

Case Report

Disseminated Congenital Cytomegalovirus Infection in Infant

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Abstract: Most cases of congenital cytomegalovirus (CMV) are thought to result from primary maternal infections. Clinical sequelae of CMV infections appear to be more severe if maternal infection occurs during first or second trimester. Here we are reporting a case of congenital cytomegalovirus of a 5 months old male infant. Our patient presented at 5 months with global developmental delay and severe lower respiratory infection with anemia and thrombocytopenia.

Keywords: Congenital cytomegalovirus, global developmental delay, severe lower respiratory infection

INTRODUCTION

Severe cases of congenital cytomegalovirus (CMV) disease have been reported from recurrent and reinfections [1-3]. However, most cases are thought to result from primary maternal infections. The clinical sequelae of congenital CMV infections appear to be more severe if maternal infection occurs during first or second trimester [4, 5]. Primary CMV infections occur in 0.15–2.0% of pregnant women, with 30–40% of mothers vertically transmitting the virus to the fetus. Approximately 10–15% of congenital CMV cases are symptomatic at birth, with up to 30% of these cases being fatal. Of the 85–90% of children born with asymptomatic congenital CMV, up to 15% will develop symptoms in later life, the most common being sensorineural hearing loss [6].

CASE REPORT

A 5 months old male infant presented with convulsions since 1 month of life, cough and breathing difficulty since 4 days, loss of consciousness for 4 hours and on evaluation found to have global developmental delay.

Antenatal history

Mother was not booked or immunized with no iron, folic acid supplements taken. Scandone at 6 months was normal. No history of fever, rash or lymphadenopathy.

Natal history

Full term normal vaginal delivery at hospital. Baby cried immediately after birth, with uneventful transition. Birth weight was 3kg.

On examination

Baby is in altered sensorium responding to painful stimuli, Heart rate: 112 b/m, Respiratory rate: 62 b/min with inter costal retractions, CFT <3sec, Peripheral pulses well felt SpO₂: 76% in room air, 94% with 4litres of oxygen.

Cyanosis present, No pallor, icterus, clubbing, lymphadenopathy or edema

Anthropometry

		Expected	Centiles
Weight	4.16kg	7.5kg	<3rd centile
Height	61cm	65cm	<3rd centile
Head circumference	35cm	43cm	<3rd centile.

Child had microcephaly with no other dysmorphic features

Systemic examination

CNS: GCS 5, bilateral increase in tone with exaggerated Reflexes in all four limbs, responds to painful stimuli.

Respiratory system : features of respiratory distress seen with b/l fine crepitations present.

CVS: S1 S2 heard, no murmurs,

Per abdomen : moderate firm hepatosplenomegaly present.

Investigations

- HB-8.8, TLC -12,200, P/L/E -46/53/01, Platelet count-46000

- Serum urea-49, Serum Creatinin-0.4, Serum uric acid-5.7, Serum electrolytes – WNL.
- Total bilirubin- 0.6, Direct bilirubin-0.3, Indirect bilirubin -0.3 .SGOT -124 ,SGPT 56, ALP 98, Total proteins-4.0, Albumin-2.1, Serum Cal -9.5, PT- 15.1, APTT- 40.0, INR -1.4,
- Urine RE/ME- NA,
- Blood c/s - no growth. Fundoscopy-no papilledema
- USG Abdomen -cholelithiasis,mild hepatosplenomegaly with ascites
- CT scan of brain: suggested of periventricular calcification with ventriculomegaly, communicating hydrocephalus.
- IgM&Ig G for CMV- POSITIVE

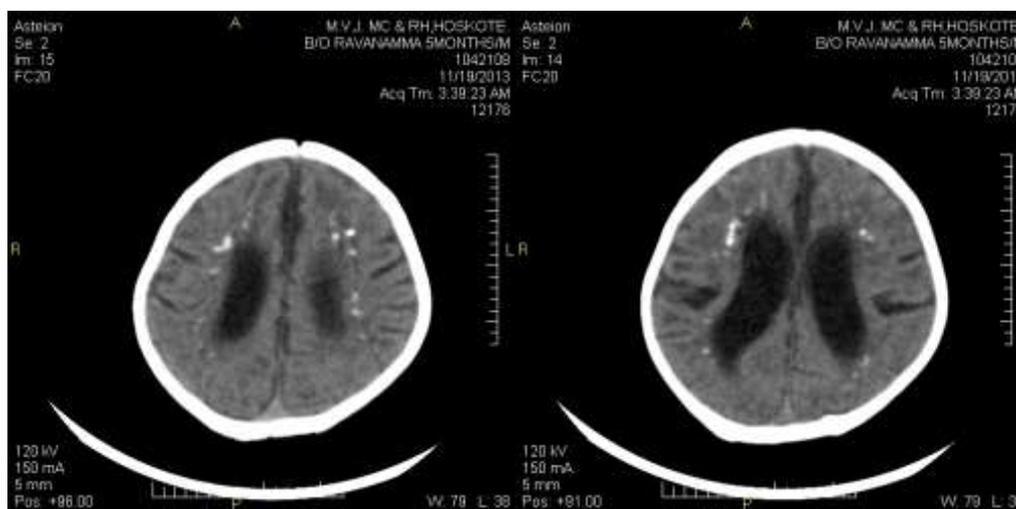


Fig. 1: Showing multiple ill-defined calcific lesions in bilateral periventricular regions with mild dilatation of bilateral ventricles

DISCUSSION

Symptomatic congenital CMV infection may present with a spectrum of clinical sequelae, which can affect multiple sites and have significant morbidity and mortality. The most common symptoms reported are IUGR at birth with jaundice, thrombocytopenia, hepatomegaly, petechiae, purpura and splenomegaly [7]. Infants may have a permanent effect on the outcome of the child, such as delayed mental development, deafness, seizures, cerebral palsy and blindness [8]. Also cytomegalovirus infections result in periventricular and subependymal calcifications.

The commonly reported presentations at birth of congenital cytomegalovirus infection hepatosplenomegaly and rash-were not seen in this case. Hepatosplenomegaly and pneumonitis noted for the first time at the age of 5 months, a finding also noted by Starr *et al.* [9]. It is possible that cytomegalovirus acts as an opportunist respiratory pathogen like chlamydia or pneumocystis carinii since the age of presentation coincides with the waning of passive maternal immunity and cytomegalovirus may depress cellular immunity [10].

CONCLUSION

Our patient presented at 5 months with global developmental delay and severe lower respiratory infection with anemia and thrombocytopenia. Interestingly, multiple calcifications in bilateral periventricular regions was also seen in our patient.

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