
Low-Cholesterol Diet and Antilipid Therapy in Managing Tinnitus and Hearing Loss in Patients with Noise-Induced Hearing Loss and Hyperlipidemia

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Abstract: The aim of our study was to outline the prevalence of hyperlipidemia in patients who had high-frequency hearing loss and tinnitus due to noise exposure. We investigated the role of a low-cholesterol diet and antihyperlipidemic therapy to alleviate the severity of tinnitus and possibly promote hearing gain after therapy in patients with acoustic trauma. Forty-two hyperlipidemic patients with subjective tinnitus and hearing loss due to noise exposure were enrolled for the study. We placed patients on a low-cholesterol diet or antihyperlipidemic therapy and followed them for up to 24 months; then we designated two groups as either “unresponsive” (n = 22; no response to either of the therapies and still experiencing hyperlipidemia) or “responsive” (n = 20; lower cholesterol or triglyceride levels). We then compared tinnitus scores and hearing levels in the two groups. The difference between tinnitus scores in the unresponsive and responsive groups and the change in tinnitus scores before and after therapy in the responsive group were significant. When we compared self-rated tinnitus severity results in two groups after therapy, we found the difference was significant ($p < .05$). The difference between average air-conduction thresholds at high frequencies after the treatment in the two groups was also significant. The incidence of hyperlipidemia is high among patients with noise-induced hearing loss, and significant improvement by way of lowered tinnitus intensity and higher frequencies in average hearing thresholds can be achieved after lowering the serum lipid level.

Key Words: antilipid therapy; hyperlipidemia; low-cholesterol diet; noise-induced hearing loss; tinnitus

Cochlear hair cells and neural and central auditory pathways are involved in the generation of subjective tinnitus [1]. Analysis of the epidemiological data indicates that exposure to noise is widespread and is one of the most common causes of tinnitus, which leads to various somatic and psychological disorders and interferes with the quality of life [2]. It has been reported that high-plasma cholesterol and

triglyceride levels are one of the major risk factors for the development of occupational hearing loss [3–5].

Decrease in internal diameter of the vascular bed resulting from hyperlipidemia compromises the oxygen transportation capability of the blood to the cochlea, and chronic hypoxia is likely to occur by the reduction in oxygen supply of the inner ear, which in turn would lead to insufficient adjustment of the cochlear metabolism. Auditory perception in such cochleas is very sensitive to reduction of the oxygenation in the inner ear [6]. An atherosclerotic effect can reduce the enzymatic detoxifying capacity of the antioxidant enzymes (e.g., superoxide dismutase, catalase, glutathione reductase, peroxidase) and can increase peroxidative activity in tissue cells,

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resulting in generation of reactive oxygen species to initiate the peroxidative damage to the cell membrane.

This may result in the overproduction and accumulation of free radicals, which disturb the microcirculation of the cochlea and play an important role in ischemia-induced cochlear injury [7]. Platelet aggregation and blood viscosity due to high lipid levels will intensify inner-ear damage owing to slowing of microcirculation and will contribute to the development of both progressive and sudden hearing loss [8,9].

The reasons for the susceptibility to noise-induced hearing loss of patients who have been exposed to a noisy environment have been the subject of various studies. Gratton and Wright [10] demonstrated the accumulation of lipid in the stria vascularis and outer hair cells in chinchillas after experimentally induced hypercholesterolemia, which might intensify the detrimental effect of ototoxic drugs or noise. It seems that noise and other metabolic disturbances have a synergistic effect on the development of tinnitus and hearing loss [5].

Previous data indicate that the cochlea and the central auditory system age faster and earlier in patients with hyperlipidemia and that the risk of noise-induced hearing loss is much higher [5,11]. A correlation between hyperlipoproteinemia and early presbycusis has been reported [12]. Noise-induced destruction first takes place in the hair cells of the organ of Corti. Destruction occurs mostly in the basal turn of the cochlea and in the spiral ganglion. Neural atrophy and changes in the labyrinthine arteriole follow this damage. Additionally, swelling and separation of the hair cells have been confirmed [13]. It seems that the order of destruction and the high-frequency nature of the auditory dysfunction are correlated not only with the frequency and severity of the noise exposure but with the presence of the secondary pathology, as the formation of vasoactive lipid peroxidation products after intense noise is similar to the condition seen in metabolic damage to the organ of Corti [7,14–17]. In this manner, preventive measures may provide restorative support to the injured cochlea.

