ORIGINAL RESEARCH PAPER

Volume-7 | Issue-2 | February-2018 | PRINT ISSN No 2277 - 8179

INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

TORCH SCREENING FOR COCHLEAR IMPLANTATION – A PRELIMINARY STUDY

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ABSTRACT

BACKGROUND: TORCH infection in early life is an established risk factor for severe to profound sensorineural hearing loss(SNHL). TORCH screening is used mainly for screening pregnant women and infants. If the screening had been performed early on, early management could prevent progression and stabilize hearing.

OBJECTIVE: We aimed to ascertain the role of TORCH (toxoplasma, rubella, cytomegalovirus, and herpes simplex) infection and screening in children with bilateral profound SNHL in comparison with children having normal hearing.

METHOD: Review of TORCH screening results in 2015 from 338 patients with bilateral profound SNHL(cases) & underwent cochlear implants between 2004 and 2015 at Department of ENT & Head-Neck Surgery, B. J. Medical College, Ahmedabad as well as 523 children with normal hearing(control) was conducted. Age group of both group was between 1 to 14 years. TORCH antibody status of both groups was compared.

RESULT: From 338 patients, 230(68.04%) positive for cytomegalovirus (CMV) IgG antibody, 146(43.2%) were positive for Rubella virus IgG antibody, and 14(4.14%) were positive for herpes simplex virus IgG antibody, 10(3%) were positive for toxoplasma virus IgG antibody and 9(2.67%) were positive for TB IgG antibody.

CONCLUSION: The data obtained suggest that IgG positivity of TORCH in test group was significantly high in comparison with control group which suggests past history of TORCH infection during childhood as most important etiology of SNHL. Though TORCH screening is not useful for the management of deaf child, If the TORCH screening was performed early that could be better prevention & treatment of childhood deafness.

KEYWORDS

Introduction

The acronym TORCH (Toxoplasma, Rubella, Cytomegalovirus, Herpes Simplex) was introduced in 1971 by Nahmis *et al*.[1] Among the many causes of hearing loss, viruses often are ignored. Viral infections, in particular Cytomegalovirus (CMV), cause up to 40% of all congenitally acquired hearing loss. Many viruses can be the cause of congenital or acquired hearing loss (Table 1) [2]. Typically, viruses cause sensorineural hearing loss (SNHL); however, a viral etiology has been proposed for Otosclerosis(Table 2)[3]. Infection with HIV can lead to conductive hearing loss (CHL) through bacterial and fungal infections, which become more frequent following the immunosuppression caused by that virus. SNHL is detected at birth in 5%–10% of neonates with evidence of congenital CMV infection. In addition, longitudinal studies of infants with congenital CMV infection clearly have shown that many infants who are asymptomatic at birth and have normal hearing will develop SNHL during early childhood. Table 3 shows potential etiologies of hearing loss due to different viral infections.

Table 1 – Infectious causes of deafness[2]

Congenital Infections	Acquired Infections
Cytomegalovirus	Borrellia burgdorferi
Lymphocytic choriomeningitis virus	Epstein-Barr virus
Rubella virus	Haemophilus influenzae
Toxoplasma gondii	Lassa virus
Treponema pallidum	Measles
	Mumps
	Neisseria meningitides
	Non – polio enteroviruses
	Plasmodium falciparum
	Streptococcus pneumoniae
	Varicella zoster virus

Universal newborn screening for hearing loss misses a number of children who will ultimately have late-onset SNHL that is linked to congenital CMV infection [4]. Neonatal herpes may be diagnosed similarly, but the features of neonatal herpes may not develop for 10–14 days after birth, and about a fifth of infected babies have no skin lesions. Failure to identify infants who may benefit from early intervention programs or hearing aids could delay the institution of useful interventions.

