Sensorineural Hearing Loss in a Pediatric Population

Association of Congenital Cytomegalovirus Infection With Intracranial Abnormalities

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Objectives: To examine the incidence of congenital cytomegalovirus (CMV) infection relative to common genetic etiologies of hearing loss in a pediatric population with sensorineural hearing loss (SNHL), and to characterize intracranial radiological abnormalities in patients with CMV-associated hearing loss.

Design: Retrospective study.

Setting: Academic tertiary care center.

Patients: A total of 112 pediatric patients with confirmed SNHL.

Main Outcome Measures: The association of congenital CMV infection status with abnormal brain magnetic resonance imaging (MRI) scans and the frequencies of congenital CMV infection, gap junction H9252-2 (GJB2) mutations, and the mitochondrial DNA (mtDNA) 1555A>G mutation in children with SNHL.

Results: Of 109 patients, 11 (10%) had positive results for CMV DNA; 10 of the 11 had normal GJB2 sequence and had negative test results for the mtDNA 1555A>G mutation. Brain MRI scans for 97 patients demonstrated a higher proportion of abnormalities in patients with positive CMV test results (80%) compared with those with no detectable CMV DNA (33%) \( (P=0.006) \). GJB2 mutations and the mtDNA 1555A>G mutation were seen in 10 of 88 patients (11%) and 1 of 97 patients (1%) with SNHL, respectively.

Conclusions: The presence of brain abnormalities in most patients with congenital CMV infection suggests that neurological damage in otherwise asymptomatic patients may not be limited to SNHL. Congenital CMV infection accounted for a significant proportion of patients with SNHL, with an incidence rate comparable with that of GJB2-related SNHL.


The etiology of hearing loss in children is almost equally attributed to genetic and environmental factors.\(^1\) A comprehensive diagnostic workup for the patient with sensorineural hearing loss (SNHL) is vital for guiding treatment and intervention options. Mutations in the gap junction β-2 (GJB2) gene that encodes connexin 26 are the most common cause of hereditary nonsyndromic SNHL,\(^2\) whereas congenital cytomegalovirus (CMV) infection is the most common nongenetic cause of SNHL. Nance et al\(^3\) estimated that CMV infection accounts for approximately 21% of cases of congenital SNHL and 25% of cases of SNHL by the age of 4 years. In addition to laboratory studies for diagnosis of inherited and infectious causes of SNHL, central nervous system magnetic resonance imaging (MRI) provides a useful tool for identification of intracranial and inner ear abnormalities.\(^4\) The SNHL evaluation at our institution includes brain MRI studies, molecular testing for CMV DNA from newborn dried blood spots (DBSs) to diagnose congenital CMV infection, and testing for GJB2 gene mutations. In the present study, we examined the incidence of congenital CMV infection relative to common genetic etiologies of hearing loss in a pediatric population with SNHL and characterized intracranial radiological abnormalities in patients with CMV-associated hearing loss. The incidence of the mitochondrial DNA (mtDNA) 1555A>G mutation, which is associated with aminoglycoside ototoxic effects and has been implicated in SNHL in the absence of aminoglycoside therapy, was examined retrospectively.\(^5\)

Congenital CMV infection occurs in approximately 1% of live births in the United States (National Congenital CMV Disease Registry\(^6\)) through transplacental transmission of a primary maternal infection acquired during pregnancy or by reactivation of a prior infection.\(^7\) It is associated with a spectrum of symptoms ranging

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from asymptomatic infection to severe multisystemic disease. Up to 90% of CMV-infected children are asympto-
matic at birth and do not present with overt clinical symp-
toms of CMV infection; however, they are at risk for later-
onset CMV sequelae, particularly SNHL.3,8 Hearing loss
resulting from congenital CMV infection varies in sever-
ity, is generally progressive, and may be bilateral or unil-
somal. Some patients have congenital hearing loss, but those
who develop delayed-onset hearing loss would not be de-
tected on newborn hearing screening.9 Additional symp-
toms of severe, multisystemic congenital CMV infection in-
clude petechiae, microcephaly, jaundice, seizures, and other
neurological abnormalities. Congenital CMV infection is
associated with a variety of brain abnormalities, such as
dilated ventricles, lissencephaly, gyral anomalies, white
matter abnormalities, paraventricular cysts, and cerebel-
lar hypoplasia.10,11