The aim of this study was to outline the prevalence of hyperlipidemia in patients who had high-frequency hearing loss and tinnitus due to chronic noise exposure and to evaluate the risk of developing sensory hearing loss in those patients as compared to risk in control subjects. We instituted a low-cholesterol diet and anti-hyperlipidemic therapy to investigate whether any benefit accrued in terms of hearing gain and tinnitus relief.

PATIENTS AND METHODS

We conducted this prospective study in 120 consecutive patients with complaints of chronic subjective tinnitus of varying degrees of severity and hearing loss

due to noise exposure. The project was approved by the local medical research ethical committee, and we obtained informed consent from each patient. A diagnosis of acoustic trauma was verified by an ear, nose, and throat physician according to a clinical history of chronic noise exposure in patients who sought medical help for complaints of tinnitus and high-frequency hearing loss. All patients who were required to complete the questionnaire and who were subjected to clinical examination were military personnel. We excluded on the basis of their response to the questionnaire or clinical examination subjects who had a history of familial hearing loss, head trauma, collagen disease, neurological or hormonal disturbance, addiction to alcohol, or ototoxic drug therapy. None of the patients had cerebrovascular disease, hypertension, electrolyte imbalance, cranial nerve deficit, or clinical signs of peripheral neuropathy.

The patients were examined otoscopically and were reviewed by an audiometric test battery including pure-tone audiometer (AC-30, IAC, Copenhagen, Denmark), stapes reflex, and tympanometry (Amplaid 775, Milan, Italy). Audiometric tests included air- and bone-conduction thresholds for pure tones of 125–8,000 Hz, tests of speech reception and speech discrimination, contralateral and ipsilateral reflex measurements, and tympanometry. Patients who had chronic otitis media or middle-ear effusion, those with an abnormal ear drum (perforation, retraction, etc.) or auditory canal, and those who had abnormal tympanometry (other than type A) were excluded.

We carried out tinnitus pitch match and loudness analysis in all patients. Measurement of loudness of the tinnitus was based on subjective assessment by the patients. The frequency of the tinnitus was estimated using a pitch-match frequency test. A pure-tone signal was presented continuously at 50-Hz intervals at between 10- and 20-dB levels. The test was repeated two times for each patient. Then, the pitch-match frequency threshold of the tinnitus was measured as the tinnitus loudness match of the pure-tone stimulus presented with a 1-dB increase of tinnitus frequency. Patients were required to complete a tinnitus handicap questionnaire in each interview. We required all patients to rate their tinnitus from 1 to 10, with 10 being loudest (Table 1). Each level was defined by short explanations to simplify the patients' grading of their tinnitus. The duration and the type of the tinnitus were noted for each patient.

We obtained from all patients a complete blood count; a glucose level from fasting blood samples; plasma cholesterol; high-density lipoprotein (HDL); low-density lipoprotein (LDL); very-low-density lipoprotein (VLDL); and triglyceride levels (Merc Vitalab Selectra Chemical Analyser, Merc diagnostic kits, California, USA). All tests were repeated twice. In the study, from 120

Table 1. Ten-Point Scale for Grading Degree of Tinnitus

1. Tinnitus is noticeable only in silence.
2. Tinnitus is not constant but audible in daily life.
3. Tinnitus is almost constant, but I do not pay much attention.
4. Tinnitus is constant and sometimes irritating.
5. Tinnitus is constant and usually disturbs me but does not affect my activity or sleep.
6. Tinnitus affects my daily communication, and sometimes going to sleep takes several minutes.
7. I always have an unpleasant sound. It affects my thought and sometimes my job. Going to sleep is often difficult without medication.
8. Tinnitus is severe. Sometimes I wake up with the same unpleasant sound.
9. Tinnitus is very severe. I feel depressed. I postpone social activities.
10. Tinnitus is unbearable. I can't work, I can't sleep, I always feel sick.

patients we chose 42 male patients (35%) with elevated cholesterol or triglyceride or both (age range, 19–60 years; mean age, 45 years). We placed all these patients on a low-cholesterol diet or antihyperlipidemic therapy (Table 2).

