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High Prevalence of Hearing Impairment in HIV-Infected Peruvian Children

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Abstract

Objectives—To measure the prevalence and to identify risk factors of hearing impairment in human immunodeficiency virus-infected children living in Peru.

Study design—Cross-sectional observational study.

Setting—Two public hospitals and 1 nonprofit center in Lima, Peru, between August 2009 and April 2010.

Subjects—A total of 139 HIV-infected children, ages 4 to 19 years.

Methods—Hearing impairment and otologic health were assessed with pure tone audiometry, tympanometry, and otoscopy. The primary outcome was hearing loss, defined as average threshold >25dB for 0.5, 1, 2, and 4 kHz, in one or both ears. Historical and socioeconomic information was

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obtained through parental survey and medical chart review. Statistical analysis included univariate analysis and multivariate logistic regression.

Results—Fifty-four (38.8%) of 139 children had hearing impairment. On multivariate analysis, risk factors included: tympanic membrane perforation (odds ratio [OR] 7.08; 95% confidence interval [CI], 1.65-30.5; $P = .01$), abnormal tympanometry (OR 2.71; 95% CI, 1.09-6.75; $P = .03$), cerebral infection (OR 11.6; 95% CI, 1.06-126; $P = .05$), seizures (OR 5.20; 95% CI, 1.21-22.4; $P = .03$), and CD4 cell count <500 cells/mm^3 (OR 3.53; 95% CI, 1.18-10.5; $P = .02$).

Conclusions—The prevalence of hearing impairment in HIV-infected children in Lima, Peru was 38.8%. Middle ear disease, prior cerebral infection, and low CD4 cell count were significantly associated with hearing impairment. The high prevalence of hearing impairment emphasizes the need for periodic hearing assessment in the routine clinical care of HIV-infected children.

Keywords

HIV; hearing impairment; Peru

The use of highly active antiretroviral therapy (HAART) has prolonged the life expectancy of human immunodeficiency virus (HIV)-infected children and expanded the spectrum of acute and chronic conditions that may develop during their life span. An increased incidence of chronic conditions such as hearing impairment (HI), as a direct result either of HIV infection or of HIV treatment, may produce life-long behavioral, educational, and socioeconomic consequences.

HIV infection has been associated with HI in both adults and children.[1,2] Although potentially associated infections, especially otitis media,[3-5] are more prevalent in HIV-infected children, no association with HI has been reported. In adults, studies examining the association between the use of HAART and HI have been inconclusive.[6-8] Other studies have suggested a correlation between disease progression (declining CD4 cell counts) and HI identified by pure tone audiometry.[1] Similar correlations have not been reported in pediatric populations.

The prevalence of HIV infection in Peru is estimated to be less than 1% in adults and children, with the majority of cases clustered around urban centers such as Lima. Domestic and international efforts have aimed to keep HIV prevalence low, with Peru being the number one recipient of funds in South America from the Global Fund for AIDS, Tuberculosis and Malaria.[9] As the majority of children contract the virus perinatally,[10] mother-to-child transmission has been a primary focus of preventive measures.

In Peru, there is no universal hearing screening of adults or children, and HI is infrequently identified in children. A previous study by our group detected HI in 6.9% of HIV-uninfected schoolchildren in Lima[11]. The objective of this study was to determine the prevalence and associated risk factors of HI in HIV-infected Peruvian children. Early identification of such conditions could permit appropriate intervention and minimize developmental delay or impairment.

Methods

This cross-sectional observational study included HIV-infected children between the ages of 4 and 19 years recruited at 3 sites in Lima, Peru. The sites were the pediatric HIV services of 2 public hospitals, Instituto Nacional de Salud del Niño (INSN) and Hospital Nacional Arzobispo Loayza (Loayza), and the Hogar San Camilo, a nonprofit center providing social services for HIV-infected children. Both hospitals are large referral hospitals in Lima: the

INSN is a tertiary pediatric hospital, and Loayza primarily serves an adult population with a small pediatric service. Parents or guardians were invited to enroll their children when they presented for outpatient care at one of the hospitals or during activities at San Camilo. The only exclusion criterion was the child's inability or refusal to cooperate with 1 or more components of the study. Informed consent was obtained from each guardian or subject older than 17 years; assent was obtained from each child 17 years old or younger. Human subjects approval was obtained from the institutional review boards of the University of Washington School of Medicine, Stanford University School of Medicine, Instituto Nacional de Salud del Niño, Hospital Nacional Arzobispo Loayza, and Hogar San Camilo.

