Case Report

Unusual pattern of magnetic resonance imaging findings in congenital cytomegalovirus infection presenting with congenital sensorineural hearing loss: A case report

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ABSTRACT

Congenital cytomegalovirus (cCMV) infection is the leading non-genetic cause of sensorineural hearing loss (SNHL) in children. This report highlights an atypical magnetic resonance imaging (MRI) presentation: Bilateral anterior temporal pole cysts and diffuse white matter abnormalities in an infant with isolated bilateral SNHL. We present a case of a male infant with bilateral congenital SNHL. MRI showed bilateral anterior temporal cysts and diffuse periventricular white matter hyperintensities with no suspicious antenatal history or more common features such as microcephaly, calcifications, or cortical anomalies. Confirmatory testing revealed cCMV infection. This case underscores the diagnostic value of MRI in detecting subtle manifestations of cCMV. Recognition of anterior temporal pole cysts, even in the absence of classic features, can guide timely diagnosis and treatment, potentially improving auditory and neurodevelopmental outcomes.

Key words: Anterior temporal pole cysts, Congenital cytomegalovirus, Sensorineural hearing loss in infants

Congenital cytomegalovirus (cCMV) is the most common congenital infection globally, affecting 0.5–2% of all live births. Of these, around 10–15% of infants are symptomatic at birth [1], and many may suffer long-term consequences, including sensorineural hearing loss (SNHL), neurodevelopmental delays, motor deficits, and visual impairment. Cytomegalovirus (CMV) is the leading non-genetic cause of SNHL, and hearing loss may be present at birth or manifest later in childhood. The diagnosis can often be challenging in cases where SNHL is the sole manifestation and where imaging findings are atypical or subtle. Classical neuroimaging features of cCMV include periventricular calcifications, polymicrogyria, ventriculomegaly, and migrational abnormalities. However, in recent years, anterior temporal lobe cysts have emerged as a more sensitive, though less widely recognized, magnetic resonance imaging (MRI) marker [2,3].

This case aims to emphasize the importance of identifying these less typical findings and initiating further evaluation, even in infants lacking classical risk factors.

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CASE PRESENTATION

A 9-month-old male child, son of a serving army personnel hailing from a rural area in south-western India, presented with complaints of not responding to verbal cues from parents and delayed speech. The child was initially evaluated in ear, nose, and throat and was referred to radiology as a case of congenital bilateral SNHL for high-resolution computed tomography (CT) of the temporal bone and MRI of the brain and inner ear.

The child was born to a 26-year-old G1P1L0A0 lady, non-consanguineous marriage, and term delivery with no known complications during antenatal or perinatal duration, in a government hospital in the patient's hometown. There was no reported maternal illness, rash, or exposure to known teratogens. Toxoplasmosis, other infections, rubella, CMV, and herpes simplex (TORCH) screening was not performed during pregnancy. Obstetric scans revealed no significant abnormalities. At birth, the child cried immediately, had a normal weight and head circumference.

Physical examination was unremarkable, with no signs of dysmorphia, petechiae, hepatosplenomegaly, or jaundice. Routine newborn hearing screening (Oto-Acoustic Emission) was not done.

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Audiological assessment revealed severe to profound SNHL. Otoacoustic emissions were bilaterally absent, and brainstem-evoked response audiometry showed no detectable waves up to 90 dB. Tympanometry demonstrated bilateral type B curves, and acoustic reflexes were absent, consistent with significant auditory dysfunction. Ophthalmological examination, including fundus evaluation, was unremarkable.

