

Review Article

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Neurological Symptoms of Zika Virus Infection at Birth

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Abstract

Objective: In 2015, it was noticed an ascent in the quantity of micro cephalic infants related with a background marked by nonspecific febrile affliction and rash during pregnancy in Brazil. Since then, microcephaly has become a major concern for public health. The connection between the Zika virus and congenital microcephaly was discovered a few months later. A brand-new TORCH member, the arbovirus Zika infects the developing brain, disrupts synaptogenesis, and causes other lesions of the central nervous system. It causes congenital infection through vertical transmission. The objective of this article is to report the Congenital Zika Syndrome (CZS) and to emphasize the importance of following up with affected children to better understand the evolutionary history of this new agent, optimize healthcare delivery, and enhance patient well-being.

Methods: To characterize the congenital Zika syndrome and recommend the systematization of some examinations and procedures for evaluating children exposed to ZIKV with or without microcephaly, based on the author's own experience, we conducted a review of the most pertinent literature regarding clinical manifestations and neuroimaging findings related to neurotropism of the Zika virus.

Conclusions: Vertical ZIKV infection can result in a wide range of neurological symptoms that go beyond microcephaly. Even children who do not have microcephaly should be monitored during their first few years of life because infection can cause neuropsicomotor delay, epilepsy, and visual abnormalities or be asymptomatic. The objective of the appropriate prospective multidisciplinary follow-up of these patients is to comprehend the natural history of this new agent and to improve their development and quality of life for themselves and their families.

Keywords: Microcephaly; Synaptogenesis; Congenital infection; Neurotropism; Neuropsicomotor; Neuroimaging

Introduction

The Zika Virus (ZIKV) is a brand new TORCH member that causes congenital infection through vertical transmission and harms the developing brain by interfering with the multiplication and migration of nervous system cells, accelerating apoptosis, altering the characteristics of CNS myelin formation, and disrupting synaptogenetic activity. The CNS is exceptionally powerless to diseases during all the gestational period; however contamination during the prior long stretches of the early stage for the most part results in more serious abnormalities. Small brains with extensive destruction, poorly differentiated cortex, few gyri, and a small skull are the result of infection. Consequently, the newborn's natural development and the achievement of developmental milestones may be severely impeded. This article examines the neurological characteristics of the Congenital Zika Virus Syndrome (CZS) in infants and young children. It also aims to emphasize how proper management can improve these patients' healthcare, life expectancy, and quality of life. Guillain Barre Syndrome, acute myelitis, and numerous other neurological disorders unrelated to intrauterine infection may be caused by ZIKV [1,2].

Zika virus syndrome during birth

A rise in the number of newborns in Brazil with a small head circumference (microcephaly) and a history of febrile illness and an itchy rash during pregnancy was observed at the end of 2015. Microcephaly has emerged as a public health issue since then. The association between congenital microcephaly and the ZIKV, an arbovirus, was discovered a few months after the increase in newborns with microcephaly in northeast and southeast Brazil was made public.

In addition, the pediatric neurology clinic is well-known for its treatment of microcephaly. Prenatal (congenital), perinatal, and postnatal events, including asphyxia and neonatal meningitis, all play a role in the disease's complex and multifactorial etiology. Genetic anomalies, disruptive traumas, teratogens (including alcohol, drugs, radiation, gestational diabetes), and congenital infections are all potential congenital causes. Microcephaly was only one occurrence of what was defined as congenital Zika virus syndrome [3,4].

Microcephaly state

In CZS, the head circumference can be normal or severe microcephaly, with a head circumference that is 3 Standard Deviations (SD) below the mean and a significant craniofacial disproportion. The most common symptom is a halt in brain development, particularly in the frontal, temporal, and parietal lobes. It causes the cranial bone plates to collapse, resulting in a biparietal depression as well as an occipital prominence (occipito-parietal crest).

Neurological outcomes

Beyond microcephaly, congenital ZIKV infection can result in a wide range of neurological manifestations. Patients might give create mental defer saw in the main long periods of life, for example, hyperexcitability and expanded muscle tension. Hyperreflexia, clonus, and the former may resemble epileptic seizures. The last option might be serious to the point that permits patients to settle neck and truncal acts still in the infant period, which can incorrectly propose an early

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accomplishment of engine achievements while, as a matter of fact, these dadtients have more extreme pyramidal parcel injury. Youthful infants can have exacerbated crude reflexes (getting a handle on, setting, venturing, Moro's reflex, search reflex, among others) that vanish later than anticipated [5,6].