Table -2 – Viral causes of hearing loss[3]

Viruses	Type of HL	Degree of HL	Incidence of HL	Prevention	Treatment
Congenital CMV	Bilateral progressive SNHL	Severe	6-23% if assymptomatic; 22-65% if symptomatic	None	Ganciclovir, val-ganciclovir, cidofovir, foscarnet
Rubella	Bilateral SNHL	Mild to severe	12-19%	MMR	None
LCMV	Bilateral SNHL	Severe to profound	7.4%	Avoidance of exposure	Ribavirin, favipiravir
Congenital and acquired HIV	SNHL, CHL, mixed	SNHL: mild to moderate CHL: mild to maximal	27.5-33.5%	Post exposure antiretroviral	HAART
HSV	Unilateral or Bilateral SNHL	Moderate to profound	Upto 33% (congenital)	None	Acyclovir
Acquired measles	Bilateral SNHL	Profound	0.1-3.4%	MMR,IV Ig	None
VZV	Unilateral SNHL	Mild to moderate	7-85%	Zostavax	Acyclovir, prednisolone
Mumps	Unilateral SNHL	Variable	0.005-4%	MMR	None
WNV	Bilateral SNHL	Mild to profound	Rare	Vaccine in trials	None

Table 3 – Potential aetiologies of hearing loss due to different viral infections[3]

Infection	Affected site
CMV	Host immune response to viral antigen affecting organ of corti, spiral ganglion
Rubella	Stria vascularis, organ of corti
Herpes	Neuronal (cochlear, central)

Methodology:
The collection and use of human materials for the present study were approved by the Ethics Committee on Human Subjects of each institute, and informed consent was obtained from parents of all patients. We enrolled 338 children (198 boys and 140 girls) with severe to profound SNHL who were referred to Department of ENT & Head-Neck Surgery, B. J. Medical College, Ahmedabad. The minimum and maximum age of the patient was 10 months and 14 years respectively. As a negative control 523 children with bilateral normal hearing were enrolled.

Hearing levels of the patients were measured by objective audiological tests: ABR and auditory steady-state response using IHS. The patients also underwent at least 1 of the following subjective tests: play audiometry, conditioned orientation reflex audiometry, and/or pure-tone audiometry. These tests were performed by audiological experts and were repeated at least twice to confirm measurements. Hearing levels of the patients were classified into 5 categories on the basis of the severity of the worst ear: profound (>90 dB), severe (71–90 dB), moderate to severe (56–70 dB), moderate (41–55 dB), and mild (20–40 dB). All 338 children were having severe and/or profoung hearing loss in both the ears(>70 dB) and underwent either unilateral or bilateral Cochlear Implantation later on. Detection of IgM & IgG antibodies of TORCH infection from blood of test & control group was done by ELISA(Enzyme-linked Immunosorbent assay). In all 338 test subjects TORCH screening was done preoperatively.

Results
Amongst all 338 test cases of SNHL analyzed, CT imaging of temporal bones identified 12 cases of Mondini malformation and 23 cases of malformation involving cochlea which includes common cavity around 8th nerve, Posteriorly rotated cochlea, abnormal superior cerebellar artery around 7th & 8th nerve complex, Dysplastic basal turn, fusion of superior and inferior vestibular nerve, dilated vestibular aquiduct, labyrinthine ossification, hypoplastic or dysplastic 8th nerve and hypoplastic semi circular canals. 8 cases had history of neonatal jaundice, 3 cases had history of bacterial meningitis,10 cases had history of difficult labour, 5 cases of congenital Rubella syndrome & 1 case was of Wardenburg syndrome. Rest 281 cases had no significant past history. Out of 338 cases 33 cases had one or more cardiac abnormality like PDA, Tetrology of Fellot's, DORV & out of them 9 cases had history of cataract. None patient was having active symptomatic infection or other significant medical condition during the time of study.

