



Abrupt Sensorineural Hearing Loss in Children: Clinical Characteristics, Etiology, Treatment Outcomes, and Prognostic Factors

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Introduction

To investigate the clinical characteristics, etiology, treatment outcomes, and prognostic factors of sudden sensorineural hearing loss (SSNHL) in children to guide the clinical diagnosis and treatment of SSNHL in the pediatric population. Children with SSNHL have a lower rate of viral infection in comparison to adults with SSNHL. Fasting blood glucose levels complement C3, C4, and fibrinogen may be closely related to childhood SSNHL. The recovery rate in children with SSNHL is comparable to that in adults, but children have a higher complete rate compared to adults. A profound hearing curve is an unfavorable prognostic factor in both children and adults with SSNHL. Sudden sensorineural hearing loss (SSNHL) is a unilateral or bilateral sensorineural hearing loss with at least 30 dB decreases in threshold in three contiguous test frequencies occurring over 72 hours or less. In the United States, the incidence of SSNHL has been reported to be 27 per 100,000 per year. The age of SSNHL mainly occurred in 25–60-year-old patients, of whom 46–49 years old was the most common. Studies of SSNHL in children are rare in the literature. There were 6.6% of patients with SSNHL fewer than 18 years of age. Due to the low incidence of SSNHL in children, its clinical characteristics, etiology, treatment outcomes, and prognosis have been most likely deemed to be less relevant. At present, the etiology of childhood SSNHL still remains unclear. It has been reported in the literature that childhood SSNHL is mostly idiopathic and may be related to viral infections (cytomegalovirus, herpes simplex virus type 1 and type 2, rubella virus, etc.), congenital causes (large vestibular aqueduct syndrome, Mondini deformity), trauma, tumors (acoustic neuroma, multiple myeloma, etc.), autoimmune diseases, systemic immune diseases (SLE, Cogan's syndrome, Wegener's granulomatosis, etc.), ototoxic drugs, vascular diseases, metabolic disease, Meniere's disease, central deafness, etc.

Currently, there are no specific tests or diagnostic criteria for children with SSNHL caused by viral infections, autoimmune diseases, and systemic diseases, vascular diseases and metabolic diseases. In the present study, a series of laboratory tests were

performed on pediatric patients with SSNHL in an attempt to find out the possible causes of hearing loss. In this retrospective study, 25 children (26 ears) with SSNHL treated and hospitalized from November 2011 to December 2017 were selected as study subjects. All subjects met the following inclusion criteria: unilateral or bilateral sensorineural hearing loss with a threshold decrease of at least 30 dB at three consecutive test frequencies in 72 hours or less; age < 18 years; specialist otolaryngology examination, laboratory tests, and imaging studies to exclude acute or chronic otitis media; no previous history of surgery for otitis media; no diabetes or hypertension. At the same time, 149 cases (159 ears) of adult SSNHL (age ≥ 18 years) diagnosed and hospitalized in the same period were used for comparative analysis. Clinical data collected included age, gender, side (unilateral/bilateral, left/right ear), timing of treatment initiation, Pure Tone Audiometry (PTA) at admission and discharge, tinnitus, vertigo, ear fullness, laboratory tests for viral infection (CMV, RV, HSV-1, and HSV-2, etc.), autoimmune (C3, C4) and Antinuclear Antibodies (ANA), erythrocyte sedimentation rate (ESR), coagulation profile (FIB, APTT), blood viscosity, fasting glucose, absolute neutrophil and lymphocyte ratio (NLR), and imaging studies including middle ear CT and/or internal auditory canal MRI. This study was approved by the institutional review board of the hospital ethic committee. All patients received a 14 day course of the following conservative treatment: corticosteroids (methylprednisolone 1 mg/kg/d, gradually tapered every 4 days), vasoactive drugs (ginkgo biloba extract, alprostadil) and anticoagulant thrombolytic drugs (fibrinolytic enzyme) were given by intravenous drop infusion. In addition, all patients also received intramuscularly neurotrophic drugs (methyl cobalt ammonium, mouse nerve growth factor) and hyperbaric oxygen therapy.

Viral Serology Examination in Children

Reports on SSNHL in the pediatric population are scarce in the literature. This may not only be related to the low incidence of sudden deafness in this population group but also to poor language expression in children. Currently, no guidelines for the diagnosis and treatment of children with SSNHL exist, and so far diagnosis and treatment protocols of childhood SSNHL have been extrapolated from guidelines for the management of sudden hearing loss in adults. The etiology and mechanism of sudden deafness are not completely clear yet. Ječmenica and Bajec-Opančina reported that in children SSNHL is mostly idiopathic and may also be related to viral infections, anatomical malformations, tumors, autoimmune diseases, and systemic diseases. In our study, out of 25 cases of children with SSNHL, only one had the history of upper respiratory tract infection before the onset of deafness while in the remaining 24 cases no known cause could be identified from their past medical history. Eleven children underwent viral serology examination and nine were found to be positive for CMV, RV, and HSV-1 and HSV-2 infection; the infection rate was 81.8%. In the 149 adult patients, two cases had a prior upper respiratory tract infection and two cases suffered from parotitis. Fifty adults received viral serology examination, all of whom showed previous or new viral infection. The infection rate was higher in adults than in the children group. These findings suggest that the disease in both children and adults may be associated with viral infection. This is inconsistent with the high rate of viral infection in children with sudden deafness reported by Hou and Wang. The possible causes for this outcome are: fewer cases of pediatric patients; the viral infection rates calculated in this study were obtained by viral serology rather than by past medical history.

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