Because CMV infection is endemic in the general popu-
lation, positive CMV test results in an older infant or child
do not differentiate between prenatally or postnatally ac-
quired CMV. Molecular analysis of newborn DBSs for
CMV DNA has facilitated retrospective diagnosis of con-
genital CMV infection beyond the perinatal period.12 The
current study is a retrospective analysis of intracranial
radiological abnormalities in pediatric patients with CMV-
related hearing loss and a comparison of the incidence
of congenital CMV infection relative to common ge-
etic etiologies of SNHL.

**METHODS**

**PATIENTS**

The study population consisted of 112 patients (70 boys and
42 girls) ranging in age from 1 to 5 years who failed newborn
hearing screening with confirmed SNHL or had confirmed hear-
ing loss at a later age (Table 1). The racial makeup was 58%
white, 15% Hispanic, 10% African American, 1% Native Ameri-
can, and 16% other. Congenital hearing loss was present in 109
children who were identified at birth through the newborn hear-
screening examination. For the remaining 3 children, the age at onset of hearing loss ranged from 9 to 24 months. Four patients presented with symptoms of CMV infection, 3 of whom had a positive perinatal screen either by urine culture or IgM antibodies. One patient with a history of intrauterine growth restriction had a prenatal diagnosis of CMV infection confirmed at 29 weeks by amniocentesis. Molecular testing was performed in a Clinical Laboratory Improvement Amendments–certified and College of American Pathology–accredited laboratory as part of the diagnostic workup for SNHL. The study was approved by the biomedically institutional review board of the University of North Carolina at Chapel Hill.

**AUDIOLOGICAL ASSESSMENTS**

All children were referred to the study institution based on an abnormal result obtained on the newborn infant hearing screening examination. All children were tested using automated audiological brainstem response (ABR) testing equipment. Following confirmatory testing, diagnostic ABR was used to estimate thresholds and make recommendations regarding the need for amplification. Conventional behavioral audiometry was then used to measure thresholds using age-appropriate methods when possible. The degree of hearing loss was classified as mild, moderate, severe, or profound.

**NEUROIMAGING STUDIES**

The cranial MRI protocol used for the assessment of SNHL in children at our institution includes whole-brain axial and sag-
tittal unenhanced spin-echo T1-weighted images, axial fast spin-
echo T2-weighted and fluid-attenuated inversion recovery imag-
es, and axial echo-planar diffusion-weighted images, as well as high-resolution, 3-dimensional, constructive interference steady state (CISS) or RESTORE images through the temporal

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**Table 1. Summary of Hearing Loss, Genetic Testing, and Magnetic Resonance Imaging (MRI) Results in CMV-Positive and CMV-Negative Patients**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Total</th>
<th>CMV Positive</th>
<th>CMV Negative</th>
<th>CMV NA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total No.</td>
<td>112</td>
<td>11</td>
<td>98</td>
<td>3</td>
</tr>
<tr>
<td>Degree of hearing loss, No.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mild</td>
<td>20</td>
<td>0</td>
<td>20</td>
<td>0</td>
</tr>
<tr>
<td>Moderate</td>
<td>23</td>
<td>1</td>
<td>21</td>
<td>1</td>
</tr>
<tr>
<td>Severe</td>
<td>16</td>
<td>1</td>
<td>14</td>
<td>1</td>
</tr>
<tr>
<td>Profound</td>
<td>52</td>
<td>9</td>
<td>42</td>
<td>1</td>
</tr>
<tr>
<td>Unknown</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Unilateral (vs bilateral), No.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bilateral</td>
<td>95</td>
<td>8</td>
<td>84</td>
<td>3</td>
</tr>
<tr>
<td>Unilateral</td>
<td>17</td>
<td>3</td>
<td>14</td>
<td>0</td>
</tr>
<tr>
<td>GJB2 SNHL</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>mtDNA 1555A&gt;G</td>
<td>1 of 97</td>
<td>0 of 10</td>
<td>10 of 78c</td>
<td>0</td>
</tr>
<tr>
<td>MRI results</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Available (positive), No.</td>
<td>97 (37)</td>
<td>10 (8)</td>
<td>85 (28)</td>
<td>2 (1)</td>
</tr>
<tr>
<td>Unavailable, No.</td>
<td>15</td>
<td>1</td>
<td>13</td>
<td>1</td>
</tr>
</tbody>
</table>