Of the 338 patients, 230(68.04%) positive for cytomegalovirus CMV IgG antibody, 146(43.2%) were positive for Rubella virus IgG antibody, and 14(4.14%) were positive for herpes simplex virus IgG antibody, 10(3%) were positive for toxoplasma virus IgG antibody and 9(2.67%) were positive for TB IgG antibody (Table 4). Out of 523 control subjects 112(21.41%) positive for CMV IgG antibody, 38(7.26%) were positive for Rubella virus IgG antibody, and 19(3.63%) were positive for herpes simplex virus IgG antibody, 3(0.6%) were positive for toxoplasma virus IgG antibody (Table 5). Though we have not taken any symptomatic cases in the study, out of 338 cases 7(2.07%) positive for cytomegalovirus CMV IgM antibody, 4(1.18%) were positive for Rubella virus IgM antibody, and 2(0.6%) were positive for herpes simplex virus IgM antibody, 2(0.6%) were positive for toxoplasma virus IgM antibody and 2(0.6%) were positive for TB IgM antibody (Table 6). Out of 523 control subjects 15(2.86%) positive for CMV IgM antibody, 8(1.91%) were positive for Rubella virus IgM antibody, and 7(1.33%) were positive for herpes simplex virus IgM antibody, 1(0.2%) were positive for toxoplasma virus IgM antibody (Table 7).

Table 4 – TORCH IgG titre of Cases

TORCH	Total	Positive	%
CMV	338	230	68.04
Rubella	338	146	43.2
HSV	338	14	4.14
Toxo	338	10	3

Table 5–TORCH IgG titre of Control

TORCH	Total	Positive	%
CMV	523	112	21.41
Rubella	523	38	7.26
HSV	523	19	3.63
Toxo	523	3	0.57

Table 6 – TORCH IgM titre of Cases

TORCH	Total	Positive	%
CMV	338	7	2.07
Rubella	338	4	1.18
HSV	338	2	0.6
Toxo	338	2	0.6

Table 7–TORCH IgM titre of Control

TORCH	Total	Positive	%
CMV	523	15	2.86
Rubella	523	8	1.91
HSV	523	7	1.33
Toxo	523	1	0.2

Discussion
In the present retrospective study of the etiology of SNHL, congenital CMV infection was associated with a substantial number of severe cases of SNHL. During the comparison of both group, data states that prevalence of IgG antibody of CMV is 3 times of that with control group. These high IgG titer signifies history of past infection. However TORCH screening was performed only single time on each cases, so we cannot determine the actual time of infection varying from birth to our notice. There are chances that a case may have been infected later in life after development of SNHL or there may be maternal IgG antibodies during infancy. Apart from all limitations, difference of titer between two group was significant which clearly states that role of TORCH infection in etiology of deafness cannot be denied. Majority of cases would have been infected either intrauterine or during immediate post natal period which leads to severe to profound SNHL. TORCH induced deafness usually occurs before the age of 2 years & in 5 of 10 cases, CMV-related SNHL was not detected before 6 months of age. In this study prevalence of Toxoplasma & Herpes does not differs significantly between two groups. That is mainly regional difference of endemicity of an infection.

In India, Rubella vaccination is not included in Universal Immunization Programme (UIP) which is followed in most of the country. It is only included in Indian Academy of Pediatrician (IAP) immunization schedule as a part of MMR vaccination at 9th and 15th month. So high prevalence of Rubella IgG titer may actually due to infection rather than vaccination programme and study also includes 5 cases of confirmed congenital rubella syndrome. The main conclusion behind the low incidence of children with rubella- induced hearing impairment may be linked to appropriate immunization of mothers to the rubella infection during their child bearing years.[5]

Shet *et al*. noted that infections acquired in utero or in the immediate post-natal period play a prominent role in perinatal and childhood morbidity, and that TORCH screening is popular among perinatologists and pediatricians even though its limitations are becoming increasingly clear.[6] A more targeted approach to test for congenital infection seems to be necessary. Early diagnosis of neonatal infections is important, along with an effective therapy for such an infection. In a phase II trial, Whitley *et al*. reported hearing improvement or stabilization in 16% of 30 infants with symptomatic congenital CMV infection following ganciclovir therapy.[7]

