones and immunomodulators. Despite the spontaneous termination of the status, the prognosis for the development of resistant epilepsy in the patient remains serious.

Keywords: encephalopathy, epilepsy, status, hyperthermia, FIRES syndrome.

Severe epilepsy syndromes in children are an urgent problem for pediatric neurologists and neuro-resuscitators. This is due to the difficulties of diagnosing and treating such convulsive disorders, which have a significant pathological effect on the child's brain and almost always have irreversible consequences. In addition to FIRES syndrome, the group of pediatric epileptic encephalopathies includes IHHE syndrome (semi-seizures induced by hyperthermia, hemiplegia and epilepsy), Duse syndrome, Lennox-Gastaut syndrome and many others [1]. It should be noted that currently a separate section of the journal Epileptology is devoted to the description of FIRES and IHHE syndromes - "Acute epilepsy with temperature and inflammatory mechanisms mediated by epileptic status: AETIMSE (Acute encephalopathy with temperature and inflammation mediated status encephalicus)' [5]. [5]. FIRES syndrome is a form of severe drug-resistant epilepsy with severe cognitive impairment that develops in school-age children (5-14 years old) after high fever and often prolonged status epilepsy. The clinical picture of FIRES syndrome was first reported by Y. Mikaeloff et al. Mikaeloff et al. 2006 in the journal Epilepsy Res. [8]. Since the appearance of the first clinical case, this condition has been called DESC-syndrome (Devastating epileptic encephalopathy in school-aged children), and only five years later, as new data accumulated, the syndrome was named FIRES [9]. The authors' opinions on the frequency of occurrence of this syndrome are different: according to S.K. Yevtushenko, FIRES syndrome is not so rare, and the number of newly diagnosed cases will increase as doctors become familiar with this condition; E.V. Levitina found data in foreign literature (world literature) and considers the syndrome a rare epileptic disorder [2, 4]. To date, the concept of the etiology of FIRES syndrome has not yet been formed. The pathogenesis of FIRES syndrome is described as a combination of acute viral polycythemia, Perisilvian damage to the cerebral cortex and bilateral damage to the hippocampus due to severe and prolonged epileptic status on the background of fever [5, 6]. The autoimmune hypothesis" of this disease has not yet been confirmed, but it continues to exist [11, 12], and all authors describing FIRES syndrome agree that it has a pronounced and clearly traceable onset dynamics. The syndrome is characterized by convulsive epileptic status, which develops suddenly against the background of high body temperature. In most cases, seizures occur a few days after fever on the background of a nonspecific infection. At the beginning of the disease, focal motor seizures are characteristic with a rapid transition to a generalized convulsive seizure state. It is characterized by a dramatically rapid increase in the frequency of seizures from

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several dozen to several hundred per day, the onset of first soporotic, and then comatose disorder of consciousness. The authors of DESC syndrome themselves indicate that mortality in the acute period can reach 50%. In addition, it is noted that the condition is relatively stable, i.e. seizures continue without transitions from one state to another or completely stop [1]. There is a restoration of motor functions and an improvement in cognitive status, but without restoring the initial level. Thus, the outcome of the syndrome may be a complete cessation of seizures or a vegetative state with residual cognitive impairment after the end of the imposed epileptic status. After regaining consciousness, patients should regain the ability to walk, talk, think and remember. This process may take several months; the most objective marker of this process is S.K. Yevtushenko et al. they called bilateral hippocampal sclerosis with diffuse cortical-subcortical atrophy according to neuroimaging data [3]. In our clinical case, patient S.A. (9 years old) was admitted to the children's intensive care unit on March 31, 2015. According to the anamnesis, it is known that before the onset of the disease, the boy grew and developed adequately for his age, the child was from the first pregnancy, the birth was spontaneous in the first trimester, during which there was a secondary decrease in labor activity, the birth weight was 3800 g, the Apgar score was 8/9 points. From the anamnestic data, it follows that since March 28, the child's temperature has increased against the background of general weakness, headache and nausea. In the morning, in addition to the above symptoms, the child had multiple vomiting and monofluidic bowel movements. The pediatrician was not contacted, the child was treated at home, but against the background of the same high temperature (38.5-39 °C) he lost consciousness and had a convulsive seizure, which was stopped by paramedics with a solution of diazepam and a dissolved mixture. He was hospitalized in a hospital at his place of residence. Upon admission, his condition was severe, the level of consciousness was coma I on the Glasgow coma scale, Kernig's symptoms were bilateral. Pupils were equal, tendon reflexes were symmetrical. No seizures were observed. Spontaneous limb movements were preserved.03/31/15, in the ward, high fever 39 ° C, resistant to antipyretics, coma 1 in the morning (Glasgow 9-10 points), unable to contact. She cries regularly. The eyes are open, the gaze is fixed, but contact is difficult. Antimicrobials, symptomatic therapy, antipyretics and infusion therapy. Laboratory studies conducted at the hospital at the place of residence did not reveal significant changes, except for an increase in the number of 7-10 cl cells in 1 mm3, up to 0.3 g / l of protein in the analysis of cerebrospinal fluid and an increase in leukocytes in the clinical blood test. The patient was delivered by a specialized resuscitation team, further treatment and examination were carried out in DROMONICS, depending on the severity. Upon admission to the pediatric intensive care unit, the child was under medical sedation and at the time of examination by the neurologist was conscious, but sluggish and sleepy. The examination was negative. There was rigidity of the muscles of the occipital region, a bilateral symptom of Kernig, hyperemia of the cheeks. The patient maintained a high axillary temperature of 37.2 °C. A general blood test immediately after admission revealed anemia and moderate leukemia with a rod shift. Biochemical blood tests revealed no abnormalities. Urine analysis also revealed no deviations from the reference values. The patient was examined by an otolaryngologist and an ophthalmologist, but no pathological findings were detected. An ultrasound examination of the thyroid gland, a blood test for the hormones T3, T4 and TSH, and a measurement of the level of antibodies to TPO were performed. Goiter was detected without thyroid dysfunction. During hospitalization, a differential diagnosis of encephalitis, vasculitis and mitochondrial cell disease of various etiologies was first performed. Chest X-ray was performed, but no pathology was detected, and lumbar puncture did not reveal inflammatory changes in the cerebrospinal fluid. The results of PCR of the cerebrospinal fluid were negative for herpes, cytomegalovirus and enterovirus and, therefore, did not give grounds for a diagnosis. IgG to cytomegalovirus were detected by ELISA. The blood test for antibodies to cardiolipin, HIV infection and tick-borne encephalitis was negative. The electrocardiogram revealed signs of incomplete blockade of the right bundle branch. Later, an MRI of the brain revealed a single