Antilipid treatment with statin derivatives was planned once-daily oral dosing for up to 1–2 years (minimum, 12.4 months). Daily dosage ranged from 10 to 40 mg

Table 2. Low-Cholesterol Diet*Never* drink or eat:

1. Alcoholic beverages of any kind.
2. Foods made with margarine (cream, chocolate, sauce, cake, pastry, etc.).
3. Foods roasted with oil (meat, vegetable, or pastry).
4. Foods made of offal or giblets (brain, liver, kidney, heart, spleen, tripe, etc.).
5. Dried fruits and nuts.
6. Ready-made foods and beverages (soup, juice, Coke, etc.).

Do not drink or eat:

1. More than 500 gm milk or yogurt in a day.
2. More than 60 gm white cheese (with less salt and fat) in a day.
3. More than 100 gm red meat or more than 150 gm chicken or fish in a day.
4. More than 6 thin slices of bread in a day.
5. More than 4 spoonfuls of sugar, 1–2 spoonfuls of honey or jam in a day.
6. More than one boiled egg in a week

Do drink or eat:

1. Boiled vegetables, salad without oil in every meal.
2. Fruits of all kinds.
3. Foods cooked with vegetable oil (corn, soybean, olive, sunflower—not much more than 3 spoonfuls in each meal).
4. Natural and fresh juice.
5. Meats, preferably cooked in an oven, boiled, or grilled.
6. Beans, chickpeas, rice, peas, lentils, etc.
7. Dessert made of milk or fruit.

Source: Department of Internal Medicine, Gulhane Military Medical School, Ankara, Turkey.

for simvastatin (Zocor) and from 10 to 80 mg for atorvastatin calcium (Lipitor). The patients were separated into two groups: the “unresponsive” group, made up of 22 patients who had no response to either of the therapies and still had hyperlipidemia, and the “responsive” group, consisting of 20 patients who had lower cholesterol or triglyceride levels.

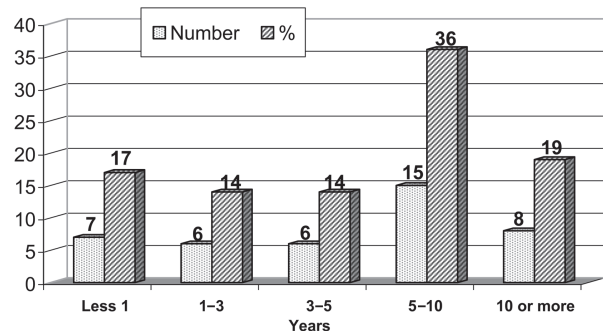
We then compared tinnitus scores and hearing levels in patients with no response to antihyperlipidemic therapy and in patients who had a normal blood cholesterol level after the treatment. Average ages of patients in the unresponsive and the responsive groups were 39 and 42 years, respectively. The results of the blood lipid profiles were withheld from the individual to prevent a placebo effect with the tinnitus therapy.

Statistical analyses were performed using SPSS 10.0 Windows PC programs (SPSS Inc., Chicago, IL). Wilcoxon and Mann-Whitney U tests were used to compare the pretreatment data with the posttreatment results and to compare the results of responders and nonresponders. The significance of the difference in patient's comments on their tinnitus after the treatment (“same,” “decreased,” “increased,” and “disappeared”) was analyzed using McNemar and chi-square tests.

RESULTS

The duration of tinnitus in patients with noise exposure varied from 2 months to 12 years (mean, 5.5 years). The distribution of tinnitus duration over the years is seen in Figure 1. Tinnitus was located in the right ear for 5 patients (11.9%) and in the left ear for 16 patients (38.1%). It was bilateral in 21 patients (50%); in all, 63 ears with tinnitus were evaluated. Of 42 patients with hyperlipidemia, 21 (50%) had elevated cholesterol and triglyceride, 11 (26.2%) had high triglyceride, and 10 (23.8%) had high cholesterol only.

