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Original Article

Viral infections in sudden hearing loss. Do we have enough evidence?

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Abstract
Objective: The aetiology of sudden deafness remains unknown even though some evidences suggest that it could be viral in origin. This study aimed to find out the relationship between viral infections and sudden sensorineural hearing loss. **Methods**: 32 patients presenting with sudden deafness and 10 healthy controls were included in the study. IgM antibodies to varicella zoster virus, measles, cytomegalovirus and herpes simplex virus were detected using micro ELISA. **Results:** Overall, 7(21.8%) patients showed seropositivity to one or more viruses. Virus specific IgM antibodies against measles and varicella zoster could be demonstrated in 4 (12.5%) and 3 (9.4%) patients respectively. None of the samples were found to be positive for herpes simplex virus (HSV) and human cytomegalovirus (HCMV) specific IgM antibodies. Controls were negative for all the viruses tested. The difference in seropositivity between the patient and control group was not statistically significant ($p>0.05$). **Conclusion:** Thus, this study suggests that sudden deafness is not commonly associated with a systemic viral infection.

Key Words: Sudden deafness, viruses, serology.

The sudden loss of hearing is a frightening experience for the patient that can result in definitive loss of an organ essential for communication. In fact, it is one of the few true emergencies in otology. Sudden hearing loss (SHL) generally refers to hearing loss of sensorineural origin and has been defined as the loss of more than 30 dB of hearing in at least three contiguous frequencies over the course of less than 3 days in an otherwise healthy person [1]. Worldwide an estimate of 15,000 cases of sudden hearing loss (SHL) per year is being reported. One in every 10,000 to 15,000 people suffer from this condition, highest incidence being in the 5th to 6th decade of life [2]. The aetiopathogenesis of sudden hearing loss has remained as an enigma thereby posing a challenge for its diagnosis. No definite cause has been identified in most of the cases. Based upon clinical and histopathologic findings, either viral or vascular role has been attributed for idiopathic sensory neural hearing loss (SNHL) in about 10% of cases only [3].Wilson *et al* [4] have postulated viral cochleitis as the most common cause. This was based upon the observation of upper respiratory infection preceding hearing loss [5]. Few cases seroconverted against some viruses during the course of illness [1,4]. Magnetic resonance imaging (MRI) studies of inner ear have shown the evidence of viral labyrinthitis [6,7]. Post mortem cochlear histopathology of SNHL patients has shown pathological changes resembling those encountered in viral labyrinthitis [8,9]. Viral infections implicated in acute hearing loss include herpes simplex virus (HSV)[10], measles, mumps, rubella, influenza, varicella zoster (VZV) and cytomegalovirus (CMV)[4,11,12].

All patients with sudden SNHL require thorough evaluation to identify and treat the underlying disorder that could play a predisposing role. Delay in detection of impaired hearing may affect the development of skills in speech, language and social interaction.

Considering the above facts, the present study was planned to address the role of common viral infections in patients with sudden sensorineural hearing loss.

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Methodology

Patients: Thirty two patients attending Otorhinolaryngology (ENT) clinics at Nehru Hospital, Post Graduate Institute of Medical Education and Research, Chandigarh, India between July, 2001 and December, 2003 with suspected acute hearing loss were enrolled in the study after taking their informed consent. Patients participating in the study met the following inclusion criteria: a) cochlear hearing loss of unknown aetiology b) hearing loss of at least 30 dB in three contiguous frequencies over the course of <3 days. Patients suffering from medical conditions like diabetes mellitus and hypertension were excluded from the study.

Controls: Ten age and sex matched apparently healthy individuals were included as the control group.

Serology: 2 ml venous blood samples were collected aseptically from all the subjects at the onset of their symptoms (during their first hospital visit). Subsequent follow up blood samples were obtained only from 12 patients within a interval of 1-2 weeks. Sera were separated and stored at -20°C till tested. Specific IgM antibodies were detected using micro ELISA system against herpes simplex virus(HSV) (EIA gen, HSV, IgM, Italy), varicella zoster virus (VZV) (Nova Tec immunodiagnostica, GmbH, Germany), measles (Vir ELISA, Viro-Immun Diagnostica, GmbH, Germany) and cytomegalovirus (CMV)(EIA gen, CMV IgM, Adaltis Italia, Italy). All the tests were performed according to the manufacturer's instructions.

