Subacute Sclerosing Encephalitis in an Adult with Congenital HIV Infection – Case Report

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Abstract: Subacute sclerosing panencephalitis (SSPE) is the result of a chronic infection of the central nervous system caused by a mutated measles virus. We present a case of SSPE in a 19-year-old female with congenital HIV-infection. The patient has been using antiretroviral therapy regularly since she was 4 years old. At the age of 15, she contracted measles of moderate severity. At the age 17, she had the HIV viral load < 20 copies/ml and the CD4 count 420 cells/μl. Three years after measles, bilateral necrotizing retinitis developed, and five months later, myoclonic seizures of the left limbs. Symptoms progressed gradually, with fever, generalised seizures, and lost consciousness. She was hospitalized in department for treatment patient with HIV-infection. Magnetic resonance imaging of the brain revealed massive areas of the altered signal without clear contours in both hemispheres of the brain, which captured the white and grey matter of the fronto-parietal, temporal-occipital lobes. The electroencephalography showed the flashes of slow-wave paroxysmal activity of the brain in the delta range, mainly in the fronto-parietal area. In the cerebral spinal fluid, anti-measles IgG was detected at a titre of 3738,408 U/ml, and in the blood – 9.4 U/ml. A diagnosis of SSPE was established. Supportive, corticosteroids and anticonvulsant treatment were ineffective. Patient died 10 months after the onset of the disease. Therefore, measles at any age in a person with congenital HIV-infection poses a risk of developing SSPE.

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Introduction

Subacute sclerosing panencephalitis (SSPE) is a severe, fatal nervous system lesion associated with the measles virus. It is caused by a mutated measles virus that constantly replicates in the cells of the nervous system. It is accompanied by damage to the white and grey matter of the brain, sometimes the thalamus, brainstem and spinal cord. The largest number of cases of SSPE was registered in children who contracted measles under age of 5. The frequency is 1:1,367 if children were affected under 5 years old, and 1:609, if they were under 12 months old. SSPE may occur after median latency period 9.5 years (2.5–34 years) at a median age of 12 years (3–35 years). Men get sick more often than women by 2.4 time (Wendorf et al., 2017). The incidence of SSPE in the world varies from 0.2 to 40 cases per million population per year and depends on the measles vaccination coverage in different countries (Alexander et al., 2020). Vaccination shortages in US (United States) led to an increase in measles incidence to 120 per million in 2019, the highest level since 2001 (Phadke et al., 2020). During COVID-19 epidemic in the UK (United Kingdom), the number of people vaccinated against measles-mumps-rubella in 2020 was 19.8% lower compared to 2019 (McDonald et al., 2020).

The pathophysiology of the disease is not fully understood, but there is evidence that the virus that causes SSPE is a hypermutated measles virus. Compared to the wild-type strain mutations involve the matrix protein (M), hemagglutinin (H), nucleocapsid (N) and membrane fusion protein (F) genes. The mutated protein F plays an exceptional role in the spread of the virus in nerve cells. The definitive receptor for the penetration of the virus into the nerve cell is unknown, perhaps it is a neurokinin-1 receptor (substance P is the mediator of the neurokinin-1 receptor), which are densely represented in interneuron synapses (Watanabe et al., 2019). It is suggested that presentation of cell adhesion molecule 1 (CADM1) and CADM2 on neuronal membrane may promote membrane fusion and the spread of measles virus (MeV) in cells. However, the exact mechanism of neuronal infection and virus spread in nervous tissue remains unknown (Shirogane et al., 2021; Takemoto et al., 2022).

Inadequate cellular immune response plays a crucial role in pathogenesis. This leads to the survival of the virus in the cells of the nervous system despite the strained humoral immunity. There is a lack cytokines of cellular immunity such as INF, IL-2, Il-10, Il-12, and excess levels of IL-4 and IL-1b, which stimulate factors of humoral immunity. Therefore, premature switching of the cellular immune response to the humoral one leads to a loss of the ability to eradicate the virus from the nerve cells (Hübschen et al., 2022).