Audiometry and Tympanometry

All studies were conducted in a closed room at each site to minimize ambient noise. Hearing was assessed through pure tone audiometry using the Interacoustics AT220 audiometer at .5, 1, 2, and 4 kHz. Patients with an average of >25dB threshold on air conduction audiometry also underwent bone conduction testing.

HI was defined according to the World Health Organization (WHO) parameters as an average threshold pure tone average (PTA) >25dB on audiometry. In cases where bone conduction testing was performed, the air-bone gap was calculated as the air conduction PTA minus the bone conduction PTA. Conductive HI was defined as air conduction PTA >25dB with normal bone conduction and an air-bone gap of 10dB or more. Sensorineural hearing impairment was identified as air conduction PTA >25dB, bone conduction PTA >25dB, and an air-bone gap less than 10dB. If impairment in both air and bone conduction was found and the air-bone gap was >10dB, the patient was given the diagnosis of mixed HI. Patients with HI in one or both ears were considered hearing impaired.

Tympanometry was used to assess for middle ear disease, with abnormal classified as B- and C-type curves. Peak pressures, compliance, and canal volume were recorded. Otoscopy with a Welch Allyn 3.5V Macroview otoscope (Welch Allyn Inc, Skaneateles Falls, New York) identified clinical conditions such as otitis media, cerumen impaction, and tympanic membrane (TM) perforation. Cerumen impaction was defined as an obstructed view on otoscopy in addition to a B-type curve on tympanometry. In patients with cerumen impaction, the affected ear(s) were irrigated for disimpaction prior to audiometry.

Chart Reviews, Questionnaires, and Statistics

Medical, family, and social histories were abstracted from medical charts; and a questionnaire regarding medical history and symptoms of HI or ear disease was administered to the participant's guardian. Malnutrition was identified through documentation in the medical records or through biometrics, using WHO criteria for moderate malnutrition (weight for age or weight for height, less than 2 standard deviations below average). Data regarding age at seroconversion, CD4 cell count, HIV viral load, use of HAART, and history of infections was also abstracted. Univariate and Backward Wald multivariate statistical analyses were performed using SPSS Version 17.0 (SPSS, an IBM Company, Chicago, Illinois).

Follow-up

Patients with abnormalities identified on otologic examination (eg, chronic TM perforations, previously undiagnosed moderate or severe HI) were referred for further evaluation and treatment at the otolaryngology service at the hospital where they were receiving HIV care.

Results

One hundred thirty-nine children were enrolled: 112 from INSN, 16 from Loayza, and 11 from San Camilo. One patient declined enrollment. Results of audiometry, tympanometry, and otoscopic examination are presented in Table 1. Demographic information is presented in Table 2. The average age was 9.9 years; 72 (52%) were female. For some children, the medical and social histories were incomplete because of hospital transfers, incomplete documentation, and change of guardianship. Opportunistic infection status was recorded when present in the chart. Medical background from hospital records and questionnaires is presented in Table 3.

Fifty-four (38.8%) children had HI detected, the majority of which was conductive loss (48, 34.5%). One child had sensorineural hearing loss and 5 had mixed hearing loss (Table 1). Sixty-seven (48.2%) children had an abnormal tympanogram. Fifty-three (38.1%) had type B tympanograms indicating middle ear pathology and 18 (12.9%) had type C tympanograms suggesting eustachian tube dysfunction. On otoscopy, 19 (13.7%) children had a TM perforation, 24 (17.3%) had cerumen impaction, and 23 (16.5%) had TM inflammation. One-half of patients with cerumen impaction (12) had hearing impairment. Of these, six also had pathology (TM perforation or otitis media with effusion) seen in the opposite ear. All children with TM perforations had conductive hearing impairment on audiometry, and 33.3% of children with conductive hearing impairment had a TM perforation—compared with only 3.3% of patients without conductive hearing impairment ($P < .001$). Otitis media with effusion was detected in 7 children (5%).