Statistical Analysis

Statistical analysis was done using the Fischer's exact test and Chi square test.

Results

The patients ranged between 19-45 years and the male: female ratio was 5.6:1. A total of 7(21.8%) patients showed the presence of antibodies to one or more viruses. IgM antibodies against measles and varicella zoster virus could be demonstrated in 4 (12.5%) and 3 (9.4%) patients respectively. None of the samples were found to be positive for IgM antibodies to HSV and HCMV. In all the 12 follow up samples, the IgM status for the viruses tested was found to be the same as in their respective first samples. The hearing loss in seropositive patients was unilateral in 3 and bilateral in 4 patients. One patient had the history of upper respiratory infection 5 days prior to the onset of hearing loss. The controls were negative for all the parameters tested. The difference in positivity between the patient and the control group was not statistically significant ($p>0.05$).

Table 1: Comparison of results of present study with published series in patients with sudden hearing loss (SHL)

Studies	Viral seroconversion(%)	Association with SHL
Maasab et al, 1973	62%	Yes (adenovirus)
Jaffe, 1978	61.7%	Yes (adenovirus)
Mercke et al, 1980	6%	Yes (adenovirus)
Veltri et al, 1981	65%	Yes (Influenza B, measles)
Wilson et al, 1983	63%	Yes (mumps, measles, VZV, CMV, Influenza B)
Schulz et al, 1998	100%	Yes (HSV-1)
Pitkaranta et al, 1998	0%	No
Gagnebin et al, 2000	0%	No
Garcia et al, 2000	12.5%	No
Present study	18.75%	No

SHL: Sudden Hearing Loss

Discussion

There is widespread controversy concerning the role of viruses in the onset of SNHL. Several studies using conventional viral diagnostic tests have refuted the belief that viral infection is a common cause of sudden deafness [12,13,14] while others have shown definite association [1,15,16,17,18,19] (Table I). To establish the viral aetiology of sudden hearing loss, ideally identification of viruses in inner ear fluids as well as confirmation of viral cytopathic effect in the cochlear region is required. Serological evidence cannot prove the viral cause of inner ear damage and similarly isolation of viruses from tissues and nasopharynx

does not necessarily imply the viral aetiology of inner ear disease since viruses like herpes simplex virus and adenovirus may remain latent and are intermittently shed in oropharynx. Identification of viral antigens in perilymph involves invasive procedure and is associated with risk of damage to the inner ear hence difficult to establish as a routine diagnostic test. Thus, immunological and serological tests continue to provide presumptive diagnosis in patients with sudden hearing loss.

In the present study, VZV and measles IgM was detected in 4 (12.5%) and 3 (9.4%) patients respectively. Seroconversion against VZV has been observed in 5% and against measles in 15% of SNHL cases [1]. Veltri *et al* [18] observed significant seroconversion to measles in 16% of 77 paired serum samples of sudden hearing loss patients.

Role of herpes simplex virus as an aetiology of SNHL is controversial. IgM immune response against HSV 1 and 2 has been demonstrated in paediatric population with hearing loss [10]. HSV DNA in temporal ganglia has been detected in patients with SNHL [19]. However in some of the studies immune response against HSV could not be demonstrated [14] and so also the response to acyclovir [20].

CMV is thought to be the most common infectious cause of congenital deafness. Bilateral sensory neural hearing loss of patients has correlated with elevated antibody titres against cytomegalovirus in serum of children as well as in adults [21]. Wilson *et al* [1] and Veltri *et al* [18] have shown the presence of CMV in 7% of SNHL patients.

The results of this study suggest that sudden deafness is not commonly associated with a systemic viral infection. However, the possibility of local viral involvement cannot be entirely ruled out. Hence, further studies need to be carried out to demonstrate the presence of viral antigen or nucleic acid in inner ear fluids or tissues to prove the causal role of viruses in this group of patients.

Conclusion

Previous studies have suggested that viruses have a role in causation of sudden hearing loss. In this study, viral antibody detection was evaluated in 32 patients with sudden deafness. Overall, 21.8% of patients showed seroconversion to measles and varicella zoster virus. No significant difference was observed in positivity between the patient and the control group ($p>0.05$). Thus, the authors conclude that sudden deafness is not commonly associated with a systemic viral infection.

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