Pathological examination of the brain reveals numerous glial nodules in the grey and white matter of the hemispheres, in brainstem and cerebellum ("nodular panencephalitis") with focuses of demyelination in subcortical formations (leukoencephalitis). Perivascular lymphomonocytic infiltration, damage and degenerative changes of neurons, and proliferation of glia were revealed. Apoptosis and oxidative stress may contribute to early neuronal damage, while lipid peroxidation and impairment glutamate transport can cause subsequent neurodegeneration (Kramarev et al., 2018). Clinical manifestations appear on average 6 years after infection with the measles virus. The disease progresses relentlessly and ends in death. It usually lasts from 6 months to 2–3 years. The clinical course of the disease includes 4 stages:

- stage I – personality changes, failure at school, strange behaviour;
- stage II – massive, repetitive, and frequent myoclonic jerks, seizures and dementia;
- stage III – rigidity, extrapyramidal symptoms, and progressive lack of unresponsiveness;
- stage IV – coma, vegetative state, autonomic failure, and akinetic mutism (Kramarev et al., 2018).

The literature describes the development of necrotizing retinitis against the background of subacute sclerosing panencephalitis. In most cases, retinitis with loss of vision joins later, already in the advanced stages of the disease (Fisher et al., 2015; Rana et al., 2023). Initially, one-sided damage to the eyes is possible, but the process quickly becomes bilateral and after 2 months ends with retinal atrophy of both eyes (Agarwal et al., 2017). In young adults, the disease does not progress as quickly as in children, but is also characterized by behavioural changes, cognitive deficits, and progressive neurological symptoms (Martins et al., 2018).

Electroencephalography (EEG) shows characteristic changes: periodic discharges of slow, spike-end waves of high amplitude every 3–8 seconds, which are replaced by short periods of reduced activity. EEG has diagnostic value in the myoclonic phase. The first type of EEG is characterized by periodic complexes consisting of bilateral, symmetrical, synchronous, high-voltage (200–500 mV) bursts of polyphasic, stereotyped delta waves. These complexes are regular with an interval of 4–10 seconds and correspond to myoclonic jerks. The second type of EEG is less common and is characterized by periodic slow giant delta waves combined with rapid spike activity. Type III is periodic, characterized by a long spike wave of discharge interrupted by giant delta waves. This type

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is characterized by rapid progression of the disease (Garg et al., 2022).

Neuroimaging in SSPE is not specific. In the early stages of the disease, brain magnetic resonance imaging (MRI) is usually normal. The first manifestations of a diffuse hyperintense signal occur in the white matter of the posterior parts of the cerebral hemispheres, especially in the parietal-occipital and posterior temporal regions, and later it moves to the frontal lobe, corpus callosum, and basal ganglia. Early involvement of grey matter in the pathological process is observed. The pathological process ends with cerebral atrophy. The final diagnosis requires the demonstration of elevated titres of antibodies against measles in the cerebrospinal fluid (CSF) (Garg et al., 2019).

Many drugs have been used to treat and stabilize the course of the disease, but without evidence in randomized clinical trials. Ribavirin, inosine pranobex, or intravenous human immunoglobulin were used. The combination of inosine pranobex and intrathecal administration of alpha interferon gave the best effect in relieving the symptoms of the disease (Sliva et al., 2019; Papetti et al., 2022).

Cases of subacute sclerosing measles panencephalitis in HIV-infected patients are described infrequently, mostly in children with congenital HIV infection. Moreover, children suffered from measles in early childhood. For example, a case of SSPE in a 17-year-old boy with congenital HIV infection and effective antiretroviral therapy (ART), who contracted measles at the age of 1.5 years. The disease developed rapidly and ended fatally 12 weeks after the onset (Sivadasan et al., 2012). There is an opinion that clinical course of SSPE depends on the degree of HIV-associated immunosuppression (Muthusamy et al., 2015).

During 2017–2018, the many incidences of measles occurred in countries of EU (European Union). For example, there were 8,274 cases in Romania, 4,885 cases in Italy, and 919 cases in Germany. In Ukraine at that time (until September 2018), 30,744 people fell ill including 18,136 children and 12,608 adults. Complications from the nervous system in the form of meningitis, meningoencephalitis, and delayed damage to the CNS (central nervous system) by the measles virus occurred in 1/1,000 patients (Prokopiv et al., 2019). According to medical records in one of retrospective study in Ukraine, 15.1% patients did not receive any dose of measles vaccine, 26.5% received 1 dose at childhood age, 15.9% had 2 doses, and in 42.5% of cases vaccination information was missing (Pryshliak et al., 2020). Since 2014, less than half of Ukrainian children have been vaccinated against measles. According to UNICEF in Ukraine, the number of children under year old, which vaccinated against measles, does not exceed 42% (Andreychyn, 2020). Therefore, the main reason of measles outbreaks in Ukraine in recent years was the low level of vaccination.