LDL was high in 13, and VLDL was high in 15 patients. However, HDL was low in 2 patients. In all, 31 patients had high cholesterol, and 32 patients had high

**Figure 1.** Distribution of patients' tinnitus duration.

triglyceride levels. In the treated patient pool (n = 42), cholesterol ranged between 157 mg/dl and 334 mg/dl (mean, 233.4 mg/dl; SD, 44.9), and triglyceride ranged between 89 mg/dl and 487 mg/dl (mean, 253.8 mg/dl; SD, 106.2). The main criteria for response to the anti-hyperlipidemic therapy was defined as a return to normal cholesterol or triglyceride levels after therapy or at least a 40% drop from either of the initial blood cholesterol or triglyceride levels if the values were slightly above the normal limits.

Of 22 patients in the unresponsive group, 6 had high cholesterol and triglyceride, 9 had high triglyceride, and 7 had high cholesterol only before the antilipidemic therapy. None of these patients demonstrated any drop to normal ranges in cholesterol or triglyceride values after the treatment. Total cholesterol level ranged between 169 and 311 mg/dl before therapy and from 136 to 350 mg/dl after therapy. Triglyceride levels ranged between 89 and 416 mg/dl before therapy and from 112 to 402 mg/dl after therapy.

Of 20 patients in the responsive group, 7 had high cholesterol or triglyceride, 10 had high triglyceride, and 3 had high cholesterol only before the antilipidemic therapy. Total cholesterol levels ranged between 155 and 334 mg/dl (mean, 236 mg/dl) before therapy and ranged between 136 and 308 mg/dl after therapy (mean, 205.3 mg/dl; SD, 43.3; $p = .05$; $n = 20$). Triglyceride levels ranged between 155 and 485 mg/dl (mean, 281 mg/dl) before therapy and ranged between 96 and 343 mg/dl after therapy (mean, 168 mg/dl; SD, 57.1; $p = .01$; Table 3).

In those in the responsive group (n = 20), the tinnitus score ranged between 2 and 9 (mean, 4.95; SD, 1.9) before therapy and ranged between 1 and 8 (mean, 3.45; SD, 2.6; $p = .05$) after therapy. Change in tinnitus score before and after therapy in the responsive group was sig-

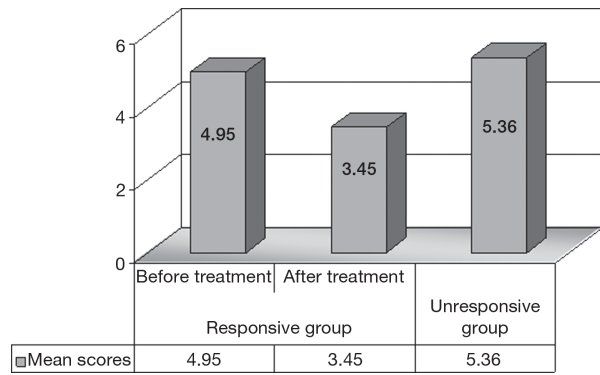


Figure 2. Comparison of mean tinnitus scores in the responsive and unresponsive groups.

nificant. In those in the unresponsive group (n = 22), the tinnitus score ranged between 2 and 9 (mean, 5.36; SD, 2.3) after therapy (Fig. 2). In those in the responsive group, pitch-match frequency ranged between 1,216 and 8,187 Hz (right ears [n = 19]: mean, 4,175.7; SD, 1,922; left ears [n = 15]: mean, 4,304; SD, 1,752.9) before therapy and after therapy ranged between 1,500 and 8,000 Hz (right ears [n = 15]: mean, 4,434.8; SD, 1,804.6; $p > .05$; left ears [n = 13]: mean, 4,532.1; SD, 1,342.3; $p > .05$).