Association with HIV Infection

HIV history is presented in Tables 4 and 5. The age at HIV seroconversion was recorded or calculated when available. Fifty-three (98%) of 54 children with HI were receiving HAART therapy (average duration 4.7 years) compared with 71 (84%) of 85 children without hearing impairment (average duration 4.2 years). Initial HAART treatment for children in this study typically included lamivudine, zidovudine, and nelfinavir. Children with CD4 cell counts declining to <250 underwent HIV genotyping and were typically switched to secondary treatment regimens. CD4 cell count levels were checked monthly and viral loads every 3 months. In children with hearing impairment, 16 (31%) had a most recent CD4 cell count of <500 cells/mm^3 and 17 (33%) children had a detectable viral load (>400 copies/mL) at the time of hearing assessment.

Risk Factors for Hearing Impairment

Clinical findings correlated with HI are displayed in Table 6. Otologic and neurologic risk factors correlated with HI included a history of TM perforation, abnormal tympanometry, seizures, and history of cerebral infections; these factors remained significant after adjustment for other variables. Specific HIV-related risk factors were also evaluated. Hearing impairment was correlated with use of HAART ($P = .01$), specifically protease inhibitors ($P = .03$), although this relationship was not significant in multivariate analysis. A CD4 cell count of <500 cells/mm^3 and undetectable viral load remained significant risk factors even after adjustment for other variables. Duration of HIV infection and duration of HAART use were inversely correlated with viral load ($P = .02$ and $P = .03$, respectively). When children were divided by CD4 cell count greater or less than 500 cells/mm^3, undetectable viral load remained a significant risk factor only for those children with CD4 cell count less than 500 cells/mm^3.

Discussion

This is the largest study to date of HI and ear disease in HIV-infected children. The prevalence of HI in HIV-infected Peruvian children (38.8%) was more than 5 times greater than the prevalence of hearing impairment (6.9%) in HIV-uninfected Peruvian children previously reported by our group[11]; this is also markedly higher than the 1% to 3% study in Angola detected hearing impairment in 26% of HIV-infected children by brainstem auditory evoked potentials or pure tone audiometry.[15] Ninety-two percent of these children had an otorhinolaryngological abnormality identified, including 27% with chronic otitis media; however, correlation between the presence of otologic pathologies and hearing impairment was not reported.

The majority (88.9%) of HI identified in our population was due to conductive loss. This finding was consistent with the results of the otoscopic examinations, which detected TM perforations in 13.7% and cerumen impaction in 17.3% of children. The strong association of TM perforation and abnormal tympanometry with hearing loss suggests middle ear pathology was a significant contributor to hearing impairment in HIV-infected children. The low prevalence of sensorineural and mixed hearing impairment suggests that hearing impairment was not predominantly because of ototoxicity associated with HAART usage.[12] The lack of correlation between HAART and hearing impairment is consistent with most recent studies of adults, which revealed no relationship between nucleoside reverse transcriptase inhibitors and hearing impairment.[10]

Compared with HIV-uninfected Peruvian children in our prior study,[11] HIV-infected children in this study had a higher prevalence of frequent ear infections (18.8% vs 11.3%). Frequent ear infections were a risk factor for hearing loss in univariate but not multivariate analysis. The association of HI in HIV-infected children with acquired ear disease, as opposed to congenital ear disease, is supported by a study from Nigeria, where universal newborn hearing screening of infants born to HIV-infected mothers did not detect an increased risk for sensorineural hearing loss.[16]

Middle ear pathology in HIV-infected children may produce HI through several mechanisms. Otitis media, especially when accompanied by effusion or damage to the TM, can produce conductive hearing loss.[17] HIV infection has been associated with recurrent otitis media, which may lead to more frequent TM perforations and impaired healing between infections. The association between hearing impairment and low CD4 cell count may be because of the accumulative damage to the TM and middle ear caused by recurrent episodes of otitis media. An undetectable viral load was unexpectedly a risk factor for hearing loss. One potential explanation is that lower viral load was a surrogate marker for longer duration of HIV infection. Univariate analysis detected a significant correlation between undetectable viral load and longer time since HIV seroconversion (odds ratio [OR] 2.67; 95% confidence interval [CI], 1.21-5.87; $P = .02$), suggesting that children with longer duration of HIV infection received effective antiretroviral therapy. Longer periods of HIV infection would thus lengthen time at risk for recurrent middle ear infections leading to hearing loss. Low viral load was also associated with longer time since initiation of HAART (OR 5.31; 95% CI, 1.147-24.586; $P = .03$). Although HAART itself was not a risk factor for hearing loss in multivariate analysis, longer duration of antiretroviral therapy suggests a longer exposure period to recurrent middle ear infections or a more aggressive process, including more severe or frequent middle ear infections.