Abbreviations: CMV, cytomegalovirus; mtDNA, mitochondrial DNA; NA, not applicable; SNHL, sensorineural hearing loss.

a Based on severity of the worst affected ear.
b Based on the presence of 2 deleterious GJB2 mutations.
c Three additional patients had positive test results for 35delG, the most common GJB2 mutation.
Table 2. Cytomegalovirus (CMV)-Positive Patients: Audiological and Intracranial Imaging Findings

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Laterality of HL</th>
<th>Severity of HL</th>
<th>ABR R</th>
<th>ABR L</th>
<th>Clinical CMV</th>
<th>Brain MRI Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Bilateral</td>
<td>Severe</td>
<td>NR</td>
<td>NR</td>
<td>N</td>
<td>Bilateral WM signal abnormalities in the frontal, parietal, and temporal lobes</td>
</tr>
<tr>
<td>2</td>
<td>Bilateral</td>
<td>Profound, P</td>
<td>NR</td>
<td>NR</td>
<td>N</td>
<td>Nonspecific WM signal abnormalities in the frontal, parietal, and temporal lobes; question of right frontal cortical dysplasia</td>
</tr>
<tr>
<td>3</td>
<td>Bilateral</td>
<td>Profound, CM</td>
<td>CM</td>
<td>NR</td>
<td>Y</td>
<td>Nonspecific WM signal changes predominantly in temporal and parietal lobes extending to U (arcuate) fibers; mild ventriculomegaly</td>
</tr>
<tr>
<td>4</td>
<td>Unilateral</td>
<td>Profound</td>
<td>NR</td>
<td>NL</td>
<td>Y</td>
<td>Diffuse deep and superficial WM signal abnormalities in all lobes</td>
</tr>
<tr>
<td>5</td>
<td>Bilateral</td>
<td>Profound</td>
<td>NR</td>
<td>NR</td>
<td>N</td>
<td>Diffuse cortical dysplasias and WM signal abnormalities (frontal, posterior temporal, occipital, and parietal); ventriculomegaly with enlarged temporal horns</td>
</tr>
<tr>
<td>6</td>
<td>Unilateral</td>
<td>Profound</td>
<td>NL</td>
<td>NR</td>
<td>N</td>
<td>Normal MRI findings</td>
</tr>
<tr>
<td>7</td>
<td>Bilateral</td>
<td>Profound</td>
<td>NR</td>
<td>NR</td>
<td>Y</td>
<td>Microcephaly; cortical dysplasia (cobblestone lissencephaly); diffusely abnormal WM; probably old L basal ganglia hemorrhage; ventriculomegaly</td>
</tr>
<tr>
<td>8</td>
<td>Bilateral</td>
<td>Profound</td>
<td>NR</td>
<td>NR</td>
<td>Y</td>
<td>Nonspecific foci of WM abnormality in the parieto-occipital regions bilaterally</td>
</tr>
<tr>
<td>9</td>
<td>Bilateral</td>
<td>Profound</td>
<td>NR</td>
<td>NR</td>
<td>Y</td>
<td>Bilateral cortical dysplasias (suggestive of polymicrogyria); abnormal deep and superficial WM signal abnormalities in all lobes; thinning of corpus callosum; ventriculomegaly with prominent temporal horns</td>
</tr>
<tr>
<td>10</td>
<td>Bilateral</td>
<td>Moderate, P</td>
<td>NR</td>
<td>NR</td>
<td>N</td>
<td>Normal MRI findings</td>
</tr>
</tbody>
</table>

Abbreviations: ABR L, ABR auditory brainstem response, left ear; ABR R, auditory brainstem response, right ear; CM, cochlear microphonic; HL, hearing loss; MRI, magnetic resonance imaging; N, no; NA, not available; NL, normal estimated thresholds (<25 dB HL); NR, no response for clicks and 250-Hz and 1-kHz tone bursts at 90 dB HL; P, progressive; WM, white matter; Y, yes (presence of clinical CMV infection).

