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Case Study

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A CASE OF NEONATAL CYTOMEGALOVIRUS INFECTION

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INTRODUCTION

Congenital infections indicates Infections acquired in utero. Such infections are generally caused by viral or other non-bacterial organisms and are often associated with injury to developing organs. Cytomegalovirus is one of the leading cause of mental retardation and sensorineural hearing loss. Majority of CMV infection are asymptomatic at birth, 10% have congenital infection.[1] Congenital cytomegalovirus (CCMV) infection is the most prevalent and consequential congenital infection, among others, that affects approximately 0.6% of all live births worldwide. About 1 in 5 babies with congenital CMV infection will have birth defects or other long-term health problems. Timing of maternal infection and maternal immune status largely determine the likelihood of a symptomatic infection.

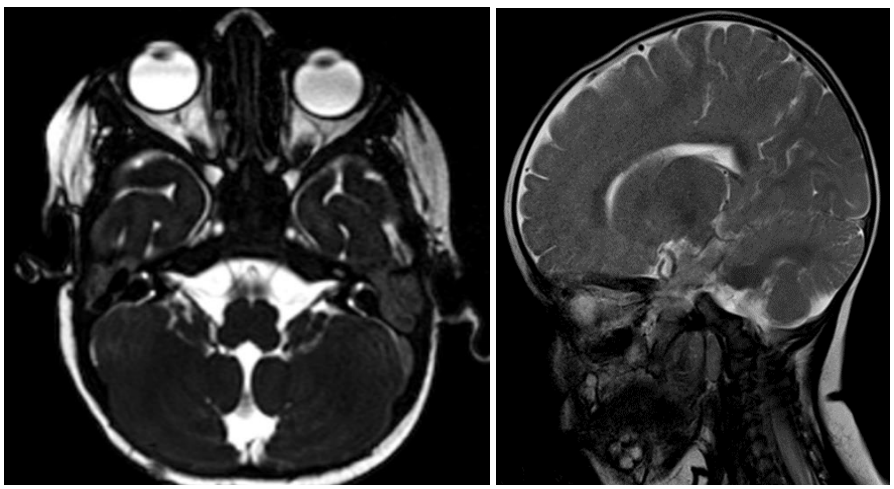
CASE REPORT

9-month-old baby (Term/Low Birth weight -1.9kg /small for gestational age), normal development found to have microcephaly at birth (Head circumference - 30 cm) & was evaluated. No history suggestive maternal fever or rash

Examination: No facial dysmorphism, Weight for age - < -3 SD, Height for age - < -3 SD, Weight for Height - < -3 SD, Head circumference - Microcephaly (40 cm as per age)
Systemic examination
CNS - No focal neurological deficits
No signs of raised intracranial pressure
CVS - S1S2 (+), No murmur
RS - Air entry bilaterally equal
Fundus - No evidence of microphthalmos/ retinitis

Investigations

Complete blood count showed Hemoglobin - 9.8, Total Count- 14110, Liver function test - Normal (Total bilirubin - 0.4mg/dl, Direct bilirubin - 0.2 mg/dl, SGOT - 50U/L, SGPT - 26U/L, Alkaline phosphatase - 204 U/L)
Torch Profile- Blood CMV IgG - (25 IU/ml), Blood CMV IgM – Negative, S.
CMV IgG (9 months)- 59.8 IU/ml
Echo - Normal study, OAE - Bilateral Absent
MRI Brain – Mega cisterna magna and prominence to IV th ventricle. No intracranial calcifications / white matter lesions



DISCUSSION

The clinical spectrum of congenital CMV infection ranges from an infant exhibiting no symptoms to one displaying signs and symptoms of widespread illness. The infant presents with hepatitis, splenomegaly, pneumonitis, jaundice, thrombocytopenia, chorioretinitis, hemolytic anaemia, petechial or purpuric rash, seizures, spasticity, microcephaly, optic atrophy, and neuroimaging evidence of intracerebral calcifications and cerebral atrophy in the most severe form of congenital CMV infection.[2]

Prenatal and postnatal are the two contexts in which a diagnosis can occur. Testing of the mother and foetus is part of prenatal diagnostics.

Currently there are only limited options for therapies and a lack of tests with a high enough sensitivity and specificity to advocate universal screening for CMV during pregnancy. However, testing makes sense in some situations. These conditions include pregnancy-related mononucleosis-like illness, contact with an individual infected with CMV, exposure at work (in healthcare or childcare), or foetal ultrasound findings indicative of congenital CMV infection, such as ventriculomegaly, hyperechogenic bowel, hydrops, and intracranial calcifications. Testing of both mother and the foetus is required in cases where primary CMV infection is suspected.[3]

Estimation of mother's antibody status is a step in the maternal testing process. Amniocentesis is used to obtain foetal diagnosis using PCR on amniotic fluid, either with or without viral culture. Quantitative PCR assays (urine, saliva,blood) used in first 2-3 weeks of life. The determination of serum antibody titres to CMV has limited usefulness for the neonate. Interpretation of a positive IgG titre in the newborn is complicated by the presence of transplacentally derived maternal IgG. Uninfected infants usually show a decline in IgG within 1 month and have no detectable titer by 4 to 12 months, whereas infected infants will continue to produce IgG. Tests for CMV-specific IgM may help in the diagnosis of an infant infection.

No definitive treatment for congenital CMV infection. Neonates without any symptoms also needed to be monitored, especially for regular hearing tests. Oral valganciclovir should be given to newborns with Congenital CMV for a period of six months. This therapy correlates with improved long-term neurodevelopmental results and has been demonstrated to maintain normal hearing and arrest the progression of hearing loss. Valganciclovir taken orally is preferable to Ganciclovir administered intravenously, which is linked to gonadal toxicity and bone marrow suppression.[4] It is essential to closely monitor both symptomatic and asymptomatic neonates for the emergence of long-term consequences.

CONCLUSION

Asymptomatic CMV should be followed up at regular intervals to assess long term neurodevelopmental outcome and hearing loss.

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