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Main Article

Evaluation of hearing loss in patients with ankylosing spondylitis

A Eryilmaz, M Dagli, H Karabulut, F Sivas Acar*, E Erkol Inal*, C Gocer

Abstract

Aims: The aim of this study was to investigate hearing loss in patients with ankylosing spondylitis.

Study design: Prospective, case–control study.

Methods: Fifty-nine ankylosing spondylitis patients (118 ears) and 52 healthy control subjects (104 ears) were included. Pure tone audiometry at 250, 500, 1000, 2000, 4000 and 6000 Hz and immittance measures, including tympanometry and acoustic reflex tests, were performed in the patients and controls.

Results: Sensorineural hearing loss was found in 21 patients (35.5 per cent), bilateral in 15 patients and unilateral in six. Pure tone thresholds significantly differed between patients and controls at all frequencies ($p < 0.05$). There was no statistically significant difference between the right and the left ears' thresholds at all frequencies, except at 4000 Hz in ankylosing spondylitis patients. The right ears' thresholds were higher than those of the left ears. Patients' pure tone average (PTA) thresholds were significantly different from those of controls in all three PTA groups (i.e. 250 Hz; 500, 1000 and 2000 Hz; and 4000 and 6000 Hz) ($p < 0.05$). The differences were most prominent in the higher frequencies.

Conclusion: Our findings suggest a decreased hearing level in ankylosing spondylitis patients, mostly at high frequencies, although the pure tone thresholds of patients and controls significantly differed at all frequencies.

Key words: Ankylosing Spondylitis; Hearing Loss; Pure Tone Audiometry

Introduction

Ankylosing spondylitis is a chronic inflammatory disease of uncertain aetiology. Its pathogenesis is unclear, but the disease is strongly associated with human leukocyte antigen (HLA) B27.[1] The disease primarily affects the spine and sacroiliac joints and usually commences in the third decade of life. It affects men three times more often than women.[2] The diagnosis of ankylosing spondylitis is based on clinical features, with the clinical diagnosis usually supported by radiologic evidence of sacroiliitis.[1]

Investigation of hearing loss in chronic rheumatic inflammatory diseases such as rheumatoid arthritis have always attracted attention. Many studies and reports have been published on hearing loss in rheumatoid arthritis.

Although some studies have been published on hearing loss in ankylosing spondylitis, they have been limited. The first report of hearing loss in ankylosing spondylitis was a case report published by Magaro *et al.* in 1984.[3] These authors stated that the hearing loss in their patient was of the conductive type. However, controversially, subsequent clinical studies stated that hearing loss in patients with ankylosing spondylitis was of the sensorineural type.[4–6]

The aim of this study was to investigate hearing loss within an ankylosing spondylitis patient population.

Materials and method

Patients

A total of 59 ankylosing spondylitis patients (118 ears), diagnosed and treated at the physical medicine and rehabilitation clinic of the Numune Research and Training Hospital, Ankara, and 52 healthy control subjects (104 ears) were included in the study. The diagnosis of ankylosing spondylitis was made according to the modified New York diagnostic criteria.[7] Informed consent was obtained from all participants. Detailed information was obtained about possible aetiological factors leading to hearing loss (ototoxic drugs, noise exposure, ear surgery, perforated tympanic membrane, Ménière's disease, cranial trauma, metabolic diseases and systemic disease). None of the patients had a history of any of these factors.

From the Departments of Otolaryngology and *Physical Medicine and Rehabilitaion, Ankara Numune Research and Training Hospital, Ankara, Turkey.
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Participants were excluded from the study if they had any of following: (1) otoscopic evidence of a perforated tympanic membrane or other middle-ear pathology; (2) a flat tympanogram or absence of acoustic reflexes at 1 kHz with contralateral stimulation; or (3) an air–bone gap $\geq$5 dB at any frequency.

Occiput-to-wall distance, chest expansion, finger–floor distance, modified Schober and other laboratory parameters were measured in all ankylosing spondylitis patients.

Audiometry

The initial hearing examination included otoscopy, tympanography and a complete audiologic evaluation, including pure tone air and bone conduction audiometry and speech audiometry. Pure tone audiometry was performed at the frequencies 250, 500, 1000, 2000, 4000 and 6000 Hz using an AC-40 diagnostic audiometer in a sound-treated cabin (Interacoustic Acoustic Company, Denmark). Normal middle-ear function was defined by immittance and acoustic reflex results using an Interacoustic AZ 26 clinical impedence meter (Interacoustic company, Denmark). The patients and controls who had normal peak compliance, peak pressure, gradient, ear canal volume and acoustic reflexes obtained by immittance measures (as defined by the American Speech Language and Hearing Association)[8,9] were included in the study.

