A rare case of cytomegalovirus parotitis and possible encephalitis in an immunocompetent child

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Introduction

Cytomegalovirus (CMV) is a human herpesvirus that is known to cause severe infections in immunocompromised hosts (1). It is a well-recognized cause for intrauterine infections that can result in severe multi-organ involvement causing microcephaly, cerebral calcifications, hepato-splenomegaly, hepatitis, chorio-retinitis, hearing loss and seizures in affected newborn (2). CMV is one of the leading causes for non-genetic sensory-neural hearing loss and neurological squeal (3). In immune-competent hosts, CMV commonly manifests as a mononucleosis-like illness with fever, fatigue and cervical lymphadenitis or even can be asymptomatic (1). However, severe CMV infections and life threatening complications are reported in healthy individuals. In a systematic review, Rafailidis et al., described 290 immunocompetent patients with severe CMV infection (4). Among them, colitis and central nervous system infections (meningitis, encephalitis, and transverse myelitis) were found to be the commonest presentations. In addition, some individuals presented with haemolytic anaemia, thrombocytopenia, venous and arterial thrombosis, uveitis, and pneumonitis (4). We report a ten-month-old boy with symptomatic CMV parotitis and possible encephalitis.

Case presentation

A ten-month-old baby boy presented with a history of fever for two days and right-sided parotid swelling. He was a developmentally normal child with no previous history of recurrent infections or immunodeficiency. Furthermore, there was no contact history of parotitis and he was immunized according to the expanded programme on immunization (EPI) schedule including measles, mumps, and rubella vaccine at 9 months of age. Examination revealed the child has grown adequately for his age. He was irritable during the presentation and had a bulging anterior fontanelle and significant right side parotid gland enlargement with cervical lymphadenopathy. The oral mucosa was erythematosus and dry. The system examination was normal. The ophthalmological examination was normal and there was no other evidence of life-threatening or sight-threatening disseminated CMV infection.

Ultrasound examination of the right parotid gland showed significant parotitis. The blood counts revealed a lymphocytosis and the blood picture was compatible with a viral infection. The C-reactive protein level was less than 5 mg/dL. Lumbar puncture was done and the obtained cerebrospinal fluid (CSF) was positive with eight polymorphs and 155 lymphocytes. CSF sugar and proteins levels were within normal range. CSF samples were sent for multiplex real-time PCR and it was negative for HSV 1 and 2, Varicella Zoster virus, Mumps virus, and enterovirus infections.

Viral studies on blood and buccal aspirations were positive with a significant CMV viral load. (Buccal aspiration CMV DNA viral load 1560,000 IU/ml and Blood CMV DNA viral load 300 IU/ml).
Case report

The source of infection was not clear. However, an asymptomatic infection of a close person is the most possible explanation. Congenital infection was highly unlikely as the presentation is well beyond the three weeks of life. Furthermore, the boy had not had any common features of congenital infection such as rash, hepato-splenomegaly, hepatic transaminitis, cataract/chorio-retinitis, microcephaly or prolonged neonatal jaundice. Therefore, we have not done the maternal TORCH screening.

The child was initially treated with intravenous cefotaxime and omitted after 10 days of treatment. With the clinical improvement, the child was discharged after 10 days of admission.

Discussion

Cytomegalovirus demonstrates a wide disease spectrum in both immune-deficient and immune-competent individuals. In immune-deficient individuals, e.g. HIV-infected or organ transplant recipients, it frequently causes life-threatening disease with multi-organ involvement. Multi organ involvement is rare in immune competent patients. However, multiorgan involvement is associated with very high mortality. The disease involving only liver or lung is fatal compared to central nervous system disease (5).

CMV is also recognized as one of the most common causes of congenital infections in the developed world (6). Furthermore, the new research evidence suggests cytomegalovirus infection could have late consequences such as the increased risk of malignancy and vasculitis in immune-competent individuals (7). Therefore, more scientific studies have been focused on CMV infections. However, the mechanism of CMV infection is unclear and under-defined partly due to its frequent asymptomatic nature in immune-competent hosts (1).

Children with congenital CMV infection and immune compromised patients with CMV infection need specific anti-viral treatment such as intravenous ganciclovir, foscarnet, oral valganciclovir. Ganciclovir and foscarnet are known to cause myelosuppression, renal toxicity and teratogenicity. As most of the CMV infections in immunocompetent patients are self-resolving, the evidence for the use of these drugs in immunocompetent patients is lacking. Tirumala et al reported several cases of CMV infection mimicking dengue fever in immunocompetent patients, successfully treated with intravenous ganciclovir (8). Nangle et al., reported 3 cases of immunocompetent adults with severe CMV infection successfully treated with valganciclovir. They have also highlighted the importance of further randomized control trials in this matter (9). However, new pharmacological and cellular therapies for CMV management are in their final stages of development, which may provide a safer option in the future (10).

Usually, encephalitis in children presents with headache and behavioral changes, associated with visual and auditory hallucinations. In our patient, only irritability and bulging anterior fontanelle was present. This could raise a suspicion about the diagnosis of CMV encephalitis. However, that might be due to the inability of eliciting all the clinical features reliably in a 10-month-old child.

We were unable to test cerebrospinal fluid for CMV. We could have done CMV DNA in CSF during the acute phase of illness to confirm or exclude the diagnosis of CMV encephalitis and anti-CMV serum antibodies in about 6 months to reiterate the diagnosis of CMV infection. Belo et al., 2012 also has reported possible CMV encephalitis in an immuno-competent child which has resolved spontaneously without any specific anti-viral therapy (11).

Conclusions

Postnatal CMV infection is usually asymptomatic and mild. However, severe postnatal infections are well documented in immune competent hosts. The hepatic involvement, pulmonary involvement or multi system disease could be highly fatal. These manifestations require treatment with anti-viral drugs.

References