Neuroimaging was notable for normal CT findings, with intact ventricular anatomy and no evidence of calcifications. MRI of the brain revealed multiple well-defined subcortical cysts measuring 5–7 mm, which were hypointense on T1-weighted images, hyperintense on T2-weighted images, and suppressed on fluid-attenuated inversion recovery (FLAIR) sequences. In addition, there were diffuse bilateral T2/FLAIR hyperintensities involving the periventricular and deep white matter, predominantly in the parieto-occipital regions (Figs. 1 and 2). No microcephaly, periventricular calcifications, polymicrogyria, or other migrational anomalies were observed. The bilateral cerebellopontine angles, inner ears, and cranial nerves VII–VIII were structurally normal. MRI findings prompted further investigations (TORCH Panel), which revealed that the child is CMV immunoglobulin (Ig) G positive (66.6 U/mL) and the mother is CMV IgM and IgG positive. MRI findings, combined with early presentation of symptoms and positive CMV serology, supported cCMV as the primary diagnosis.

The child was further evaluated, and bilateral cochlear implantation was done. The child is under routine follow-up and doing well.

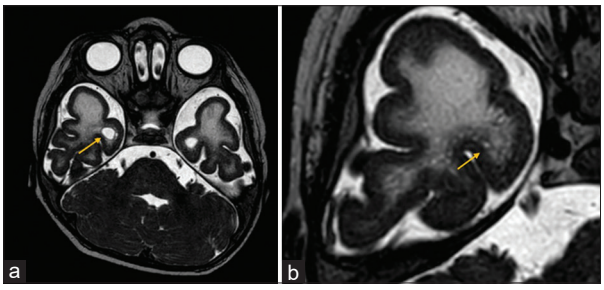

Figure 1: (a) Axial T2 DRIVE image shows multiple subcortical cysts in bilateral temporal lobes and (b) enlarged axial T2 DRIVE images show multiple tiny 2–3 mm subcortical cysts in bilateral temporal lobes

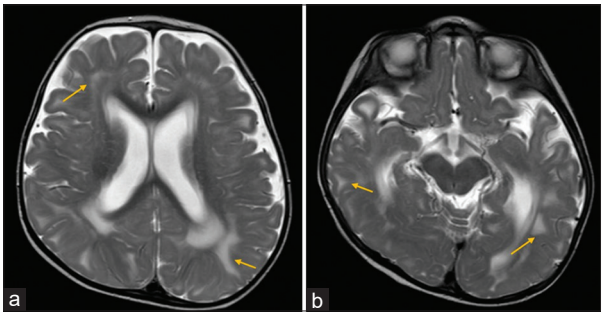

Figure 2: (a) Axial T2 fluid-attenuated inversion recovery (FLAIR) images show periventricular, deep and subcortical white matter hyperintensities involving bilateral frontal and parietal lobes; and (b) axial T2 FLAIR images show periventricular, deep and subcortical white matter hyperintensities involving bilateral temporal lobes

DISCUSSION

cCMV is the most common intrauterine infection and a leading cause of non-genetic SNHL in children. Affecting approximately 0.5–2% of all live births, it accounts for nearly 25% of all cases of permanent hearing loss in childhood [1,4,5]. The clinical presentation of cCMV varies widely, ranging from asymptomatic cases to severe neurodevelopmental impairment, depending significantly on the timing of fetal infection [6]. This case highlights the growing importance of subtle MRI findings, particularly anterior temporal pole cysts, in diagnosing cCMV in infants presenting solely with SNHL. The gold standard for diagnosis of cCMV is the detection of CMV DNA in urine or saliva within the first 21 days of life using polymerase chain reaction [4]. In our case, this confirmatory test could not be performed as the patient presented beyond the diagnostic window. However, the presence of early features of developmental delay, maternal serology showing both CMV IgG and IgM positivity, and supportive child serology raised strong clinical suspicion. The diagnosis was further reinforced by MRI findings, notably anterior temporal pole cysts and white matter signal abnormalities, imaging features characteristic of congenital CMV infection [7]. Together, these clinical, serological, and radiological findings helped clinch the diagnosis in the absence of virologic confirmation.