Statistical analyses were performed using the Statistical Package for the Social Sciences version 13.0 software for Windows. A p value of <0.05 was considered significant. For overall comparisons of the groups (i.e. ankylosing spondylitis patients and controls), independent samples t-tests were performed. Chi-square testing was used to compare the ages of patients and controls, and the Mann–Whitney U test was used to compare the genders of patients and controls. Pearson correlation analysis was used to investigate the relationship between quantative variables.

Results

The mean age of patients with ankylosing spondylitis was 35.2 years (range, 16–65). Ten patients were female and 49 were male. The mean age of the control group was 33.9 years (range, 20–60); 16 women and 36 men. There were no statistically significant differences between the ages and genders of the patient and control groups ($p > 0.05$). Otoscopic examination was normal in all participants.

The following data for the ankylosing spondylitis patients are shown in Table I: occiput-to-wall distance, chest expansion, finger–floor distance, modified Schober test, duration of disease and laboratory parameters. The patients were treated with either sulphasalazine or methotrexate, or both.

Because participants showed no air–bone gaps, only bone conduction thresholds were taken into consideration. The mean pure tone audiometry values for patient and controls are shown in Table II. Speech discrimination scores were within normal limits in all patients and controls. Normal peak compliance, peak pressure, gradient, ear canal volume and acoustic reflexes were obtained by immittance measures in all patients and controls. Sensorineural hearing loss was found in 21 patients (35.5 per cent), bilateral in 15 patients and unilateral in six.

The patients' and controls' pure tone thresholds significantly differed at all frequencies ($p < 0.05$) (Figure 1). There were no statistically significant differences between the right and the left ear thresholds at all frequencies, except at 4000 Hz in the ankylosing spondylitis patients. The right ear thresholds were higher than the left ear thresholds. Three pure tone average (PTA) groups were calculated: PTA one (250 Hz), PTA two (500, 1000 and 2000 Hz) and PTA three (4000 and 6000 Hz). The patients' and controls' PTA thresholds significantly differed in all three PTA groups ($p < 0.05$). The differences were most prominent at the higher frequencies (Figure 2).

No correlation was found between the patients' results for occiput-to-wall distance, chest expansion, finger–floor distance, modified Schober test, duration of disease or laboratory parameters and their pure tone audiometry findings.

TABLE I

PATIENTS' CLINICAL FEATURES

Parameter	Minimum	Maximum	Mean
ESR (mm/h)	3.00	95.00	35.48
CRP (mg/L)	2.00	147.00	29.68
Disease duration (years)	1.00	40.00	9.50
Chest expansion (cm)	0.00	8.00	3.25
Occiput-wall (cm)	0.00	30.00	5.75
Finger–floor distance (cm)	0.00	65.00	35.27
M Schober test (cm)	1.00	14.00	3.08

ESR = erythrocyte sedimentation rate; CRP = C reactive protein; M = modified

TABLE II

PURE TONE AUDIOMETRY FINDINGS (BONE CONDUCTION)

Frequency (Hz)	Right ear			Left ear		
	Range (dB)	Mean (dB)	SD	Range (dB)	Mean (dB)	SD
Patients						
250	0–70	15.84	11.63	0–55	14.49	10.24
500	0–55	12.03	10.26	0–50	12.28	9.61
1000	0–35	10.00	8.30	0–35	8.98	7.53
2000	−5–35	9.57	9.61	−5–35	9.23	8.34
4000	0–90	20.76	20.08	0–55	16.52	13.59
6000	0–85	21.61	19.72	0–85	22.20	16.95
Controls						
250	0–25	10.18	5.36	0–20	10.37	4.78
500	0–20	7.73	4.65	0–15	7.92	3.85
1000	−5–15	6.03	4.31	0–15	6.69	3.91
2000	−5–15	4.81	4.15	−5–15	5.47	4.52
4000	−5–20	6.50	4.95	0–20	8.30	6.19
6000	−5–25	9.33	6.28	−5–30	10.28	6.96