Case report

Female, 19 years old, was born on January 14, 2004. The HIV infection was diagnosed in the girl at the age of 2 years (2006), through index testing after the diagnosis of HIV infection in her mother. At the age of 4 years, on August 3, 2008, she started ART with scheme AZT+3TC+LPV/rtv. In 2019, at the age of 15, she suffered from measles, of moderate severity. In 2019, the CD4 count was 280 cells/μl, and HIV viral load (VL) was < 20 RNA copies/ml. In 2020, the ART regimen was replaced by TNF+3TC+DTG. On March 29, 2022, the CD4 count was 420 cells/μl and VL < 20 copies/ml. In April 2022, visual disturbances appeared, there was no fever. She was hospitalized at the Ivano-Frankivsk Regional Clinical Infectious Disease Hospital, where the diagnosis was established: HIV infection, clinical stage II. Ophthalmologist (March 30, 2022) – neuroparalytic mydriasis, neuroretinitis of both eyes.

Blood tests for infectious agents: DNA of Epstein-Barr virus (EBV), *Toxoplasma gondii*, herpes simplex virus 1 or 2 type (HSV 1/2), cytomegalovirus (CMV), a-*Borrelia burgdorferi* IgM, IgG were negative. Chest X-ray, abdomen ultrasound, general clinical and biochemical tests were without specifics changes. Neurologist (March 31, 2022) – no focal neurological symptoms were detected. MRI of the brain (March 31, 2022) – no pathological changes in the signal of the brain substance were detected.

She was examined twice at the Institute of Eye Diseases in Odesa, Ukraine. The diagnosis was the same and the treatment was unsuccessful. Her health gradually worsened, irritability and personality disorders appeared, her memory and academic performance at the university deteriorated.

In September 2022, in the 5th month of the onset of retinitis, twitching in the left hand and the left corner of the mouth and dysmetria of the left hand appeared. She was observed by a neurologist. Tendon reflexes were brisk on both sides symmetrically. No pathological foot reflexes were found. Stato-coordinator tests were satisfactory. The diagnosis was established – symptomatic epilepsy with focal motor seizures with awareness (left-sided myoclonus?). On September 9, 2022, levetiracetam 500 mg twice a day was prescribed.

EEG was performed three times with the conclusion: outbursts of slow-wave paroxysmal activity in the

delta range with an amplitude of 300 μV, mainly in the fronto-parietal area with a transition to the temporal and occipital areas, which coincided with involuntary muscle contractions of the left limbs. The condition improved somewhat, myoclonus decreased, but there was crying, a change in mood. The neurologist regarded this as a side effect of levetiracetam and added lamotrigine 50 mg 2-time a day, but there was no effect. On November 10, 2022, clonazepam 1 mg twice a day was additionally introduced into the treatment regimen. In relation to myoclonus, the condition improved significantly but temporarily. However, the patient's condition gradually worsening, generalized convulsions appeared. In December 2021, the body temperature periodically rose to 40.0 °C, the convulsions did not stop, the disturbance of consciousness increased. In December 2022, severe stiffness of the left limbs and spasm of the muscles of the right half of the face, tonic convulsions of the limbs, severe hyperthermia (up to 40.0 °C) with pronounced oiliness and sweating of the skin appeared. She was hospitalized on January 21, 2023 – in the Communal non-commercial enterprise "Center of Infection Diseases of Ivano-Frankivsk Regional Council". During hospitalization, the patient's condition was severe, she was unconscious with readiness for convulsions, her body temperature was 38 °C.

A preliminary diagnosis was established: HIV infection, clinical stage IV. Meningoencephalitis is probably toxoplasmic. Signs of retinal degeneration. Symptomatic epilepsy. Oropharyngeal candidiasis. Bilateral pneumonia? Tuberculosis?

Trial antitoxoplasmosis therapy was prescribed: dalacin 600 mg 4-times a day, TMP/SMX-480 4 tablets 3-times a day. Also, fluconazole 200 mg per day, and dexamethasone 16 mg daily intramuscularly for 7 days, then 12 mg for 5 days, after that 8 mg. Sibazon was prescribed as an anticonvulsant.