The correlation between TM perforation and hearing loss supports the theory of middle ear disease as an important cause of hearing impairment in HIV-infected children. The prevalence of TM perforation in our HIV-infected children (13.7%) was much higher than in

our historical control group of HIV-uninfected Peruvian children (<4.5%).[11] In addition to the increased susceptibility of HIV-infected children to middle ear infections, low CD4 cell count may directly lead to chronic TM perforations. A study of immune cell response in a mouse model of artificially created TM perforations detected a strong lymphocyte-mediated response within the first week after perforation that was greater than the response observed during acute otitis media.[18] T-cell presence peaked at day 3 and B-cell presence at day 6—with B cells producing the most vigorous immune response. The authors hypothesized that CD4 helper T cells were first recruited to help activate B cells, which then carried out the majority of the healing response. Incomplete healing due to immunocompromise in our HIV-infected children may have led to chronic TM perforations with impaired healing and resultant conductive hearing impairment.

HIV infection may put children at higher risk for developing hearing impairment, likely due to a combination of recurrent ear infections and impaired healing between infections. The identification of middle ear disease as a risk factor for hearing impairment in HIV-infected children can help address long-term effects on linguistic, social, educational, and vocational development. Given the high prevalence of HI detected in this study, and the lack of universal hearing screening for HIV-infected Peruvian children, expanding the scope of evaluation and care of chronic HIV infection should include evaluation for HI and prompt treatment of recurrent otitis media.

Conclusion

Hearing impairment was identified in 38.8% of HIV-infected children in Lima, Peru. Risk factors included abnormal tympanometry, TM perforation, and low CD4 cell count, suggesting middle ear disease is a significant factor causing hearing impairment. The link between hearing loss and low HIV viral load may indicate that longer periods of immunocompromise contribute to increased susceptibility to middle ear disease or longer history of recurrent episodes of otitis media. Other risk factors include cerebral disease such as seizures and cerebral infection. The proper identification, prevention, and treatment of these conditions should be taken into account during the management of HIV-infected children in Peru and other countries.

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Table 1

Hearing Impairment and Ear Pathology on Examination

Examination	Unilateral (%)	Bilateral (%)
Hearing impairment		
>25dB pure tone average	26 (18.7)	28 (20.1)
Conductive	24 (17.3)	24 (17.3)
Sensorineural	0	1 (0.7)
Mixed	2 (1.4)	3 (2.2)
Mild	24 (17.3)	51 (36.7)
Moderate	6 (4.3)	3 (2.2)
Moderate-severe	5 (3.6)	1 (0.7)
Severe	0	2 (1.4)
Ear pathology		
Abnormal otoscopic examination	25 (18.0)	58 (41.7)
Abnormal tympanometry	22 (16.2)	41 (30.1)
Tympanic membrane perforation	14 (10.1)	5 (3.6)

Table 2

Patient Demographics

	Hearing Impairment (n = 54)		No Hearing Impairment (n = 85)		
Variable	**n**	**(%)**	**n**	**(%)**	***P* Value**
Female	31	(57)	41	(48)	.29
Age, y					
4-7.9	13	(24)	30	(35)	
8-11.9	21	(39)	38	(45)	
>12	20	(37)	17	(20)	
Lives in orphanage	11	(20)	25	(29)	.24
Birth city					
Greater Lima area	33	(72)	58	(78)	.72
Rural province	13	(28)	16	(22)	

