Congenital Cytomegalovirus Presenting with Macrocephaly and Intracranial Abscess: A Case Report

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ABSTRACT

Congenital cytomegalovirus (cCMV) infection is a leading cause of neurologic disability in infants, often presenting with microcephaly, seizures, and sensorineural hearing loss. However, atypical manifestations such as macrocephaly and intracranial abscess are extremely rare.

We report a case of an infant presenting with progressive macrocephaly, with intracranial abscess and obstructive hydrocephalus. She had a history of prematurity and neonatal sepsis. Neuroimaging revealed a rim-enhancing lesion at the quadrigeminal cistern and aqueductal stenosis. Cerebrospinal fluid (CSF) analysis from ventricular tap showed signs of ventriculitis, and both serum and CSF tested positive for CMV IgG and IgM, associated with a high viral load. Antiviral and antibiotic therapy led to resolution of the abscess and ventriculitis. A ventriculoperitoneal shunt was subsequently placed.

This case illustrates a rare neurological complication of cCMV presenting with an intracranial abscess and hydrocephalus in an immunocompetent infant. It highlights the wide spectrum of cCMV manifestations and the challenges of diagnosis in settings without routine CMV screening. Early recognition and timely intervention are critical in improving outcomes, especially in low- and middle-income countries where systematic CMV testing is lacking.

Keywords: congenital cytomegalovirus, abscess, ventriculitis

INTRODUCTION

Congenital human cytomegalovirus (CMV) infection is the leading congenital infection worldwide, occurring in about 0.48% of live births in high-income countries and up to 1.42% in low- and middle-income countries.[1] Congenital CMV (cCMV) infection can cause grave consequences, including death as well as permanent complications such as neurocognitive sequelae and hearing loss.[1,2]

Studies have shown that while CCMV is commonly reported in high-income countries, its prevalence and severity are notably higher in low-income countries.[1,3] However, reliable data on prevalence and outcomes from developing countries remain limited, which may be due to the lack of systematic reporting and available testing in low- and middle-income countries.[1,3] In the Philippines, there have been 160 cases of congenital CMV in the past ten years; however, this may be underreported.[4]

Distinguishing between congenital and early postnatal CMV infections is critical and challenging. Congenital

eISSN 2094-9278 (Online)
Published: August 31, 2026
https://doi.org/10.47895/amp.v60i16.13921

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CMV infections are clinically silent in approximately 90% of affected newborns, making early suspicion uncommon and delayed until later in infancy.[5] Appropriate testing at birth and after three weeks is necessary; however, there is no systematic screening implemented in pregnancy or in the postnatal period, hence diagnosis is more difficult.[1,5,6]

Neurological manifestations of congenital CMV infection commonly include microcephaly, seizures, and developmental delay.[7] In this study, we present the first case of an infant presenting with macrocephaly and intracranial abscess with ventriculitis, associated with CMV infection. This report aims to highlight the potential for CMV infection to cause atypical neurological complications, emphasizing the importance of early detection for improved outcomes.

CASE PRESENTATION

A four-month-old born to a then 26-year-old Gravida 1 Para 1 (G2P1) was admitted at the neurosurgery department for increasing head circumference. The patient was born preterm at 34 weeks of gestation (AOG) via spontaneous vaginal delivery at a local hospital where she was subsequently admitted due to prematurity and neonatal sepsis. The patient's mother had a history of urinary tract infection during the third trimester leading to emergency premature delivery; she did not have other illnesses or comorbidities during and prior to the pregnancy and denied exposure to chemicals and viral exanthematous diseases. On testing, she was negative for Human Immunodeficiency Virus, syphilis, and hepatitis B virus. Regular prenatal check-ups were done at a lying-in clinic, and the mother had one prenatal ultrasound at 26 weeks AOG, which showed normohydramnios and unremarkable results. The patient was treated with unrecalled antibiotics for two weeks and was discharged well. At birth, her head circumference was appropriate for gestational age (HC = 30.5cm, 50th percentile). No CSF analysis and cranial imaging were done. Her birth weight was two kilograms, which was also appropriate for gestational age. Newborn screening was normal.