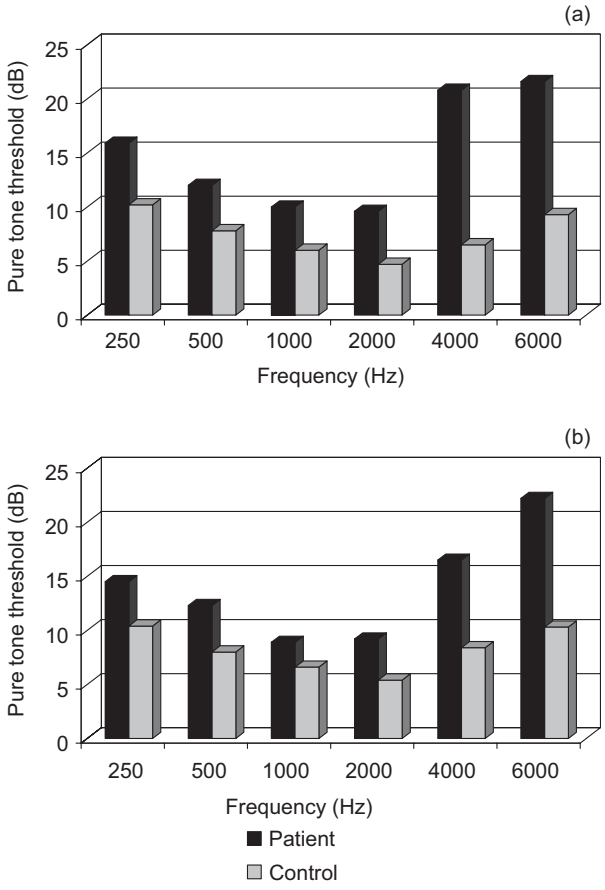

FIG. 1

Pure tone thresholds for patients and controls, showing significant differences ($p < 0.05$). Note that differences were greater at higher frequencies. (a) Right ear; (b) left ear.

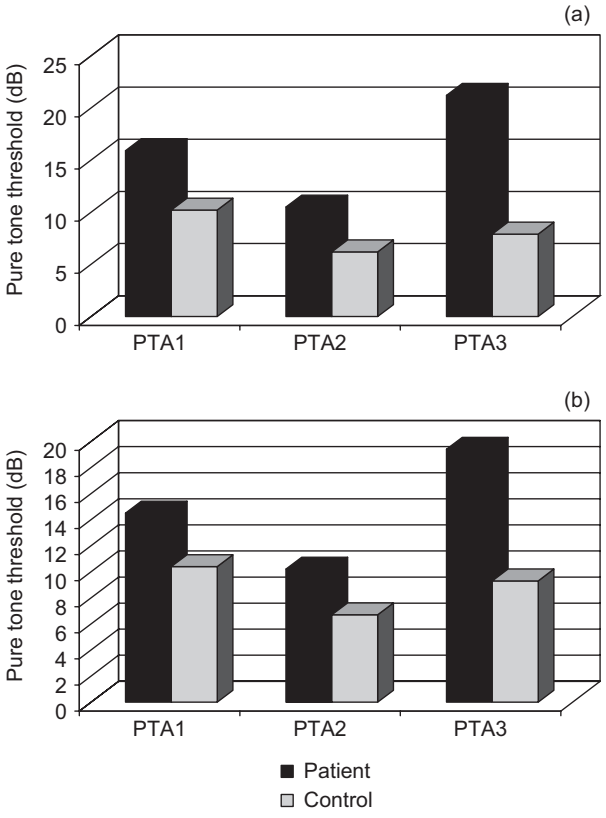

FIG. 2

Pure tone average thresholds for patients and controls at 250 Hz (PTA1), at 500, 1000 and 2000 Hz (PTA2), and at 4000 and 6000 Hz (PTA3). Note significant differences ($p < 0.05$). (a) Right ear; (b) left ear.

Discussion

In a review of the literature, we found only one clinical study related to hearing loss in patients with ankylosing spondylitis. In this study, audiometric examination and auditory brainstem response were used to assess hearing. Three case reports of hearing loss in ankylosing spondylitis patients have been published to date. This hearing loss was conductive in one report[3] and sensorineural in the other two.[4,5] In addition to these case reports, a clinical study was recently published in which the investigators found sensorineural hearing loss in 28.6 per cent of ankylosing spondylitis patients. The sensorineural hearing loss was bilateral in four patients and unilateral in four patients, and was particularly marked at the high frequencies. Auditory brainstem response testing showed no significant differences between patients and controls.[6] In our study, sensorineural hearing loss was found in 21 patients (35.5 per cent), bilateral in 15 patients and unilateral in six. This finding has been suggested by the results of previous studies.[4–6]

