International Journal of Medical and Health Research
www.medicalsciencejournal.com
ISSN: 2454-9142
Received: 21-09-2021, Accepted: 05-10-2021, Published: 22-10-2021
Volume 7, Issue 10, 2021, Page No. 19-21

Congenital *Cytomegalovirus* (CMV) infection→still the silent killer

Salma Hossain[1], Rebecca Akhter[1], Shamima Sultana[2], Sadia Binta Rahman[3]

[1] Assistant Professor, Department of Pediatrics and Neonatology, Ashiyan Medical College Hospital, Dhaka, Bangladesh
[2] Indoor Medical Officer, Department of Pediatrics, Dhaka Medical College Hospital, Dhaka, Bangladesh
[3] Assistant Professor, Department of Pediatrics, Mugda Medical College Hospital, Dhaka, Bangladesh

Abstract

Congenital *Cytomegalovirus* (CMV) infection is the most common congenital infection worldwide. It affects every 150-200 live births in developing countries. Yet there is low public and healthcare provider awareness about this preventable disease. It mainly affects the central nervous system and causes microcephaly, mental retardation. Progressive and permanent SNHL, chorioretinitis and developmental delay. Here we present a case of a 15 months old girl with repeated convulsion, hearing loss, visual impairment, motor delay, and mental retardation organomegaly and growth failure. CMV is seen in approximate 1% of all newborns. From 10%-15% of congenitally infected newborn cases are symptomatic at birth. Among them 90% having various types of sequels like hearing loss (most common) to severe mental retardation, motor delay chorioretinitis (inflammation of choroid and retina), behavioural disorder and seizure.

Keywords: congenital *Cytomegalovirus* (CMV); perinatal; neonatal

Introduction

The most common non-genetic cause of sensory-neural hearing loss and neurodevelopmental sequelae is congenital *Cytomegalovirus* (CMV) infection. Despite these troubling statistics, the general public healthcare system is frequently unaware of CMV and not doing enough to prevent congenital CMV infection. Ninety percent of the symptomatic cases have some form of sequelae, ranging from isolated hearing loss to severe mental and/or motor impairment. Intrauterine growth retardation, chorioretinitis, neural hearing loss, prematurity, hepatosplenomegaly, jaundice, microcephaly, and intracranial calcification are all symptoms of this condition [1–4]. Congenital *Cytomegalovirus* (CMV) infection affects about one percent of all newborns. Between 10% and 15% of congenitally infected newborn cases are symptomatic at birth [5].

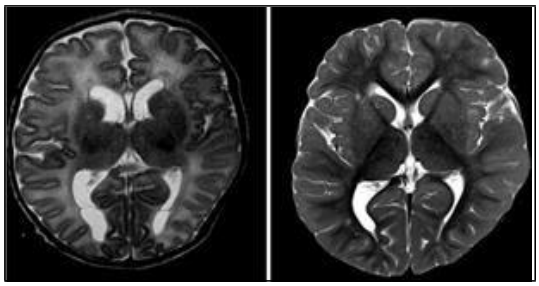

Fig 1: (a) Corresponding axial T2W images of the same child showing evolution of congenital CMV abnormalities. (b) Follow-up scan at the age of 2 years.

Case Presentation

Fatema Tabassum a 15 months old female patient with only issue of consanguineous parents presented with repeated myoclonic jerky movements, hearing and visual loss, non-responding to surroundings, motor delay, (can't stand even at 15-month age) mental retardation and failure to thrive. Mother had a caesarean delivery at term after trial of normal delivery at home. Due to delayed cry newborn received inpatient treatment at the neonatal intensive care unit for 3 days. She had no H/O fever, rash, offensive body or urine odours, repeated vomiting or trauma. On physical examination, her weight, height, head circumference, body temp, pulse respiratory rale and blood pressure were determined to be 8.6 kg (5th centile), 68cm (<3rd centile), OFC (<3 centile) 36 degrees C (axillary), pulse 100/m R/R 25/mint and BP=80/50 mm respectively. So, anthropometry revealed severe underweight and stunted and microcephaly. She has organomegaly, developmental assessment showed gross motor function corresponding to 7 months of age. She achieved neck control at 5 months. Bilateral chorioretinitis was determined at the ophthalmological examination. She has low vision and hearing, can only utter some sound. Still, she has no social smile.