In the interim, the patient was otherwise healthy with normal development. She received complete immunizations for age. The mother observed increasing head circumference at 2 months old but with no associated decrease in activity, loss of appetite, fever, or seizures. They sought consultation at a private clinic where they were advised referral to a pediatric neurologist and requested a cranial ultrasound, which revealed communicating hydrocephalus. Two weeks later, there was progression of the patient's head circumference enlargement, associated with sunsetting eyes, prompting consultation with a neurosurgeon. Cranial magnetic resonance imaging (MRI) was done, which showed an extra-axial, well-defined, rim-enhancing lesion in the right aspect of the quadrigeminal cistern and obstructive hydrocephalus with periventricular edema.

On admission, the patient was noted to have increased head circumference (HC = 51.5 cm, Z-score >3), soft bulging anterior fontanelle, and sunsetting eyes. She had upward gaze paresis, with full horizontal extraocular muscle movements on observation, and clear funduscopic findings. The rest of the systemic physical and neurological examination was unremarkable. She was developmentally at par with age.

Cranial MRI was performed on admission, which showed a well-defined, rim-enhancing lesion with T1W hypo-/T2W hyperintense signals and restricted diffusion in the right aspect of the quadrigeminal cistern, measuring approximately 1.4 x 0.9 x 1.6 cm (CCxWxAP) (Figure 1). There is marked dilatation of the third and both lateral ventricles with associated effacement of the bilateral cortical sulci and Sylvian fissures. There is also suspicious funneling of the aqueduct of Sylvius superiorly; hence, aqueductal stenosis is considered. The fourth ventricle is normal in size. Workup for infectious etiology was done, including a serum TORCH panel revealing positive results for CMV IgG and IgM with high CMV viral load. Furthermore, the mother also tested positive for serum CMV IgG. Maternal testing for Human Immunodeficiency Virus (HIV) yielded negative results. Other workups tested negative, including tuberculosis and toxoplasmosis. Auditory Brainstem Response and Multiple Auditory Steady State Response tests were also done, which showed results consistent with moderate to severe sensorineural hearing loss in the patient's left ear.

Cerebrospinal fluid (CSF) studies were assessed throughout the course of the patient. Initially, there was leukocytosis with neutrophilic predominance, associated with low glucose and high protein, consistent with ventriculitis. However, there was no bacterial growth in the CSF. An Ommaya reservoir was inserted, and antibiotics were started. The patient was first given ceftriaxone, but it was eventually shifted to meropenem to cover for bacterial meningitis. Her CSF also tested positive for CMV IgG and IgM; hence, she was started on valganciclovir. CSF cultures tested negative for bacterial growth, tuberculosis, and cryptococcus. After eight weeks of antibiotics and antiviral therapy, there was resolution of the ventriculitis and decrease in the size of the previously seen abscess (Figure 2). A ventriculoperitoneal (VP) shunt was subsequently placed when the ventriculitis resolved and antiviral therapy was continued for six months. After treatment at ten months old, the head circumference was still high for age (HC = 53 cm, z-score >3) but the anterior fontanelle was soft and non-bulging. She had an inconsistent response, spastic extremities, and global developmental delay (social and motor).[8]

DISCUSSION

Congenital cytomegalovirus (cCMV) is a leading cause of neurologic disability in infants, especially in symptomatic cases.[7] Although cCMV most often presents with microcephaly and other neurodevelopmental sequelae,