An estimate that congenital CMV infection may account for 10%–60% of cases of SNHL[8]. Barbi et al. reported that dried blood spots were positive for 9 (10%) of 87 infants with SNHL who had hearing loss at 140 dB. This prevalence may have been an underestimate, because (1) the relatively small amount of DNA that was extracted from the blood spots and (2) testing was conducted only for children whose SNHL was diagnosed at age <2 months.[9]

Like CMV, HHV-6 belongs to the betaherpesvirus subfamily, and the viruses have a similar genomic structure. A recent study demonstrated asymptomatic congenital infection with HHV-6 but not HHV-7 at a frequency of 1%.[10] In addition, it is well known that HHV-6 is neurotropic and that it causes neurological diseases.[11] Therefore, it would be interesting to determine whether HHV-6 causes SNHL, as CMV does.

Conclusion
This retrospective analysis of the etiology of SNHL directly demonstrates that congenital TORCH infection & particularly CMV and Rubella are responsible for a substantial proportion of early-childhood SNHL and that almost half of the infants at risk for the

development of late-onset CMV associated SNHL. Development of vaccination against CMV will be boon in future. Rubella vaccination or MMR should be included in UIP and every woman should be immunized before or during their child bearing age. Though TORCH screening is not useful for management of deaf child, if the TORCH screening was performed early then that could be better prevention & management of childhood deafness.

References

1. Nahmias AJ, Josey WE, Naib ZM, Freeman MG, Fernandez RJ, Wheeler JH. Perinatal risk associated with maternal herpes simplex infection. Am J Obstet Gynecol 1971;110:825-37.
2. Richard J H Smith, James F Bale Jr, Karl R White. Sensorineural hearing loss in children. Lancet 2005; 365: 879–90.
3. Brandon E. Cohen, Anne Durstenfeld and Pamela C. Roehm. Viral Causes of Hearing Loss: A Review for Hearing Health Professionals. Trends in Hearing 2014, Vol. 18: 1–17
4. Boppana SB, Fowler KB, Vaid Y, et al. Neuroradiographic findings in the newborn period and long-term outcome in children with symptomatic congenital cytomegalovirus infection. Pediatrics 1997; 99:409–14.
5. Reddy MVV, Bindu HemaL, Reddy PP, Rani UshaP Role of intrauterine Rubella infection in the causation of congenital deafness. Indian Journal of Human Genetics, Vol. 12, No. 3, September-December, 2006, pp. 140-143
6. Shet A. Congenital and perinatal Infections: Throwing new light with an old TORCH. Indian J Pediatr 2011;78:88-95.
7. Whitley RJ, Cloud G, Gruber W, Storch GA, Demmler GJ, Jacobs RF, et al. Ganciclovir treatment of symptomatic congenital cytomegalovirus infection: Results of a phase II study. J Infect Dis 1997;175:1080-6
8. Pass RF. Congenital cytomegalovirus infection and hearing loss. Herpes 2005; 12:50–5.
9. Barbi M, Binda S, Caroppo S, Ambrosetti U, Corbetta C, Sergi P. A wider role for congenital cytomegalovirus infection in sensorineural hearing loss. Pediatr Infect Dis J 2003; 22:39–42.
10. Hall CB, Caserta MT, Schnabel KC, et al. Congenital infections with human herpesvirus 6 (HHV6) and human herpesvirus 7 (HHV7). J Pediatr 2004; 145:472–7.
11. Yamanishi K. Human herpesvirus 6 and human herpesvirus 7. In: Knipe DM, Howley PM, eds. Fields virology. 4th ed. Philadelphia: Lippincott Williams & Wilkins, 2001:2785–801.