The exact mechanism of immune-mediated hearing loss and hearing loss in rheumatic diseases remains unclear, despite much investigation. However, some theories have been proposed to explain sensorineural hearing loss in rheumatic diseases. The autoimmune theory is the most commonly investigated of these theories. 'Immune inner-ear disease' is the term used to describe cochleovestibular pathology or dysfunction caused by the immune system. 'Autoimmune inner-ear disease', on the other hand, is a more specific entity that implies a direct immune attack against an endogenous tissue. Immune inner-ear disease encompasses those cases of cochleovestibular dysfunction related to other systemic immune-mediated illnesses, such as the collagen vascular diseases. In these cases, the inner ear may not be the primary target of an immune attack but is injured indirectly by immune complex deposition or other mechanisms.[10]

Ankylosing spondylitis is a chronic, systemic inflammatory disorder which is strongly associated with HLA B27.[1] Genes in the HLA complex are among the strongest predisposing genetic factors. The HLA complex genes primarily involved are most often those encoding the peptide-presenting HLA class one or two molecules. A probable mechanism is preferential presentation, by the disease-associated HLA molecules, of peptides from auto-antigens to T cells.[11] Autoimmune

diseases are the result of an interaction between predisposing genes and triggering environmental factors, leading to loss of self-tolerance and an immune-mediated destruction of autologous cells and tissues. Immune-mediated mechanisms in ankylosing spondylitis are suggested by inflammatory histology and raised serum levels of immunoglobulin A and acute phase reactants. In an immunohistologic study of sacroiliac biopsies, CD4+ and CD8+ T cells and macrophages were present in the inflamed sacroiliac joints.[1]

Sensorineural hearing loss can be caused by some autoimmune diseases, such as systemic lupus erythematosus, rheumatoid arthritis, autoimmune sensorineural hearing loss, relapsing polychondritis, disseminated vasculitis and polymyalgia rheumatica.[12]

Gussen described the temporal bone of a patient with polyarteritis nodosa as having perivascular infiltration of the labyrinthine artery, along with fibrosis, bone formation and hydropic changes in the cochlea.[13] In a study of immunoglobulin G antibodies against mesenchymal structures of the inner ear, the basement membrane of strial capillaries, the dark cell area, the spiral ligament and spiral lamina could be demonstrated.[14] Some animal experiments have documented the existence of immunopathological changes in the inner ear.[15–17]

Many of the autoimmune diseases can cause vasculitis, resulting in a variety of secondary degenerative changes. The most widely documented effects of autoimmune diseases resulting in sensorineural hearing loss are mediated by a vascular mechanism.[12]

In an animal experimental study, Lewis and Wistar rats were shown to develop sensorineural hearing loss with atrophy of the organ of Corti, spiral ganglion and vestibular degeneration, otospongiosis-like lesions in the tympanic annulus, and cochlear vasculitis. Both cellular and humoral responses to type two collagen were identified.[18]

- **The aim of this study was to investigate hearing loss in patients with ankylosing spondylitis**
- **Fifty-nine ankylosing spondylitis patients (118 ears) and 52 healthy control subjects (104 ears) were included in the study**
- **Hearing level decreased mostly at high frequencies in ankylosing spondylitis patients, although the pure tone thresholds of patient and controls differed significantly at all frequencies**

Several studies have demonstrated that the inner ear was the source of the antibody.[19–21] According to these studies, the inner ear is capable of responding to antigen challenge. Harris and coworkers have shown a parallel rise of antibody titres over a three-week period in guinea pigs immunised by either inner-ear or peritoneal routes of antigen presentation.[22] These studies indicate that the inner ear is an effective route of antigen processing, which can result in the acquisition of systemic humoral immunity as well as cellular immunity.

Conclusion

Our findings suggest that our ankylosing spondylitis patients' hearing loss was sensorineural and that their hearing level decreased mostly at high frequencies, although the pure tone thresholds of patient and controls differed significantly at all frequencies. However, further studies are needed to explain the cause and mechanism of hearing loss in ankylosing spondylitis patients.

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Address for correspondence:
Dr Muharrem Dagli,
Cemal Gursel Caddesi No 48/1,
Cebeci 06600,
Ankara, Turkey.

Fax: 903124358832
E-mail: mdagli@hotmail.com

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