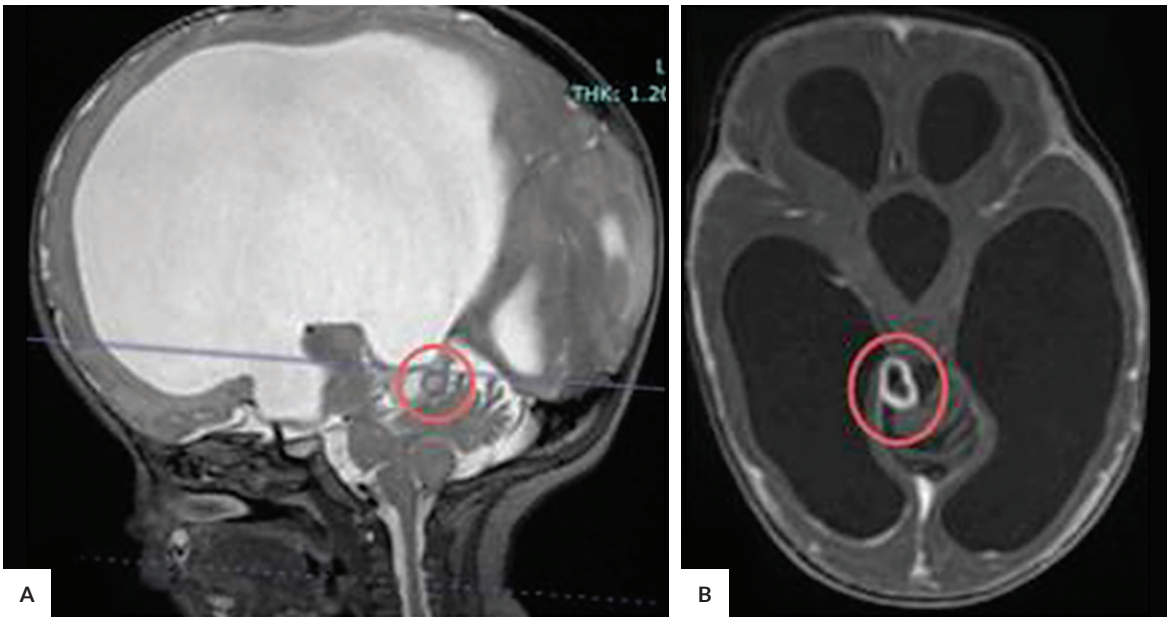

Figure 1. (A) Sagittal T2 image showing a well-defined, hyper-intense lesion surrounded by a hypo-intense rim *(encircled in red)* seen in the right aspect of the quadrigeminal cistern. **(B)** Axial contrast T1 image showing thick peripheral rim enhancement of the said supracerebellar lesion (*encircled in red).*

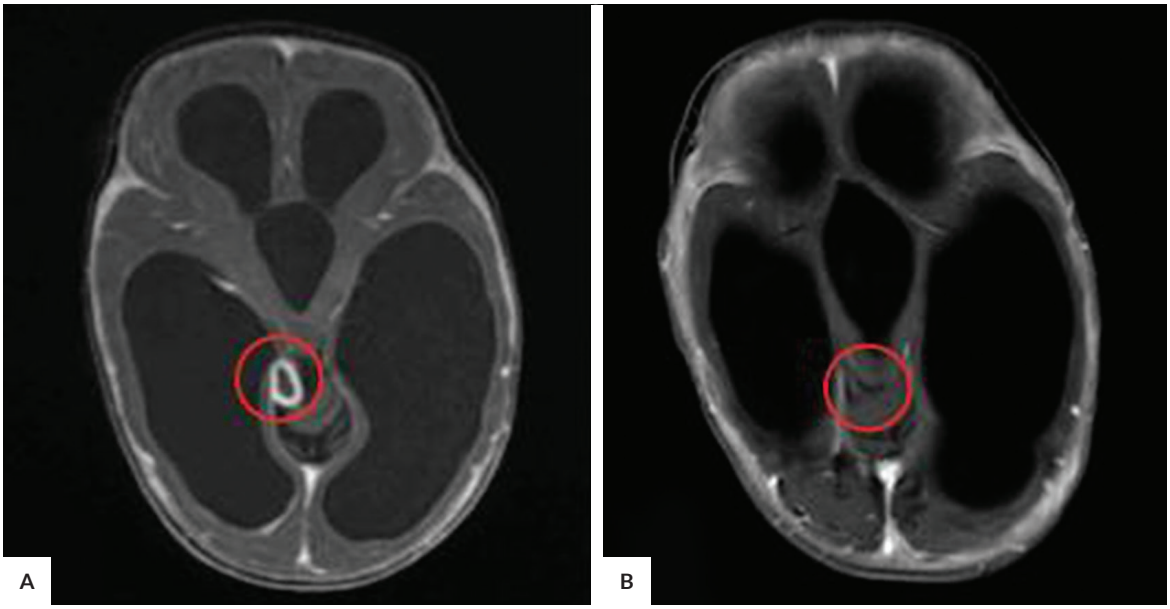

Figure 2. Comparison of brain scans before **(A)** and after **(B)** eight weeks of antibiotic and antiviral therapy, with the lesion encircled in red.

this case demonstrates that macrocephaly with concurrent bacterial abscess may also occur. These atypical features expand the known spectrum of cCMV and can delay diagnosis, emphasizing the importance of considering cCMV in macrocephalic infants with unusual intracranial findings.

