Infectious dynamics of cytomegalovirus: A case of transient increase of middle cerebral arterial peak systolic velocity

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Constitutional cytomegalovirus (CMV) infection is the leading cause of nonhereditary sensorineural hearing loss and other long-term neuro-developmental impairment, including cerebral palsy, intellectual disability, vision impairment, and seizures.

Transplacental infection is clinically important because prenatal CMV infection causes neurological dysfunction, though postnatal acquired CMV infection in immunocompetent patient is generally subclinical. CMV is transmitted from mother to fetus in approximately 30-39% of pregnancies in which a seronegative mother becomes infected during pregnancy. Some prospective studies demonstrated that fetal neurological damage could also occur following non-primary infection. The prevalence of congenital CMV infection is reported to be 0.4-2.3% of all live births.

The nervous system is one of the main targets of congenital CMV infection, but the infectious dynamics of CMV in vivo brain has not been sufficiently studied.

We report a case of congenital CMV infection, in which middle cerebral arterial peak systolic velocity (MCA-PSV) was transiently increased in uterus without fetal anemia.

**Case: 28-year-old woman (gravida2 / para1), Blood group of A Rh(D) positive**

**Introduction**

- Past medical history
  - Unremarkable

- History of present illness
  - She referred to our hospital for pregnancy. The estimated fetal weight (EFW) at 24\(\frac{5}{7}\) weeks of gestation was 705 g (-0.29 SD) and there were no abnormal findings on ultrasound; MCA-PSV measured 34.9 cm/sec. At 26\(\frac{5}{7}\) weeks of gestation, fetal MCA-PSV elevated to 77.7 cm/sec without any other abnormalities or fetal distress, and it returned to normal range at 28\(\frac{2}{7}\) weeks of gestation. Cerebral ventriculomegaly was confirmed at 30\(\frac{3}{7}\) weeks of gestation. At 34\(\frac{3}{7}\) weeks of gestation, MCA-PSV increased to 75.4 cm/sec again and it was persistent. There were no maternal infectious episodes and signs of fetal distress through the pregnancy.

- At 39\(\frac{0}{7}\) weeks of gestation, she delivered a male infant weighed 2,590 g with Apgar score 8 at 1 minute and 9 at 5 minutes.

**Baby’s labs: Day1**

- WBC 6.8 \(\times\) 10\(^3\) /μL
- AST 635 IU/L
- BUN 9 mg/dL
- Hb 17.3 g/dL
- Pt 20.6 μ/mL

**Automated auditory brainstem response**

- Refer: sensorineural hearing loss

**Urine test: real-time PCR**

- CMV DNA (+)

**Discussion**

The mechanisms of MCA-PSV increase are generally considered to be follows: fetal anemia, hypoxia, and inflammation. When MCA-PSV becomes abnormal, in general, it will not be normalized without interventions, and there are no cases whose MCA-PSV transiently increased in previous reports. The transient increase of MCA-PSV in our case may have been affected by the biological properties of CMV.

CMV is a member of the herpes virus family, and it has latency properties as other herpes viruses. The infected neural precursor cells disturb neuronal migration and reduce the number of neuronal cells, which cause the morphological and functional brain disorders.

It is reported that the MCA-PSV correlates with the inflammatory response. MCA-PSV increases before the appearance of clinical features because of vasodilation and increased cerebral blood flow in early hours of infection. The first transient MCA-PSV increase in our case was confirmed at 26\(\frac{5}{7}\) weeks of gestation; the values of MCA-PSV at 24\(\frac{5}{7}\) weeks and 28\(\frac{2}{7}\) weeks of gestation were in normal range.

**Conclusion**

The transient increase of MCA-PSV in our case may have been affected by the biological properties of CMV which has latency properties.