MOLECULAR DETECTION OF CMV DNA

Following parental consent, a perinatal DBS card was requested from the North Carolina State Laboratory of Public Health. The DNA extraction and molecular detection of CMV DNA from the DBS card was performed according to a previously validated real-time polymerase chain reaction (PCR) assay. The assay has been shown to reliably detect approximately 1000 CMV copies/mL of blood. Each sample was run in duplicate, and positive, negative, and no-template controls were included in each run.

CONNEXIN 26 (GJB2) MUTATION ANALYSIS

The GJB2 full gene sequencing was performed for 88 patients. The coding region (exon 2) and flanking intronic regions of GJB2 were amplified by PCR, followed by bidirectional sequencing with the BigDye Terminator kit (Applied Biosystems, Carlsbad, California). Sequence analysis was performed using SeqScape software (Applied Biosystems) and visual inspection of the electropherograms. For 3 patients who were evaluated prior to implementation of the full gene sequencing test, analysis was limited to the 35delG mutation by allele-specific PCR and gel electrophoresis.

MITOCHONDRIAL DNA 1555A>G MUTATION ANALYSIS

The presence of the mtDNA 1555A>G mutation was analyzed by melting curve analysis on the LightCycler (Roche Diagnostics Corp, Indianapolis, Indiana). Hybridization probes were designed to yield distinct melting curves for the wild-type and mutant alleles, respectively.

RESULTS

Testing results for congenital CMV infection, hearing loss, genetic analysis, and neuroimaging by MRI are summarized in Table 1. Nearly half of the patients (52 of 112 [46%]) had profound hearing loss, 95 of 112 (85%) had bilateral hearing loss, and 16 of 112 (14%) had progressive hearing loss. Eleven of 109 patients (10%) tested positive for CMV DNA from the perinatal DBS card (amplifiable DNA could not be obtained for 3 patients). This included all 4 patients presenting with symptomatic CMV infection and 1 patient who had been diagnosed as having CMV infection prenatally. Except for 1 patient with moderate hearing loss, all of the 11 patients who tested positive for congenital CMV infection had either severe or profound hearing loss, and 2 patients had progressive hearing loss. In all 11 patients, SNHL was present at birth. All the patients with a positive CMV DNA result had normal mtDNA 1555A>G results, and 10 of 11 of the patients had normal GJB2 full-sequence analysis; the remaining patient did not have testing for GJB2 mutations. All of the children with positive CMV test results were considered significant.
Cytomegalovirus (CMV) Status

<table>
<thead>
<tr>
<th>MRI Findings</th>
<th>CMV-Positive (n=10)</th>
<th>CMV-Negative (n=87)</th>
</tr>
</thead>
<tbody>
<tr>
<td>White matter signal changes</td>
<td>8 (80)</td>
<td>13 (15)</td>
</tr>
<tr>
<td>Cortical dysplasia</td>
<td>4 (40)</td>
<td>1 (1.1)</td>
</tr>
<tr>
<td>Ventriculomegaly</td>
<td>4 (40)</td>
<td>5 (5.7)</td>
</tr>
<tr>
<td>Dysgenesis or dysmorphism of the corpus callosum</td>
<td>0</td>
<td>5 (5.7)</td>
</tr>
<tr>
<td>Prominent extra axial cerebral spinal fluid</td>
<td>0</td>
<td>5 (5.7)</td>
</tr>
</tbody>
</table>

Table 3. Intracranial Imaging Findings Based on Patient Cytomegalovirus (CMV) Status

Abbreviation: MRI, magnetic resonance imaging.

