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Role of cytomegalovirus in sensorineural hearing loss of children: A case—control study Tehran, Iran

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KEYWORDS

Cytomegalovirus; Congenital cytomegalovirus; Sensorineural hearing loss; Ganciclovir

Summary

Background: Congenital CMV is the most common cause of nonhereditary sensorineural hearing loss in children. More than 40% of deafness cases with an unknown cause are due to congenital CMV. When CMV infection is diagnosed in the first year of life, treatment with gancyclovir is recommended and is associated with diminished occurrence of SNHL. Previous studies in Tehran showed congenital CMV infection developed in 2.6% of neonates.

Objective: To determine the role of cytomegalovirus in sensorineural hearing loss in children by comparing the CMV antibodies quantitatively between SNHL and controls.

Methods and materials: This case—control study was done in the ENT Department of Hazrat Rasoul Akram Hospital in Tehran (2002–2003). This study was carried out based on diagnostic parameters (AAO criteria) for SNHL and a healthy control. We compared the specific cytomegalovirus antibodies (IgM and IgG measured by ELISA method) in 95 blood samples of infants with SNHL (mean age = 35 \pm 30 months) and 63 healthy matched infants (mean age = 38.7 \pm 27.3 months) as controls. 59.6% of cases were male; 40.4% were female. Acute and previous immunity to cytomegalovirus (IgM and IgG) was found in 34.7% and 72.6% of SNHL children, acute infection detected in 3.5% of controls, previous immunity (IgG) detected in 94.7% of them. Acute CMV infection was higher in the cases (*P*-value = 0.000) but previous immunity (CMV-IgG) was higher in the controls (*P*-value = 0.001). Mean age of cases with acute

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infection (CMV-IgM) was 40 months and for previous immunity (CMV-IgG) was 35 months in SNHL cases and 40 months in controls, respectively.

Conclusion: Cytomegalovirus is one of the most common infectious agents in SNHL children compared to the healthy children. Probably both congenital and acquired CMV can induce progressive hearing loss in our cases. We prefer at least in our country to consider seropositive (CMV-IgM) SNHL children (less than 1 year old) as congenital form. But we are not able to differentiate the congenital from the acquired infection in seropositive (CMV-IgM) SNHL children after first year of life. It should be subjected to randomized controlled trial for treatment of acquired type of CMV infection in SNHL children with ganciclovir.

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1. Introduction

According to Ohlms et al.'s study, in 38.75% of children with SNHL the etiological factor remained unknown. In 7.5% of cases unilateral deafness were diagnosed accidentally, most often between the 7th and the 10th year of age [1,2].

Neither children nor their parents can precisely determine the time of its appearance, especially when it is not accompanied by other symptoms, such as dizziness or tinnitus [1,2].

Congenital CMV is one of the most common causes of congenital infections in developed countries with reported incidences varying between 0.15% and 2.0% with the higher rates in populations with a lower standard of living [3—7].

The effects of congenital CMV infection may vary from a congenital syndrome to an asymptomatic course. Infants that are asymptomatic at birth may still present handicaps at a later age [3,4]. In approximately 5% of infants CMV becomes clinically manifest with damage to many organs including the liver, spleen, brain, eye, and inner ear [5,6,7]. Most infants who are infected congenitally with CMV are asymptomatic at birth.

Many studies suggest that congenital CMV infection has a more relevant role in the etiology of SNHL than previously reported. More than 40% of deafness cases with an unknown cause, needing rehabilitation, are caused by congenital CMV [6,7].

The percent of congenital CMV cases alone appears to account for all the cases previously attributed to all congenital infections. Sensorineural hearing loss (SNHL) is found in most of the clinically affected children and accounts for approximately 4000 cases of deafness yearly in the US. This hearing loss is symmetric and may be progressive [7].

Yet 10—17% of these infants may have unilateral or bilateral deafness later (which is often progressive), differences in higher level auditory function and possibly other neurodevelopmental sequelae [7].

Diagnosis of congenital CMV is by CMV detection in the urine [9]. Detection of IgM antibody in blood [8,9] or perilymphatic fluid [4,10].

The diagnosis of perinatal infection with CMV is difficult but is documented best by negative CMV viral culture and CMV-IgM antibody level at birth, positive viral culture and CMV-IgM antibody at 8–16 weeks of age antibody and persistence of CMV-IgG antibody. Detection of antibody or virus in urine after the first year of life is of no use as most children develop immunity to the virus [8,9].

Postnatal primary CMV infection is diagnosed by a CMV-IgG seroconversion presence of CMV-IgM antibody and viral shedding in salvia, urine and other bodily fluids [4,8,10].

Treatment of children with congenital cytomegalovirus infection with ganciclovir recommended [11,9].

Previous studies in Tehran [12,13] showed congenital CMV infection developed in 2.6% of neonates.

Also CMV was the most common cause of TORCH in "TORCH-suspected infants less than 1 year old" [12].

Due to significant role for congenital CMV infection in the etiology of SNHL, we decided to study the role of CMV in deaf children (<10Y).

Main goal: To determine the role of cytomegalovirus in sensorineural hearing loss in children by comparing the CMV antibodies quantitatively between SNHL and controls.

2. Methods

This case—control study was carried out in the ENT Department of Hazrat Rasoul Hospital in Tehran (2002—2003).

Our study group consists of 95 children with SNHL and 63 children without SNHL as controls (healthy group). The age of children in all cases and controls were less than 14 years.

Diagnostic parameters for SNHL were based on AAO (American academy of Otolaryngology)

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