Infants typically exhibit intense, difficult-to-soothe crying when they are extremely irritated. Irritability is exacerbated by posture disorganization. As the patient gets older, the physiological functions of the nervous system don't develop properly, and the patient develops cervical hypotonic and keeps the appendicular hypertonia. This makes it hard for the patient to reach developmental milestones, like in people with cerebral palsy.

Mental capabilities like social communication and intentional correspondence might be impacted in microcephaly patients, and furthermore in non-microcephaly tainted children. Some of these kids have been said to have sensory problems. The brainstem neurons' dysfunction is the secondary cause of sensor neural hearing loss or hypoacuity. A pale optic disk, chorioretinal atrophy, and colobomas are among the ophthalmological abnormalities that have been reported in children who are both microcephaly and non-microcephaly. In a subsequent chapter, eye abnormalities will be discussed in greater detail [7,8].

Dysphagia and micro aspirations can occur when a child's sucking and swallowing reflexes disappear around the age of four months and their motor incoordination worsens. As a result, gastrostomy may be required due to increased risk of malnutrition and recurrent pneumonia. Seizures with epilepsy are common symptoms.

It is common for epileptic seizures to begin as subtle startles or spasms that the family may not notice or misinterpret as nonepileptic events. They usually become more and more difficult to treat, eventually developing into complex, focal or generalized seizures that are usually resistant to anti-epileptic medications. While others develop Lennox-Gastaut syndrome, which results in even more limitations, some patients exhibit flexion spasms, which are typical of infantile spasms (West Syndrome), with or without hypsarrhythmia. Motor incoordination or injury to the basal ganglia may cause tremors. Video Electro Encephalogram (VEEG) is a very useful test in this population because it can be difficult to tell if it is an epileptic equivalent in these circumstances. In severe cases, arthrogryposis and other osteoarticular malformations are frequent findings that account for some patients' poor intrauterine movements [9,10].

Diagnosis using brain imaging techniques

All patients should have a CT or MRI because of the variety of neurological manifestations, the possibility of severe developmental impairment, and for diagnostic purposes. In the event of an open anterior fontanel, cranial ultrasound can also be performed, but its resolution is lower. Imaging findings include more frequently calcifications of the juxtacortical and basal ganglia, cortical and subcortical atrophy, particularly in the frontal, parietal, and occipital lobes and cerebellum, ventricular dilation, pachygyria, polymicrogyria, and a thin brainstem due to a reduced number of fibers.

Clinical follow up stage of the patient

The patient should be able to be identified, prioritized, and referred

to specialized care by a pediatrician or primary care physician. The clinician is able to monitor the child's development and spot early signs of malnutrition, reflux, feeding issues, and developmental anomalies by consulting with the child on a clinical basis once every three months or as often as necessary. The neuropsychological development, possible seizures, tonus abnormalities, and the need for pharmacological and multidisciplinary treatment will all be evaluated by the pediatric neurologist. The Head Circumference (HC) should be measured frequently: monthly, during the first trimester, and at the very least every three months thereafter. Even if there are no developmental abnormalities, patients with normal HC should have their cranial growth rate checked. A disproportional increase in cranial growth and tension at the fontanels, both of which suggest hydrocephalus, must be evaluated in patients with microcephaly. A Ventriculo Peritoneal Shunt (VPS) may be necessary in this situation.

Conclusion

Clinical and radiological manifestations of CZS may be comparable to those of other TORCH group congenital infections. The objective of the appropriate prospective multidisciplinary follow-up of ZIKVexposed patients is to gain a deeper comprehension of the infection, its natural history, and the appropriate clinical, pharmacotherapeutic, and surgical interventions that may improve their development and quality of life for themselves and their families. In contrast to the other infections in the group, the true impact of this disease is still unknown. Therefore, it is of the utmost importance to monitor and evaluate all children exposed to the ZIKV, not just those with CNS abnormalities that can be detected.

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