The current study population consisted of patients who were referred to an otolaryngology clinic and who had failed newborn hearing screening and/or had confirmed hearing loss. Because a reliable diagnosis of congenital CMV infection is limited to samples obtained within the first 3 weeks after birth, retrospective diagnosis of congenital CMV infection by molecular analysis of newborn DBS cards is of value in guiding appropriate medical treatment, early intervention, and avoiding further testing. A positive CMV result was obtained for 11 of 109 patients (10%), which is comparable with a previously published report for children with congenital hearing loss. Considering that as many as 50% of congenitally infected patients develop late-onset hearing loss, it has been estimated that congenital CMV infection accounts for 21% to 25% of SNHL cases. Most CMV-positive patients had profound hearing loss that required cochlear implantation, and progression was observed in 2 patients at ages 2 and 4 years, respectively.

The incidence of congenital CMV infection was comparable with that of connexin 26–related hearing loss in our study population. Mutations in the GJB2 gene that encodes connexin 26 are the most common cause of hereditary nonsyndromic hearing loss; therefore, testing for GJB2 mutations is recommended in the workup for individuals with nonsyndromic SNHL. GJB2-related hearing loss was identified in 10 of 88 of our patients (11%). All 10 patients were white, consistent with the high prevalence of GJB2 mutations in that population. The mtDNA 1555A>G mutation, which is associated with aminoglycoside ototoxic effects and nonsyndromic SNHL, was identified in 1 patient, but its role in the etiology of SNHL is unclear because the patient had no history of exposure to aminoglycosides. The low (1%) prevalence of the mtDNA 1555A>G mutation in children with hearing loss in the current study is consistent with that of a previous report. One limitation of this study is that GJB2 muta-
tion testing was not available from all patients, including 1 of the 11 CMV-positive patients, and 3 patients had testing for only the most common 35delG mutation. Other, less common genetic causes of SNHL cannot be excluded in these patients.

Brain abnormalities were significantly more prevalent in patients with congenital CMV infection (80%) compared with those without CMV (33%). Congenital CMV infection is associated with a variety of brain abnormalities, such as dilated ventricles, lissencephaly, gyral anomalies, white matter abnormalities, paraventricular cysts, and cerebellar hypoplasia. By far, the most common brain finding we observed in our sample of CMV-positive patients was a hemispheric white matter signal abnormality of variable extent. Unfortunately, white matter abnormalities are not pathognomonic of CMV infection because similar white matter signal changes of unknown etiology are a common finding in non–CMV-related pediatric neurological disorders. This fact was underscored by the presence of white matter lesions in a substantial number (13 of 87 [15%]) of CMV-negative patients in our study (Table 3).

Although the imaging findings identified do not allow reliable differentiation between CMV-positive and CMV-negative patients, certain features were more suggestive of CMV as an etiology of hearing loss. Specifically, cortical dysplasias were more frequently seen in patients with congenital CMV infection (4 of 10 [40%]) than in non–CMV-infected patients (1 of 87 [1%]). In addition, our findings are in keeping with those of van der Knapp et al, who suggested that the neuroimaging pattern of white matter abnormalities, particularly those involving the parietal region, with or without gyral abnormalities, is characteristic of congenital CMV etiology. All of the CMV-positive patients in our sample with brain abnormalities had white matter signal changes that involved the parietal lobes. Additional studies with larger sample sizes might better define imaging abnormalities that are specific and diagnostic for congenital CMV infection.

One of the patients (patient 6) (Table 2), who was diagnosed as having profound unilateral hearing loss after failing the newborn hearing screening test, had abnormal brain findings at 11 months of age, including diffuse cortical dysplasia and white matter abnormalities, and later developed seizures and developmental delay consistent with congenital CMV infection. A previous study has reported seizures and SNHL in patients with symptomatic CMV, and a recent study demonstrated that asymptomatic congenital CMV infection is a significant cause of developmental delay. These results suggest that a subset of children with congenital CMV infection who present with SNHL but who seem to be otherwise healthy may be at risk of developing additional neurological sequelae. Studies with a larger group of patients with symptomatic congenital CMV infection are needed to evaluate whether children with congenital CMV infection who present with isolated SNHL are at risk of developing additional neurological sequelae.