Blood analysis for infectious agents (January 21, 2023): CMV DNA, HSV 1/2 DNA, EBV DNA, *Toxoplasma gondii* DNA, *Mycobacterium tuberculosis* DNA were negative. Blood ELISA tests (January 22, 2023): a-CMV IgG – 9.42 (with a norm 0.8–1.0), and a-CMV IgM, anti-*Toxoplasma gondii* IgG and IgM, anti-HSV 1/2 IgG and IgM, a-*Chlamydia trachomatis* IgG and IgA, anti-HBsAg, anti-HCV total Ig, antibodies to *Treponema pallidum* were negative. In blood D-dimer was 300 ng/ml (N < 250 ng/ml) (on January 24, 2023). The HIV viral load was determined to be ≤ 20 copies/ml and CD4 T-cells count was 218 cells/μl (January 21, 2023). Complete blood count (CBC) on 21.01.23 and 30.01.2023 were normal.

CSF (22.01.2023): volume – 0.5 ml, colourless, transparent, Pandey's test – negative, cytosis – 4 per μl, lymphocytes – 76%, segmented cells – 24%. PCR examination of CSF: DNA CMV, EBV, and human herpes viruses (HHV) – 1, 2, 3, 6, 7 types, MBT (*Mycobacterium tuberculosis*), *Toxoplasma gondii* were negative. Bacteriological examination of CSF – culture without growth. Microscopy for cryptococcus was negative, no cryptococcal antigen was detected.

Chest X-ray twice (at 21.01.2023 and 23.01.2023) – no pathology was found. Abdominal ultrasound (23.01.2023) – moderate diffuse changes in the liver and pancreas were found. Ultrasound of the heart (23.01.2023) – no pathologies were detected.

Ophthalmologist (01.23.2023) – anisocoria, neuroretinitis of both eyes. Neurologist (01.23.2023) – meningoencephalitis? Symptomatic epilepsy. MRI of the brain is recommended.

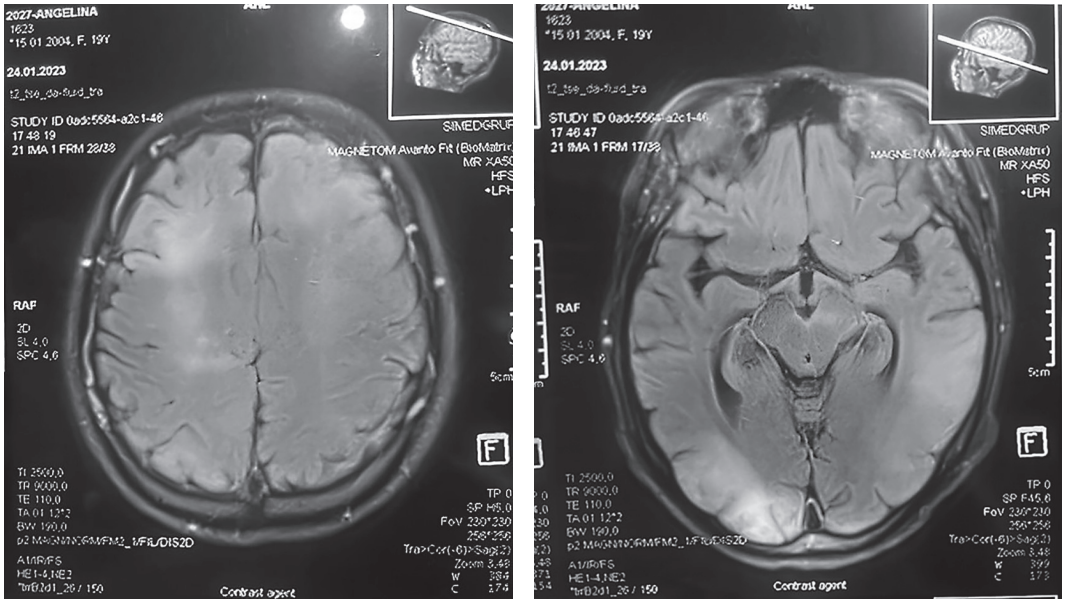

Figure 1: Brain magnetic resonance imaging of a 19-year-old perinatal HIV-infected female with subacute sclerosing panencephalitis.

