

**CONGENITAL CYTOMEGALOVIRUS INFECTION -
AN UNUSUAL PRESENTATION - A CASE REPORT**

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Introduction :

Cytomegalovirus (CMV) is a herpes viral genus of the Herpes Virus group¹. In human it is commonly known as human CMV or human herpes virus 5 (HIV-5)¹. People are usually infected during their teenage years². Most of the common causes of congenital infections, which occasionally causes the syndrome of cytomegalic inclusion disease (Hepato-splenomegaly, Jaundice, Petechiae, Purpura and microcephaly) in neonates³. Disease may result from primary or recurrent CMV infection. Primary CMV infection is commonly associated with severe disease, resulting hepatitis and mononucleosis³. Mild transient increases in hepatocellular enzymes and rarely jaundice. The disease typically has a favorable prognosis, but death had been reported in immunosuppressed patients. Typically mononuclear cell infiltration occurs in portal areas but also reveals granulomatous inflammation⁴. CMV can cause meningitis and lead to serious long term consequences⁵.

Case:

A two (2) months old male child weighing 4.5 kg 1st ssac of a consanguineous parents of middle class family admitted with the complaints of fever for 2 days which was initially low grade then became high grade along with convulsion involving right side of the body without any residual weakness or frothy discharge. Bulging of anterior fontanelle for same duration. No history of pneumonia, head injury or travelling to malaria endemic zone. Mother had a history of rash at 4 month of gestation. On

examination, the child was ill looking, fontanelle was bulged, OFC -38cm, HR -130/min, temperature 103.0 F, moderately pale, non icteric, non cyanosed, no lymphadenopathy. Neck rigidity and hepatosplenomegaly were present. Provisional diagnosis was meningitis & differential diagnosis were encephalitis & cerebral malaria. We started treatment by keeping nothing per oral, infusion, inj. Ceftriaxone, inj. Phenobarbitone, inj. Diazepam, O2 inhalation, N-G & O-P suction were given. On admission convulsion occurred about 4 times which was not controlled by inj. Phenobarbitone, & inj. Diazepam, subsequently inj. Phenytonin was added. Investigation showed hemoglobin 7.17 gm / dl, total leucocyte count - 16000/cmm, with a differential count of neutrophil -34%, lymphocyte- 61%, monocyte -3%, eosinophil -2%. ESR -22 mm in 1st hour, blood group B (+ve). CSF report was - cell count -10 cmm, neutrophil 20%, lymphocyte -80%, protein -50mg/dl, sugar -84 mg/dl, pandy test +ve, bacteria not found. On day 5th, per rectal bleeding started and subsequently, blood transfusion, inj. ranitidine, inj. konakion, inj. metronidazole were given. On 6th day investigations' showed hemoglobin 11.1 g / dl platelet -3,20,000 / cmm, PT -24 sec, activated partial thromboplastin time 48 sec, fibrin degradation product-5. On 7th day, patient developed weakness of right limb and OFC was increase, USG of brain showed right ventricle were compressed and midline shifting towards right side and inj. Mannitol was added. On 10th day, patient developed jaundice, S. bilirubin 6.20 mg / dl. SGPT - 580 U / L, Alkaline phosphatase 1278 U/L, USG of Hepatobiliary system was normal. On 13th day, TORCH screening & liver function tests were done. On 14th day S. bilirubin -21.5 mg/dl direct-20.5 mg/dl, indirect 1mg/dl, SGPT -39 U/ L. Alkaline phosphatase -980 U/L, on day 17th, TORCH report came, CMV test positive, IgG-121, IgM->100, Rubella-IgG positive. On 18th day, USG of brain showed communication hydrocephalus and subdural effusion possibly sequelae of meningitis and we consulted with neurosurgery department. Our confirmed diagnosis was CMV hepatitis with meningitis with hydrocephalus with subdural diffusion and we started inj. Gancyclovir with proper dose and schedule and patient ultimately died on 23rd day.

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Discussion :

Human CMV infection is more widespread in developing countries and in communities with lower socio-economic status and represents the most significant viral cause of birth defects in industrialized countries⁶. In our study the

patient came from low economic background. Transmission sources of CMV include saliva, breast milk, cervical and vaginal secretion, urine, semen, stools, blood and tissue or organ transplants. Prenatal transmission is common accounting for an incidence of 10-60% through first six months of life. The most important sources of virus are genital tract secretions during delivery and breast milk. Among CMV seropositive mothers, virus is detectable in breast milk in 96% with postnatal transmission occurring in approximately 38% of infants resulting in symptomatic infection in nearly half of very low birth weight babies. Infected infants excrete virus for years in saliva and urine.³ However, CMV can cause problems in people who have weak immune systems and in a newborn if the mother gets the infection during pregnancy.⁷ In our case, mother had history of rash during pregnancy. Most healthy people who are infected by HCMV after birth have no symptoms.¹ Some develop as infection mononucleosis/glandular like fever having prolonged fever and a mild hepatitis.⁸ Symptomatic CMV infection of fetus has two presentations. Early manifestation includes petechiae or (63%), blueberry muffin spots consistent with extramedullary haematopoiesis. Laboratory findings include increased hepatic transaminase and bilirubin level (as much as half is direct or conjugated). Anemia and thrombocytopenia were also present. A second early presentation includes those infants who are symptomatic but without the life threatening conditions. These babies may have IUGR or disproportionate microcephaly 48% with or without intracranial calcification.⁹ Studies have shown that asymptomatic children with neurological findings are more likely to have CMV infection.¹⁰ Traditionally, CMV antibody tests were performed to diagnose. Antibody titer peak 4-7 weeks after infection.¹¹ In our study, patient had fever, convulsion, and jaundice, on examination, anemia, jaundice, hepato-splenomegaly were present. On investigation, Hb was low, SGPT was high, bilirubin (direct) was increased, CMV -IgG and IgM were positive. Treatment is not indicated for immunocompetent person, but is recommended for immunocompromised person, recommendation for treatment remains controversial for infants with symptomatic congenital infection. CMV-IgG immunoglobulin containing a standardized amount of

antibody to CMV, it may be used for the prophylaxis of CMV disease during transplantation of kidney, lung, liver, pancreas and heart alone or in combination with an antiviral agent to reduce CMV related disease and death.³ The drug of choice for treatment of CMV is ganciclovir.¹²

Conclusion :

CMV infection is increasing and has become a great challenge. Antenatal early detection prevents such type of infected baby.

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