In this study, we found brain abnormalities consistent with congenital CMV infection in a significant propor-

### Table 4. GJB2 Sequencing Results

<table>
<thead>
<tr>
<th>Classification</th>
<th>GJB2 Allele 1</th>
<th>GJB2 Allele 2</th>
<th>Patients, No.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Autosomal recessive</td>
<td>35delG</td>
<td>35delG</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>35delG</td>
<td>139G&gt;T</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>35delG</td>
<td>71G&gt;A (W24X)</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>35delG</td>
<td>167delT</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>35delG</td>
<td>269T&gt;C (L90P)</td>
<td>1</td>
</tr>
<tr>
<td>Heterozygotes</td>
<td>35delG</td>
<td>NA</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>416G&gt;A</td>
<td>NA</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>(S139N)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>510G&gt;A</td>
<td>NA</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>(R184Q)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>101T&gt;CT</td>
<td>NA</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>(M34T)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>249C&gt;G</td>
<td>NA</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>(F83L)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Abbreviation: NA, not applicable.  
May be associated with autosomal dominant hearing loss.  
Variants of uncertain clinical significance.
portion of CMV-positive patients with SNHL who had no family history of hearing loss or evidence of any other causes of hearing loss, including common genetic causes of SNHL. These findings are supportive of, but are not proof of, an etiologic role for occult congenital CMV in hearing loss in this study. Although congenital CMV infection may be incidental to hearing loss, there is mounting evidence that occult congenital CMV infection may account for a significant proportion of SNHL in children. The significant role that CMV plays in SNHL has prompted discussions regarding establishment of newborn screening for this common infection, raising issues such as study methods, target population, cost-effectiveness, and cost benefit.17 Such a program would be particularly advantageous in early identification of patients who are at risk of delayed-onset hearing loss and who would not be detected through the newborn hearing screen. In addition, recent studies18,19 show that ganciclovir therapy, when initiated early in symptomatic patients, may halt the onset of hearing loss. A 7-year multicenter study funded by the National Institute on Deafness and Other Communication Disorders to examine the role of congenital CMV infection in hearing loss is expected to elucidate the feasibility of adding CMV to newborn screening programs.

In conclusion, this study indicated that in a U.S. tertiary referral center the incidence of hearing loss associated with congenital CMV infection (approximately 10% of cases) is similar to the incidence of hearing loss caused by mutation in GJB2 (approximately 11% of cases), the most common cause of inherited congenital SNHL, and emphasizes that both tests should be considered in the workup of children with isolated SNHL. Abnormal brain MRI findings, consisting primarily of white matter abnormalities extending to different lobes of the brain, were significantly more prevalent in patients with congenital CMV infection compared with those with SNHL resulting from other or unknown causes. Limitations of this study include the small number of patients, partial missing data, and inclusion limited to children with hearing loss causing potential ascertainment bias. Additional studies are needed to assess the impact of central nervous system damage in patients with congenital CMV infection who are otherwise asymptomatic.

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Author Contributions: Drs Kimani, Buchman, Booker, Huang, Castillo, and Weck had full access to all the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis. Study concept and design: Buchman, Powell, and Weck. Acquisition of data: Kimani, Buchman, Booker, Huang, Castillo, and Weck. Analysis and interpretation of data: Kimani, Buchman, Booker, Powell, and Weck. Drafting of the manuscript: Kimani, Buchman, Huang, Castillo, Powell, and Weck. Critical revision of the manuscript for important intellectual content: Kimani, Buchman, Booker, Huang, Castillo, Powell, and Weck. Administrative, technical, and material support: Buchman, Powell, and Weck. Study supervision: Buchman, Booker, Powell, and Weck.

Financial Disclosure: None reported.

REFERENCES


**Correction**

Error in Table. In the article titled “Sensorineural Hearing Loss in a Pediatric Population: Association of Congenital Cytomegalovirus Infection With Intracranial Abnormalities” published in the October issue of the Archives (2010;136[10]:999-1004), there was an error in Table 3. The column subheading “CMV-Negative (n=7)” should have read “CMV-Negative (n=87).”