Table 3

Medical and Otologic History

	Hearing Impairment (n = 54)		No Hearing Impairment (n = 85)		
Variable	**n**	**(%)**	**n**	**(%)**	***P* Value**
Birth and neonatal history					
Prematurity	7	(13)	9	(11)	.30
Jaundice	3	(6)	11	(17)	.15
Infections and other medical conditions					
Pneumonia	36	(67)	51	(61)	.55
Tuberculosis	19	(39)	25	(36)	.73
Cytomegalovirus	28	(70)	33	(62)	.44
Epstein-Barr virus	13	(33)	21	(40)	.54
Toxoplasmosis	4	(11)	3	(6)	.44
Syphilis	0	(0)	1	(2)	1.00
Cerebral Infections	6	(11)	1	(1)	.01
Varicella	22	(41)	24	(28)	.13
Convulsions	11	(21)	5	(6)	.01
History of hospitalization	48	(89)	66	(78)	.09
Malnutrition	36	(67)	46	(54)	.14
Otologic History					
≥3 ear infections in 1 year	15	(28)	11	(13)	.03
Otorrhea	29	(54)	17	(20)	.00
Antibiotic use	26	(49)	22	(27)	.01
Ear trauma	4	(8)	2	(2)	.21
Family member with auditory problem at <35 years of age	5	(10)	9	(11)	.78
Headphone use	26	(49)	29	(35)	.96

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Table 4

Human Immunodeficiency Virus (HIV) Clinical History

	Hearing Impairment (n = 54)		No Hearing Impairment (n = 85)		
Variable	**n**	**(%)**	**n**	**(%)**	***P* Value**
Mode of acquisition of HIV					.15
Vertical	53	(98)	78	(92)	
Nonvertical	1	(2)	7	(8)	
Years since HIV seroconversion					
<5	17	(39)	34	(47)	.44
≥5	27	(61)	38	(53)	
HAART therapy	53	(98)	71	(84)	.01
Average years on HAART	4.7		4.2		
<3yrs	15	(28)	21	(30)	.20
3-5.9	20	(37)	35	(50)	
≥6	18	(33)	14	(20)	
Nucleoside analog reverse transcriptase inhibitor	52	(98)	74	(88)	.07
Non-nucleoside analog reverse transcriptase inhibitor	19	(36)	36	(43)	.40
Protease inhibitor	44	(83)	55	(65)	.03

Abbreviation: HAART, highly active antiretroviral therapy.

Table 5

Human Immunodeficiency Virus (HIV) Laboratory History

	Hearing Impairment (n = 54)		No Hearing Impairment (n = 85)		
Variable	**n**	**(%)**	**n**	**(%)**	***P* Value**
HIV laboratory values CD4 (most recent)					.85
≤500 cells/mm^3	16	(31)	24	(29)	
>500	36	(69)	58	(71)	
CD4 (nadir)					.39
≤500	42	(81)	61	(74)	
>500	10	(19)	21	(26)	
Viral load (most recent)					
Undetectable (≤400 copies/mL)	35	(67)	39	(48)	.03
Detectable	17	(33)	42	(52)	
Viral load (peak)					
Undetectable	10	(19)	10	(12)	.28
Detectable	42	(81)	71	(88)	

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Table 6

Independent Predictors of Hearing Loss

	Unadjusted		Adjusted[a]	
Predictor	**OR**	**95% CI**	**OR**	**95% CI**
Cerebral infection	10.50	(1.23-89.8)	11.60	(1.06-126)
HAART	9.60	(1.21-75.4)		
Protease inhibitor	2.40	(1.06-5.44)		
Otorrhea	4.57	(2.15-9.72)		
Seizures	4.02	(1.31-12.4)	5.20	(1.21-22.4)
Antibiotic use	2.58	(1.25-5.35)		
≥3 ear infections a year	2.55	(1.07-6.09)		
CD4 count ≤500 cells/mm^3	1.07	(.504-2.29)	3.53	(1.18-10.5)
Undetectable viral load (≤400 copies/mL)	2.22	(1.07-4.58)	4.33	(1.58-11.9)
TM perforation	11.50	(3.16-41.9)	7.08	(1.65-30.5)
Abnormal tympanometry	3.85	(1.86-7.97)	2.71	(1.09-6.75)

Abbreviations: CI, confidence interval; HAART, highly active antiretroviral therapy; OR, odds ratio; TM, tympanic membrane.

[a] Adjusted for other variables in table.