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POST-COVID ACUTE DISSEMINATED MENINGOENCEPHALITIS IN A 2-YEAR-OLD CHILD: A CLINICAL CASE

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Among all published studies, it has been noted that in COVID-19, in addition to respiratory system dysfunction, one-third of patients (30–35%) exhibit signs of nervous system (NS) involvement. Descriptions of various neurological diseases complicating the course of coronavirus infection (COVID-19) or representing its unique clinical manifestation are available. One of the neurological complications of COVID-19 is meningoencephalitis.

This article examines the clinical presentation, diagnosis, and treatment of acute disseminated meningoencephalitis (ADME) that developed in a child following COVID-19. Special attention is given to the pathogenesis of the disease, which is associated with the body's immune response to the SARS-CoV-2 virus, leading to inflammatory processes in the central nervous system. Data from instrumental and laboratory diagnostics, as well as treatment, are presented. The importance of early detection and comprehensive treatment to prevent severe neurological complications and improve prognosis in young patients is emphasized.

Keywords: COVID-19, meningoencephalitis, central nervous system disease, neurological complications

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Introduction. Acute disseminated meningoencephalitis (ADME) is an inflammatory disease of the central nervous system (CNS) characterized by damage to the brain's soft tissues and meninges, leading to various neurologi-

cal consequences. Despite its rarity, this condition can have serious outcomes, including disability and even death, underscoring the need for deeper study of its diagnostic, therapeutic, and pathogenetic aspects.

The primary cause of ADME is considered to be an autoimmune response triggered by a viral infection. The SARS-CoV-2 virus, which causes COVID-19, can induce inflammation, leading to disseminated *encephalitis* foci. Potential mechanisms include both direct neuronal damage and the body's immune response, resulting in an attack on its own tissues.

The literature describes isolated cases of post-infectious encephalitis and meningitis following acute coronavirus infection (CVI) [1,6]. One of the most studied complications of CVI is multisystem inflammatory syndrome, including involvement of the meninges [3]. It has been reported that nervous system involvement may manifest as encephalitis, although direct evidence of SARS-CoV-2 neurotropism is lacking [1, 2].

Most cases of meningoencephalitis in COVID-19 result from an immune-mediated response to the virus, occurring when the virus enters the CNS through the blood-brain barrier (BBB) [5]. The clinical picture of meningoencephalitis may appear alongside the initial symptoms of COVID-19, including fever, depressed consciousness, neurological deficits, focal neurological signs, meningeal symptoms, and altered mental status. Cerebrospinal fluid (CSF) analysis reveals leukocytosis and cell-protein dissociation. Neuroimaging shows the presence of encephalitic foci. Most patients exhibit sinusitis or pansinusitis [1]. The question of whether meningoencephalitis is a clinical manifestation or a complication of COVID-19 remains debated.

Involvement of the central nervous system (CNS) is a harbinger of poor prognosis. Therefore, clinical assessment should focus not only on viral and infectious manifestations but also on the emergence of focal neurological symptoms.

Objective: To present a clinical case of meningoencephalitis following a coronavirus infection.

Materials and Methods. A retrospective analysis was conducted using the medical records of a patient admitted to Psychoneurological Department No. 2 of the Pediatric Center at the GAU RS(Y) "Republican Hospital No. 1 – National Medical Center named after M.E. Nikolaev". Data included clinical observations, laboratory tests, and instrumental diagnostics.

Clinical Case. A 2-year-old patient was admitted to the intensive care unit of a children's infectious disease hospital in critical condition, presenting with complaints of repeated vomiting, diarrhea, and fever up to 39°C.

According to the history, the child fell acutely ill, with symptoms beginning as repeated vomiting (up to 10 episodes), diarrhea (3-4 times daily), and fever up to 39°C. On the second day, vomiting and diarrhea persisted, and the parents called a doctor, but no medical assistance was provided. By the third day, the child remained febrile (39°C), with persistent vomiting (10 episodes), watery diarrhea, lethargy, and weakness. Emergency medical services were called, and the child was transported to the infectious disease hospital. Upon admission to the Intensive Care Unit, the child was in critical condition with worsening neurological deficits: stupor, seizures, and limited active/passive movements due to spastic tetraparesis. Muscle tone was notably increased in the arms. Tendon reflexes in the limbs were diminished. The skin exhibited pronounced marbling. Meningeal and cerebral symptoms included hyperesthesia and nuchal rigidity (1 fingerbreadth).

At the pediatric infectious disease hospital confirmed: Positive PCR for SARS-CoV-2 RNA. Concurrent rotavirus infection detected.

Treatment included: Antibacterial therapy: meropenem (20 mg/kg). Dexamethasone (20 mg/m² body surface area) for mast cell stabilization and anti-inflammatory effects. Heparin (200 IU/kg) for anticoagulation. Topiramate (2 mg/kg) for seizure control. Symptomatic therapy.

Due to the severity of the condition and clinical/laboratory improvement for COVID-19 and rotavirus infection, the child was transferred to Psychoneurological Department No. 2 of the Pediatric Center.

In the psychoneurological department, neurological symptoms persisted: the child remained stuporous, with oral automatisms, twitching of the right hand, and limited active and passive movements due to spastic tetraparesis.

Laboratory findings included leukocytosis with a left shift, anemia, erythropenia, and thrombocytopenia (77 x 10^9/L). Biochemical blood tests showed elevated C-reactive protein, creatinine (198 µmol/L), transaminases (ALT - 791 U/L, AST - 1474 U/L), and ferritin (1000 ng/mL). Urinalysis revealed proteinuria, leukocyturia, and hematuria.

