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Congenital Cytomegalovirus Infection in Pregnancy

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Abstract

CytoMegaloVirus (CMV) is an enveloped double stranded DNA virus that belongs to Herpesviridae family. Most of the CMV infections during pregnancy are asymptomatic; but cause congenital malformations in fetus. The characteristic features of congenital infection are intrauterine growth retardation, prematurity, hepatomegaly, jaundice, thrombocytopenia, purupura, microcephaly and intracranial calcification. In our case report we aimed to present a newborn with intrauterine growth retardation due to congenital CMV infection and review the literature.

Keywords: Cytomegalovirus; Congenital Infection; Gancyclovir; Intrauterine Growth Retardation

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Introduction

CytoMegaloVirus (CMV) is an enveloped double stranded DNA virus that belongs to Herpesviridae family. The first reproduction of the viral particles takes place 48-72 hours after the infection. The virus does not affect the cellular metabolism or cause rapid cellular death. It affects the host cell by speeding up the RNA, DNA and protein synthesis [1].

Most of the CMV infections during pregnancy are asymptomatic; but cause congenital malformations in fetus [2]. 11% of congenital CMV cases are asymptomatic infection. The characteristic features of congenital infection are intrauterine growth retardation, prematurity, hepatomegaly, jaundice, thrombocytopenia, purupura, microcephaly and intracranial calcification. The diagnosis of CMV infection is carried out by detection of CMV DNA in urine or blood sample [3]. Gancyclovir and valagancyclovir treatment are recommended in symptomatic, congenital cases and in cases with central nervous system symptoms [4].

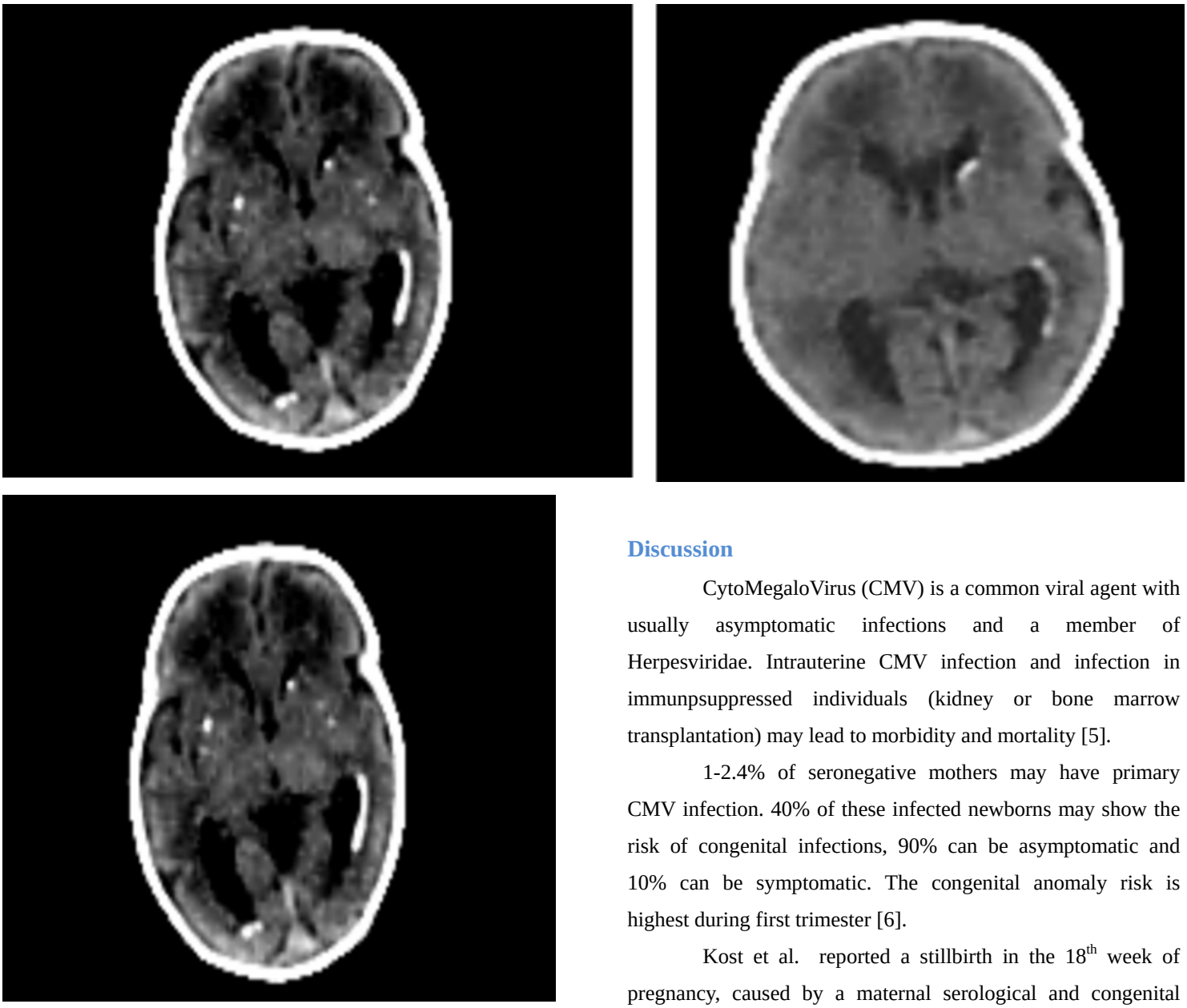
In our case report we aimed to present a newborn with intrauterine growth retardation due to congenital CMV infection and review the literature.

