



Case Report

Acute Fulminant Type of Cytomegalovirus

Authors

Swathi Eluri¹, Tanvi Pai², Murali Keshava³

¹Junior Resident, ²Senior Resident, ³Assistant Professor

Department of Pediatrics, KMC Mangalore, Manipal University Pin 575001

Mobile-9481550665, Email: dr.swathieluri@gmail.com

Abstract

Acute fulminant type of Cytomegalovirus (CMV) is a rare entity with only 8-10% prevalence rate in symptomatic neonates. Late preterm neonate delivered to primigravida mother presented with symptoms of fulminant type of CMV. Diagnosed by CMV antigen positivity in urine by Polymerase chain reaction (PCR). Neonate survived and was discharged at 24 days of life.

Keywords: Rare entity, Polymerase Chain Reaction.

Congenital Cytomegalovirus (CMV) infection occurs worldwide. In the developed countries prevalence rate is 0.6%¹. Annually in the United States around 40,000 infants are born with CMV². Among them 10% are symptomatic at birth.

We herein report a female baby born to a primigravida mother by Emergency Cesarean Section conducted in view of non reassuring fetal heart sounds, subsequently diagnosed as having acute fulminant type of CMV.

Case Report

A late preterm, low birth weight (1.80kg), female neonate born out of non-consanguineous parentage by cesarean section was admitted to our Neonatal Care Unit with respiratory distress and required mechanical ventilation. Antenatal Ultrasound scans of the mother showed mild cardiomegaly with minimal pericardial effusion with prominent right ventricle. On admission the

baby had microcephaly with the head circumference of 30cm, multiple petechiae, blueberry muffin spots, icterus, and hepatosplenomegaly with the liver span of 10cm and the spleen palpable 2cm below the left costal margin (figure 1). Supportive therapy was continued and appropriate investigations were sent. Investigations were suggestive of hemolytic anaemia (Hb 8.2gm/dl, PCV 28%), thrombocytopenia (platelet count 16,000), altered hepatic enzymes (SGOT 228, SGPT 280, ALP 670), raised total bilirubin (6mg/dl), Prothrombin time and INR was normal. As there was no coagulopathy disseminated intravascular coagulation due to sepsis was ruled out. Maternal blood investigations were normal ruling out autoimmune thrombocytopenia. X-ray of upper limb was normal hence there were no features suggestive of thrombocytopenia-absent radius (TAR) syndrome and megakaryocytic thrombocytopenia associated with radioulnar

synostosis (ATRUS or Fanconi anemia). X-ray chest was suggestive of pneumonitis (figure2). Neurosonogram showed multiple periventricular cysts with dilated lateral ventricles. In suspicion of TORCH infections IgM and IgG TORCH pannel was sent which was positive for CMV. Subsequently Urine and Blood for CMV by PCR were positive. Mother's IgG TORCH panel was positive for CMV. In view of financial constraints baby could not be started on Ganciclovir or Valganciclovir and only supportive treatment was continued. Gradually baby improved with supportive treatment, was weaned off from ventilator and feeds were established. Was discharged at 24 days of life.



Figure 1

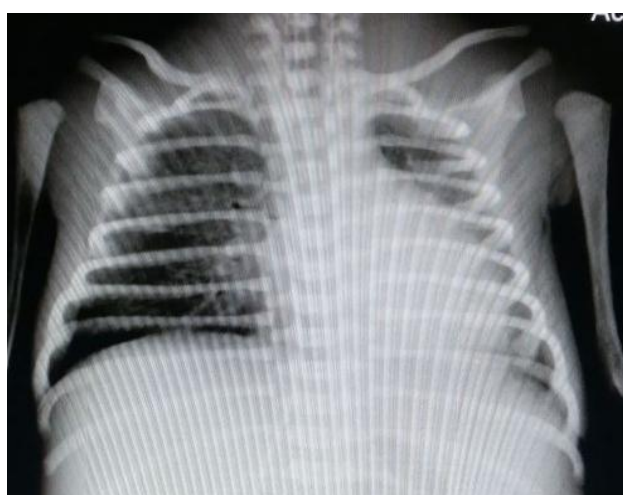


Figure 2

Discussion

CMV is one of the vertically transmitted infections. Congenital early symptomatic disease can present as an acute fulminant infection involving multiple organ systems with as high as

30% mortality³. Premature infants and infants with primary immune disorders of natural killer cells or T cells are at a greater risk of mortality. Mortality is due to viral associated hemophagocytic syndrome or severe end organ disease of the liver, lungs, bone marrow or cranial nervous system. Signs include petechiae or purpura, hepatosplenomegaly, jaundice, prematurity and blueberry muffin spots reflecting extra medullary hematopoiesis. Laboratory abnormalities include elevated hepatic transaminases and bilirubin levels, anaemia, and thrombocytopenia. One-third of these infants are preterm and have intrauterine growth restriction. Diagnosis is suspected in the following conditions

Newborns with symptomatic congenital CMV	Newborns with cranial imaging consistent with CMV like Periventricular calcifications/leukomalacia	Abnormal fetal ultrasound
Newborns with abnormal hearing screen	Ventriculomegaly Polymicrogyria	Echogenic bowel
		Intrauterine growth restriction
		Microcephaly or abnormal fetal brain imaging
		Hepatosplenomegaly
		Fetal hydrops or ascites

Urine for CMV PCR is highly sensitive. Other diagnostic techniques include spin enhanced culture or shell vial, detection of CMV antigen using specific antibody.

Negative IgG titers in maternal and infant sera exclude CMV. IgM CMV has limited specificity. Can be treated with Intravenous Ganciclovir 6mg/kg/dose every 12th hourly for 2-6weeks followed by Oral Valganciclovir 16mg/kg/dose twice a day for 6-12 months depending upon the response. Treatment response is assessed by clinical evaluation and measurement of viremia levels⁴.

References

1. Goderis J, De Leenheer E, Smets K et al. Hearingloss and congenital CMV infection: a systematic review. *Pediatrics*. 2014 Nov;134 (5):972-82.
2. Harrison GJ. Cytomegalovirus. In: Feigin and Cherry's Textbook of pediatric infectious diseases, 7th ed. Cherry JD, Harrison GJ, Kaplan SL, et al (Eds),

Elsevier Saunders, Philadelphia 2014.p.
1969

3. Lopez AS, Ortega Sanchez IR, Bialek SR. Congenital cytomegalovirus related hospitalizations in infants <1 year of age, United States, 1997-2009. *Pediatr Infect Dis J.* 2014 Nov;33 (11):1119-23.
4. Amir J, Wolf DG, Levy I. Treatment of symptomatic congenital cytomegalovirus infection within travenous ganciclovir followed by long-term oral valganciclovir. *Eur J Pediatr.* 2010 Sep;169(9):1061-7.