Investigation

From lab examination, haemoglobin, white cell count and thrombocyte count were determined to be a 9 gm/dl, TC=15000/cumm3 and 2 lac respectively. Nuclear leukocyte, lymphocyte, monocyte and eosinophil rates were 65%, 30%, 5%, 2% and no atypical cell was determined. ESR was 10 mm CRP was negative. Blood biochemistry value for glucose 90mg/dl, creatine 0.39 mg/dl, urea (15 mg/dl), creatinine (18 U/L), alanine aminotransferase (39 U/L) as per rate 260 mmol/L, alkaline phosphatase (34mmol/L), sodium (1.35 mmol/L)), potassium (3.4 mm/L) phosphorus (5.7 mg/dl) and calcium (9.6 m/dl) of normal. The urine exam was normal, no abnormality was found in stool microscopy. Hepatitis markers are HBS Ag (-ve). Regarding torch-screening toxoplasmosis, Epstein Barr Virus, Herpes simplex virus type I and Type II, Rubella, parvovirus B19 and HIV serologies were all negative. Serum CMV IgM was positive IgG was positive (104.00 Au/ml) (Ref→<15 AU/ml) and urine CMV DNA PCR was 3300 million copies/ml. Mother's serum CMV IgM was (-

ve) and IgG was (+Ve). Chest X-ray of pt. was normal. Abdominal ultrasound reveals Hepato-splenomegaly and echocardiography were normal. Hearing Test shows sensory neural deafness in both ears. Epileptiform activity was observed in the sleep-electro encephalography. Brain CT revealed calcification areas in periventricular regions and lateral vent rides were wide In Brain MRI, monitoring there were pachygyria and lateral ventricles were wider.

Differential Diagnosis

Other viral infection of TORCH should be considered.

Treatment and follow-UP

The patient was diagnosed with congenital CMV infection as a result of the clinical and laboratory examinations, and then given ganciclovir 10 mg/kg/day for 6 months. During the follow-up period, the clinical and laboratory findings of the patient are to be observed to recover via the ganciclovir treatment. The patient's seizures are now under control and she remains an outpatient at the clinic. Myoclonic seizures were controlled by valproic acid (vales) and Neurolept treatment. Developmental speech, physiotherapy and occupational therapy are also to be given routinely.

Discussion

Pregnant women or women who develop an active CMV infection during pregnancy can pass the virus to their babies-who might then experience symptoms. CMV infection may be Fatal. This infection can present as congenital, perinatal or acquired forms. Though mostly asymptomatic, it can lead to fatal complications. CMV spreads from person to person through body fluid such as blood, Saliva, urine, semen, breast milk and by bone marrow transplantation. There is no cure, but there is medicines that can help to treat the symptoms. Pregnant women can be (a) infected by CMV (b) Reactivation of an old infection (3) by the new stain of CMV [6]. In-utero CMV transmission occurs in 0.5% to 22% of all live births. It is less common than perinatal transmission, but it is more severe. Primiparous mothers, primary infection, infection in the first and early second trimesters of pregnancy, and poverty are all significant risk factors [7]. Congenital CMV infection is the most common cause of intrauterine infection in developed countries.1 the most common clinical symptoms are jaundice, hepatosplenomegaly, splenomegaly and petechia. These symptoms are seen in approximately two-thirds of the affected children. They cause severe neurological sequelae in affected cases perceptible risk factors are primary mothers, primary infection, first and early second trimesters infection and poverty [8]. The following signs and symptoms are more common in babies who have congenital CMV and who are sick at birth; Premature birth, low birth weight, Jaundice, Hepatomegaly, Rash in the skin, Macrocephaly, Enlarged spleen, Pneumonia, Seizure [9].

They cause severe neurological sequelae like hearing loss (unilateral or bilateral), intellectual disability, Vision problem due to chorioretinitis, seizures, lack of coordination, motor delay. Prenatal diagnosis can be done by amniocentesis, viral culture and fetal blood sample for IgG and IgM for CMV. For post-natal diagnosis PCR or viral isolation of CMV in urine, saliva sample at least at 3 weeks of age. Careful hygiene is the best prevention against CMV- hand washing with soap for 15-20 sec especially after contact young children or their diapers, saliva or other oral secretions especially at the child care centre [10]. One patient was reported with bilateral blindness due to CMV retinitis [13-17]. During kissing the child avoid contact with tears and saliva. Sharing food and drinking in same utensils to be avoided. Be careful about disposable items, cleaning of toys and countertops.

Conclusion

CMV *Cytomegalovirus* is a common virus. Once infected, our body retains the virus for life. Congenital CMV is a common congenital infection that is under-recognized. The development of a vaccine against this virus is a major public health priority. Until the goal of a CMV vaccine is relished, educating women of childbearing age about the risks of CMV and about how to avoid disease transmission are the only control strategies available. Several ways to be associated with transmission of infection, particularly kissing and sharing, eating with utensils can be avoided and careful hand-washing after diaper changes should be stressed. Antiviral medications are the most common type of treatment. They can slow the reproduction of the virus, but can't eliminate it researchers are studying now medications and vaccines to treat and prevent CMV. Last but not least, despite its health, social and economic burden, congenital CMV infection often goes undetected at birth because the majority of cases are asymptomatic screening programmes both in pregnant women and newborns have not been developing or implemented. So, it remains still.

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