Case Report

18 year old (G1P10) female gave birth to girl baby at 3th week of parity with a weight of 1680 gr. by C/S. The baby was admitted our University Hospital Neonatal Intensive Care Unit with prematurity and intrauterine growth retardation. The patient had petechia, respiratory distress and sub costal restriction. O_2 support was provided. She had no abnormality on lab reports except thrombocytopenia. Respiratory distress disappeared during follow up. The patient was CMV IgM (+) (Architect, Abbott; Germany) and because of this result she was isolated. Urine and plasma samples were sent for CMV DNA (Light cycler, Roche Diagnostics, Germany) and reported as positive. The cranial CT of the patient revealed periventricular

calcification at basal ganglion level was detected (Figure 1). The US image and fundus examination was normal. The patient was diagnosed as congenital CMV infection and initiated gancyclovir treatment. Thrombocytopenia of the patient recovered on the fourteenth day of gancyclovir treatment. Nutrition education was given to the mother and the patient was discharged on the 18th day of gancyclovir treatment with a weight of 1950 gram and she did not require further intensive care unit monitorization.

Figure1: MR image of calcifications at ventricular and basal ganglion level



Discussion

CytoMegaloVirus (CMV) is a common viral agent with usually asymptomatic infections and a member of Herpesviridae. Intrauterine CMV infection and infection in immunpsuppressed individuals (kidney or bone marrow transplantation) may lead to morbidity and mortality [5].

1-2.4% of seronegative mothers may have primary CMV infection. 40% of these infected newborns may show the risk of congenital infections, 90% can be asymptomatic and 10% can be symptomatic. The congenital anomaly risk is highest during first trimester [6].

Kost et al. reported a stillbirth in the 18th week of pregnancy, caused by a maternal serological and congenital CMV infection and benzodiazepine abuse. She was also an alcohol addict. She could not be monitorized until her 18th week of gestation. Ultrasound imaging revealed an intrauterine fetal

death in the 18th week of gestation. The abortion of the pregnancy gave stillbirth to a male fetus. Serologically, maternal IgM and IgG antibodies against CMV were demonstrated, showing a primary or a recurrent CMV infection of the mother [7]. In our case the baby was CMV IgM positive and CMV DNA was also detected in plasma and urine samples.

Walter-Nicolet et al. reported two cases of persistent pulmonary hypertension related with congenital CMV infection. The two infants had severe refractory hypoxemia. And one of them required treatment. Ganciclovir treatment was initiated in the two cases on the 12th day of postnatal life. One of them died and the other baby survived. The authors have concluded that neonatal persistent pulmonary hypertension may be a result of congenital CMV infection. Respiratory support and IVganciclovir are important under these conditions [8]. In our case report the baby also required respiratory support and ganciclovir therapy was initiated.

Lazzarotto et al. reported two pregnant women with twin pregnancies and one woman with a triple pregnancy with primary CMV infection detected by the presence of Immunoglobulin (Ig) M and low IgG avidity and/or by the presence of clinical symptoms and abnormal liver enzyme values.

CMV infection was shown in six fetuses/ newborns, and three were symptomatic. In the first twin pregnancy CMV symptomatic infection of the female twin was demonstrated by positive virus isolation and high viral load in amniotic fluid. The male fetus was not infected and this was detected by negative CMV culture and DNA detection in amniotic fluid. In the triple pregnancy, quantitative PCR reported 10^3 Genome Equivalents (GE)/mL in female's amniotic fluid and 1.9 $*10^5$ GE/mL in male amniotic fluid. Female twins were asymptomatic at birth; but the male had petechiae, thrombocytopenia, and cerebral ventriculomegaly [9]. In our case report the baby had intracranial calcification and a positive CMV DNA both in urine and plasma samples.

Kundu et al. reported a case of congenital CMV infection associated with lissencephaly. A 7 months old male baby admitted in paediatricneurology department due to repeated convulsion since 1 months of age. The ultrasound of cranium indicated cerebral sulci are poorly defined, lateral ventricles are moderately and symmetrically dilated, calcifications are detected around the lateral ventricles and suggested CMV infection. CT image detected moderate generalized cerebral cortical atrophy, ventriculomegaly, periventricular calcification and lissencephaly. Anti CMV antibody (both IgG and IgM) and CMV DNA were positive [10]. Our case was admitted to pediatrics department due to intrauterine growth retardation and prematurity.

The assay for the diagnosis of congenital infection is detection of virus in the amniotic fluid by culture or molecular microbiological methods. Detection of CMV in amniotic fluid by culture or PCR detects the infected fetus. The principal ultrasound findings associated with congenital CMV infection can be placentomegaly, intrauterine growth restriction, microcephaly, ventriculomegaly, periventricular calcifications, isolated serous effusions, and echogenic bowel [11,12].

As a conclusion in case of a congenital CMV infection a detailed serological profile should be tested and multisystemic examination should be carried out and woman at childbearing age should avoid contact with urine or saliva of infected ones and apply proper hand-washing.

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