The neurological manifestations of cCMV are closely linked to the gestational age at which the fetus acquires the infection. First-trimester infection typically results in the most severe brain anomalies, such as lissencephaly, polymicrogyria, ventriculomegaly, cerebellar hypoplasia, and periventricular calcifications, features reflecting major disruption in neuronal migration and cortical development [6,8]. Infection during the second trimester is often associated with delayed myelination, white matter abnormalities, and more subtle migrational defects, without overt cortical malformations [3]. When infection occurs in the third trimester, findings are often milder or even absent on imaging, although the risk of delayed-onset SNHL or neurocognitive impairment persists [1].

MRI has proven to be a superior imaging modality compared to CT in detecting subtle brain abnormalities in cCMV. Classical CT features such as periventricular calcifications may be absent, especially in infants diagnosed later or with isolated hearing loss [2,9]. In this case, the MRI revealed bilateral anterior temporal pole cysts, which have emerged in recent literature as a highly specific, though under-recognized, finding in cCMV [2,3,9]. These cysts are believed to reflect a destructive viral process in the developing temporal lobes, typically presenting bilaterally and symmetrically. Their presence, particularly in infants with unexplained SNHL, should prompt immediate serological evaluation for CMV. The coexisting periventricular and deep white matter T2/FLAIR hyperintensities in this child likely represent gliosis or delayed myelination, which, although

nonspecific, gain diagnostic significance when found alongside temporal cysts [9]. Importantly, the inner ear structures and cranial nerves were normal, pointing toward a central etiology of hearing loss.

Management of cCMV hinges on early detection. Antiviral therapy such as valganciclovir is most effective when initiated within the 1st month of life, especially in symptomatic infants [7]. While the benefit of antiviral therapy in isolated SNHL remains under investigation, its consideration is justified in cases with neuroimaging evidence of brain involvement. In this child, diagnosed at 9 months, cochlear implantation was chosen as the primary intervention, enabling timely auditory rehabilitation. Ongoing audiological follow-up is essential given the risk of progression and potential late-onset hearing deterioration. Furthermore, early auditory stimulation through hearing aids or implants supports speech and language development during critical early periods [9].

This case reinforces the growing body of evidence supporting the diagnostic value of anterior temporal pole cysts on MRI in cCMV. Studies by Rangankar *et al*., Gowda *et al*., and Alarcón *et al.* have collectively emphasized the relevance of these findings in the absence of more classical markers [2,3,8]. Radiologists and pediatricians must remain vigilant for these subtle signs, particularly in infants with unexplained SNHL, as early diagnosis facilitates timely intervention and can significantly improve long-term developmental outcomes. However, anterior temporal pole cysts with associated white matter signal abnormalities are not entirely pathognomonic, and a focused set of alternative diagnoses must be considered in this imaging context.

Genetic causes of SNHL typically show normal brain MRI without characteristic temporal pole cysts. Other congenital infections (toxoplasmosis, rubella, and herpes simplex virus) usually demonstrate calcifications, ocular involvement, or diffuse cortical abnormalities. Cystic leukoencephalopathy without megalencephaly presents with anterior temporal cysts but is commonly associated with macrocephaly and progressive leukodystrophy. Aicardi-Goutières syndrome is characterized by intracranial calcifications, severe developmental delay, and inflammatory markers. Metabolic leukodystrophies generally show macrocephaly and widespread symmetric white matter disease. The absence of these features, together with the characteristic MRI pattern and supportive serology, favored congenital CMV infection as the final diagnosis.

CONCLUSION

This case highlights an atypical but diagnostically important MRI pattern in congenital CMV, bilateral anterior temporal pole cysts with white matter abnormalities, even in the absence of classical imaging features like calcifications. MRI plays a critical role in early diagnosis, particularly in infants presenting with isolated SNHL. Prompt recognition facilitates appropriate clinical management, with potential implications for hearing and developmental outcomes. In cases detected later, hearing aids are essential for auditory stimulation and language development, while further evaluation guides decisions on cochlear implantation or other interventions. A multidisciplinary approach ensures optimal long-term outcomes in affected children.

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