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MRI of brain (24.01.23): in both hemispheres, at the level of the fronto-parietal, temporal-occipital lobes, there are massive areas of the changed signal without clear smooth contours, which capture the white and grey matter of the brain, mostly weakly hyperintense. There is a weakly hypertense signal in the T2 and T2 TIRM modes, in the diffusion-weighted imaging (DWI) mode, slightly hypointense in the T1 mode. After intravenous contrast in the T1 mode, the presence of a slight contrast enhancement along the course of the gyri in the parietal-occipital area on the left side is noted. The middle structures are not displaced. In the right maxillary sinus, there is content up to 0.45 cm thick. Conclusions: MRI signs are more consistent with encephalitis in large hemispheres of the brain. Progressive multifocal leucoencephalopathy? Slightly pronounced left-sided maxillary sinusitis (Figure 1).

Anti-measles IgG was detected in CSF – 3738,408 U/ml and in the blood – 9.4 U/ml (01.02.2023).

Against the background of treatment, the patient's condition remained serious. Coma I-II. No positive dynamics were noted. The patient died on 02.02.2023.

Final diagnosis: HIV infection, III clinical stage. Subacute sclerosing panencephalitis, severe course. Symptomatic epilepsy. Neuroretinitis of both eyes, retinal degeneration, neuroplastic mydriasis. Oropharyngeal candidiasis.

Discussion

The special contingent of children with congenital HIV infection in Ukraine is quite significant. By the end of 2022, 3,334 children with congenital HIV infection were registered in Ukraine (Center for Public Health Kyiv, 2023).

According to the vaccination schedule, when the level of CD4+ T-cells is < 200 μl, live vaccines cannot be administered to HIV-infected children (Ministry of Health Protection of Ukraine, 2022). Therefore, many HIV positive children in country may not be vaccinated with measles, mumps and rubella vaccine (MMR).

After measles the insufficiency of cellular immunity in HIV-infected people can contribute to the replication of the mutated measles virus in the CNS (Hübschen et al., 2022). In patients using ART HIV replication in glial cells of the brain can continue despite aviremia in the blood and cause locally immunodeficiency in the brain (Wahl and Al-Harthi, 2023).

On the basis of the described case of the disease, it is possible to establish a risk of SSPE in persons who contracted measles in adolescence. This risk persists regardless of the number of CD4+ T-lymphocytes and the regular use of ART, because of all patient's cases describing in the literature regularly received ART (Sivadasan et al., 2012; Muthusamy et al., 2015). It is likely that the majority of childhood HIV-infected patients were vaccinated with MMR at 12 month or 6 years old, although reliable vaccination data are often not available (Muthusamy et al., 2015). In the reported case the possibility of vaccination the child at the age of 12 months also cannot be excluded, because of at that time the child was not diagnosed with HIV. So, there was no contraindication for MMR vaccination.

This patient suffered with measles at the age of 15 years, and 3 years later SSPE developed. On admission to the hospital the diagnosis of toxoplasmosis encephalitis was incorrect but acceptable at that time because of unknown etiology of brain lesion. Diagnosis of the IV stages of HIV was also incorrect, but corresponded to hypothetical toxoplasmosis encephalitis. Antitoxoplasma treatment was carried out 10 days and cancelled due to ineffectiveness.

The clinical picture of this case of the disease had its own characteristic. The disease began with severe chorioretinitis, marked loss of vision in both eyes and paralytic mydriasis. Other clinical manifestations were typical: personality disorders, loss of cognitive abilities, deterioration of academic performance, myoclonus, and in the terminal period loss of consciousness, comatose state and fatal outcome. The patient's disease lasted 10 months. The correct diagnosis of SSPE was established at the final stage of the disease, 4 days before death, because of lack of vigilance of physicians to such a pathology. The EEG study which registered a pathological delta rhythm and bursts of activity corresponding to myoclonus was quite informative. A confirmatory study was the detection of intrathecal antibodies to the measles virus in high titres.

Conclusion

Individuals with congenital HIV-infection are at risk for SSPE if they have a history of measles-like illness at any age. For the timely diagnosis of SSPE, it is necessary to conduct an examination of the cerebrospinal fluid and blood for the detection of intrathecal antibodies to the measles virus.

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