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focus with paraventricular localization in the parenchyma of the left hemisphere of the brain (Fig. 1). The temperature returned to normal on the second day after admission, but after a few days it rose again to 38.5 ° C, a generalized convulsive state appeared, which was not removed with a solution of diazepam. It was decided to transfer the patient to a ventilator and administer sodium oxybutyrate 20% - 10 ml. Convulsive syndrome persisted even after the transfer to pharmacological therapy and manifested itself in the form of increased muscle tone of the limbs and trunk against a background of high temperature. Repeated lumbar punctures were performed, but no pathological changes were detected. To relieve the convulsive state, sodium valproate 2000 mg / day and levetiracetam 500 mg / day were used twice, after which the drug was canceled due to psychomotor agitation. Due to the sensitivity of microorganisms, antimicrobial therapy with meropenem 4 g / day and ceftriaxone 4 g / day was carried out. Interferon-alpha 2b was empirically prescribed as antiviral therapy. High doses of B vitamins, intravenous fluids, neurometabolic and antipyretic therapy were prescribed. Dexamethasone was added to the treatment regimen, the initial dose of which was 12 mg / day, and then was increased to 17 mg / day. The convulsive state lasted for four days, but ended spontaneously, the child was taken out from under medical sedation and regained consciousness. As a result of treatment, the condition stabilized, the meningeal syndrome regressed. Attention is drawn to the change in the child's behavior: a negative attitude towards medical personnel, medical procedures (including painless), visits to specialists and attending physicians, regular increase in appetite. These behavioral features persisted throughout the hospitalization. Electroencephalography during the first week from the onset of the disease revealed delta activity (delta coma) in the anterior leads of an even background (Fig. 2). Repeated EEG scanning performed in the second week from the onset of the disease (the patient was conscious) also showed a predominance of slow activity (Fig. 3). This case is a typical picture of FIRES syndrome: a debut at the age of 9, an initially healthy child without neurological changes, a rapidly increasing high fever without signs of intracranial infection. The epileptic status imposed on the background of hyperthermia did not respond to antiepileptic drugs, as well as antipyretics, immunoglobulins and corticosteroids, and the increased muscle tone of the limbs and trunk persisted despite drug sedation and ventilation. The data of anamnesis, clinical, laboratory and instrumental examination are consistent with the description of the syndrome in the literature [13]. On the other hand, the course of the syndrome is relatively mild, with spontaneous resolution of neurological symptoms and cognitive impairment, the severity of which has not yet been assessed. The prolonged course of drug-resistant epilepsy, requiring prolonged administration of a solution of sodium oxybutyrate, undoubtedly exacerbates the violation of higher cortical functions. It is known that there is a "light gap" during the epileptic status after the patient leaves the epileptic superimposed state, but in some cases epilepsy can quickly turn into resistant focal epilepsy. After intensive therapy, the patient was transferred to the neurological department of the hospital at his place of residence with an improvement in his condition. 10 days after discharge, an MRI of the brain was performed, but no new changes were detected. However, in the future, taking into account the peculiarities of the clinical picture and the general course, the development of bilateral hippocampal sclerosis is expected and requires dynamic verification using periodic neuroimaging studies. Such pathological changes are a typical morphological substrate of the outcome of FIRES syndrome.

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