From the instrumental diagnostic data on MRI of the brain: a series of tomograms obtained images of sub- and supratentorial structures of the brain and the craniovertebral junction. The topographic position of the anatomical structures of the craniovertebral junction is not disturbed. The midline structures of the brain are not displaced. In the white matter of the cerebral hemispheres periventricularly, in the subcortical section of the right frontal lobe, in the structure of the basal ganglia on both sides, multiple pathological foci are detected, hyperintense in T2WI, TIRM, isointense in T1WI, with signs of diffusion restriction, ranging in size from 3 mm to 8 mm. After contrasting, most foci do not accumulate the contrast agent, point accumulation of contrast is noted in the lesion in the right frontal lobe. The corpus callosum is of normal thickness and signal intensity. The brainstem and cerebellum have a normal configuration and signal intensity.

The lateral ventricles are slightly and uniformly dilated (width at the level of the bodies: 34 mm; width of the third ventricle: 5 mm). The fourth ventricle is midline. No periventricular edema is observed. The convexity subarachnoid spaces are moderately and unevenly widened in the frontoparietal regions. The pituitary gland is normal in size, with smooth and clear contours; its structure is unaltered. The pituitary stalk is midline. The optic chiasm and suprasellar cistern are unremarkable. The internal auditory canals are symmetrical and of normal width bilaterally. No pathological formations are detected in the cerebellopontine cisterns.

Conclusion: Multiple pathological foci in the periventricular regions, bilateral basal ganglia, and subcortical area of the right frontal lobe require differentiation between acute ischemic lesions and inflammatory foci (encephalitis). Mild ventriculomegaly. Moderate widening of convexity spaces in the frontoparietal regions.

Follow-up MRI: Regression of pathological FLAIR hyperintensities is noted in the periventricular white matter and subcortical right frontal lobe. Complete resolution of acute ischemic foci is observed.

EEG Findings: On admission to the hospital, the electroencephalogram showed a picture of the soporous stage of coma. No epileptic activity was detected.

In the dynamics of the EEG, a pronounced diffuse slowing of the bioelectrical activity of the brain in both hemispheres was noted with the replacement of the main activity by slow waves of the delta and theta range of high amplitude.

No epileptic activity was detected. EEG picture of encephalitis or diffuse encephalopathy.

Based on clinical and instrumental studies, a final diagnosis was made. Primary diagnosis: Acute disseminated meningoencephalitis (ADME), severe course. Convulsive syndrome with status epilepticus (resolved). Critical illness polyneuropathy (recovery phase). Comorbidities: 1. Mixed infection with systemic inflammatory syndrome (convalescent stage): Laboratory-confirmed COVID-19 (SARS-CoV-2 identified). Acute rotavirus gastroenteritis complicated by severe dehydration (grade 3) and toxemia (grade 2).

Dynamics of the condition and the treatment performed. The patient underwent intensive therapy, including antibacterial therapy with meropenem (20 mg/kg) and bacperazone (40 mg/kg) to treat the infectious process. Also administered: anti-inflammatory therapy, which includes dexamethasone (2 mg) and methylprednisolone (4 mg) with a gradual dose reduction to reduce the inflammatory process in the central nervous system; anticonvulsant therapy, consisting of valproic acid (30 mg/kg/day) to control seizure activity; detoxification therapy. Positive dynamics were noted against the background of treatment, with improvement in clinical and laboratory parameters.

The patient's condition at the time of discharge with clinical improvement. The child's consciousness is clear, adequate response to examination, sufficient motor activity, gaze fixes. Neurological status: diffuse muscle hypotonia, tendon reflexes are reduced in the arms and legs. The range of motion improves dynamically. The patient is able to hold his head up, sits with support. Dysarthria is observed, expressed in the pronunciation of individual syllables and sounds. The patient was discharged in a satisfactory condition with clinical, laboratory and neurological improvements, requiring further rehabilitation and observation.

Discussion. This case highlights ADME development in a child following COVID-19 and rotavirus coinfection. Combined infections exacerbated systemic inflammation and multiorgan dysfunction. ADME pathogenesis involved both immune-mediated mechanisms and indirect hemorheological effects of SARS-CoV-2, compounded by rotavirus.

The clinical picture was characterized by severe neurological deficit, seizures and pronounced systemic manifestations. It is important to note that ADME can develop both in the early stages of infection and against the background of the leveling of general infectious symptoms. Diagnosis of ADME requires a comprehensive approach, including clinical assessment, laboratory tests (CSF analysis, general and biochemical blood tests) and neuroimaging (MRI of the brain). Ischemic foci in the brain could arise due to microthrombosis, endothelial dysfunction and systemic hypoperfusion. The issue of penetration of the SARS-CoV-2 virus through the blood-brain barrier remains controversial. In this clinical case, PCR diagnostics of the virus in the cerebrospinal fluid (CSF) was not carried out, which does not allow this hypothesis to be unambiguously confirmed [4]. However, existing studies and clinical observations provide data supporting the possibility of virus translocation through the blood-brain barrier [3]. Early diagnostics and complex therapy, including anti-inflammatory, anticoagulant and neuroprotective agents, are key to improving the prognosis in such patients. In dynamics, 6 months after the primary disease, neurological symptoms completely regressed, and rehospitalization to the psychoneurological department was not required.

The authors declare no conflict of interest in the submitted article.

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