There are limited reports on cases of brain mass lesions caused by CMV, most of which were seen in immunocompromised adult patients and were diagnosed through brain biopsy and cerebrospinal fluid (CSF) Polymerase Chain Reaction (PCR) testing.[9] Nevertheless, CSF antigen test for CMV can also detect CMV proteins within the CSF, indicating an active infection of the central nervous system.[10] A positive result of a CSF test for CMV in a patient with an intracranial mass lesion should raise suspicion that CMV may be its main cause.[9] In all the previously reported cases, including ours, the mass lesions

were ring-enhancing lesions. Studies suggest that CMV should be considered a differential diagnosis when a slowly progressive ring-enhanced intracranial mass lesion in patients, which is compatible with our patient's case.[9,11] Furthermore, CMV may also be associated with ventriculitis, although uncommon.[12] This is also consistent in our case as shown in the CSF analysis.

Ventriculomegaly is a known imaging finding in cCMV and is often associated with underlying brain parenchymal damage or cerebrospinal fluid flow obstruction.[3] In our patient, severe ventriculomegaly (>15 mm) with associated aqueductal stenosis resulted in progressive macrocephaly. Notably, advanced ventriculomegaly can mimic hydrocephalus related to aqueductal stenosis, depending on the timing and severity of the insult.[13,14]

In this case, the aqueductal stenosis may plausibly be related to congenital CMV infection. Developmental abnormalities during embryogenesis are recognized contributors, and intrauterine infections such as CMV have been implicated in this process. CMV can trigger neural inflammation and subsequent scarring, leading to aqueductal narrowing. According to inflammatory theories, persistent infection-driven inflammation may result in fibrosis and constriction of the aqueductal lumen.[15]

A study in Uganda showed that CMV is associated with both congenital and post-infective hydrocephalus, and CMV was frequently detected in the CSF.[14] While the exact mechanism remains unclear, its presence may reflect blood-brain barrier dysfunction—allowing CMV-infected leukocytes to enter the CSF—an underlying immune deficiency that results in prolonged viral shedding, or direct CMV-induced inflammation and necrosis of the ependymal and subependymal regions, which can lead to fibrosis and obstruction of CSF outflow pathways. CMV may initiate the primary infectious or autoimmune cascade that results in hydrocephalus, such as in our case.[14]

Another unique aspect of this case is the presence of an intracranial abscess, which is an uncommon presentation of congenital CMV. As of this writing, no cases of congenital CMV presenting with an abscess have been reported in the literature; however, there have been studies reporting intracranial abscesses associated with CMV infection in immunocompetent and immunocompromised adults.[11,16-18] Cerebral mass lesions due to CMV are nonspecific and difficult to distinguish from other intracranial lesions; however, recent advances in CSF-PCR—demonstrating sensitivities typically exceeding 80% and specificities over 90%—have significantly reduced the reliance on brain biopsy for the evaluation of focal lesions.[18] Another report described a patient with CMV with radiographic findings of focal, enhancing intraparenchymal lesions consistent with marked necrosis but nonspecific for infection or tumor. The mechanism of necrosis remains unclear, though direct CNS invasion by CMV has been proposed, leading to inflammatory necrosis with astrocytic response, particularly after a large viral inoculum.[11] In this case, the focal CNS lesions are consistent with congenital CMV in view of the high viral load on serum PCR and positive CSF antibodies, although the absence of brain biopsy and CSF PCR remains a limitation as it could have strengthened diagnostic certainty.

CONCLUSION

This case highlights an atypical presentation of congenital CMV infection in an infant with macrocephaly, ventriculitis, and intracranial abscess. Presence of these findings in an immunocompetent host underscores the potential for CMV to contribute to uncommon and severe neurological manifestations. This case emphasizes the need for increased clinical suspicion, improved access to diagnostic testing, and consideration of CMV in the differential diagnosis of neonatal infections with atypical neuroimaging findings, even in the absence of known immunodeficiency.

Ethical Considerations

Written informed consent for publication was obtained from the patient's legal guardian (mother).

Statement of Authorship

All authors certified fulfillment of ICMJE authorship criteria.

Author Declaration

All authors declared no conflicts of interest.

Funding Source

None.

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