The difference between the pitch-match frequencies before and after therapy was not significant. When those patients were asked to qualify the severity of their tinnitus, seven rated their tinnitus as the same (35%), two rated it as increased (10%), seven rated it as decreased (35%), and four had no tinnitus (20%). In those in the unresponsive group (n = 20), pitch-match frequency ranged between 1,216 and 7,216 Hz (right ears: mean, 3,468.2; SD, 1516.0; left ears: mean, 4,301.3; SD, 2110.7) after therapy. When patients were asked to qualify the severity of their tinnitus, 10 rated their tinnitus as

Table 3. Range of Total Cholesterol, Triglyceride, HDL, LDL, and VLDL Levels in Patients Who Had Response to Therapy and Those Who Had High Levels Despite the Therapy after Antihyperlipidemic Treatment

	Total Cholesterol (mg/dl)	Triglyceride (mg/dl)	HDL (mg/dl)	LDL (mg/dl)	VLDL (mg/dl)
Unresponsive group (n = 22)					
Before therapy	169–311 (mean, 232)	89–416 (mean, 229)	32–64 (mean, 44.5)	73–237 (mean, 144)	14–68 (mean, 39)
After therapy	136–350 (mean, 246)	112–402 (mean, 240)	31–63 (mean, 48.9)	66–257 (mean, 153)	17–61 (mean, 37)
Responsive group (n = 20)					
Before therapy	155–334 (mean, 236)	155–485 (mean, 281)	33–53 (mean, 43.5)	80–290 (mean, 144)	18–97 (mean, 38.7)
After therapy	136–308 (mean, 205)	96–343 (mean, 167)	33–53 (mean, 43.1)	57–234 (mean, 128)	22–56 (mean, 35.5)
Normal range	150–240	50–200	35–65	60–160	150–240

HDL = high-density lipoprotein; LDL = low-density lipoprotein; VLDL = very-low-density lipoprotein.

the same (47%), 9 rated it as increased (42%), 2 rated it as decreased (9%), and 1 patient had no tinnitus (2%).

Comparison of self-rated results in the two groups after therapy revealed a significant difference ($p < .05$). Conversely, loudness-matching measurements revealed that in the responsive group ($n = 20$), tinnitus severity ranged between 7 and 65 dB (right ears: mean, 37.9; SD, 22.4; left ears: mean, 47; SD, 29.8) before therapy and ranged between 10 and 65 dB (right ears: mean, 40; SD, 26.5; $p > .05$; left ears: mean, 44; SD, 28) after therapy. No significant difference in loudness level was found between the groups.

Average air-conduction pure-tone threshold for 500, 1,000, and 2,000 Hz was 18.2 dB (SD, 9.5) for the right ears and 17.3 dB (SD, 19.4) for the left ears in the responsive group ($n = 20$) before therapy. In this same group, it was 16 dB (SD, 10.5; $p > .05$) for the right ears and 15.2 dB (SD, 8.9; $p > .05$) for the left ears after therapy. In those in the unresponsive group ($n = 22$), average air-conduction pure-tone threshold for 500, 1,000, and 2,000 Hz was 20.1 dB (SD, 13.2) for the right and 21.3 dB (SD, 15.5) for the left ears after therapy.

Air-conduction pure-tone threshold for two high frequencies (4,000 and 8,000 Hz) was 43.1 dB (SD, 21.6) for the right ears and 45.2 (SD, 22.3) for the left ears in the responsive group ($n = 20$) before therapy. Air-conduction thresholds after therapy were 37.4 dB (SD, 23.2; $p < .05$) and 40.9 dB (SD, 24.3; $p < .05$) in the right and left ears, respectively. In the unresponsive group ($n = 22$), it was 54.4 dB (SD, 21.4) in the right and 55.5 dB (SD, 22.7) in the left ears after therapy. The difference in average air-conduction thresholds at high frequencies after the treatment was significant.

Speech reception threshold (SRT) ranged between 52% and 98% (right ears: mean, 87.9; SD, 13.5; left ears: mean, 98.1; SD, 9.2) in the responsive group ($n = 20$) before the therapy. SRT ranged between 72% and 100% (right ears: mean, 95.3%; SD, 8.1; $p > .05$; left ears: mean 90.3; SD, 20.6; $p > .05$) after the therapy. In the unresponsive group ($n = 22$), the speech discrimination score (SDS) ranged between 32% and 100% (right ears: mean, 88.9%; SD, 15.4; left ears: mean, 86.4; SD, 21.5) after the therapy. The difference in speech discrimination scores after therapy was not significant.

DISCUSSION

Epidemiological investigations emphasizing the relationship between hearing loss and the risk factors for coronary artery disease date back to the 1960s [18]. The correlation between serum lipids and auditory dysfunction has been investigated before, and it has been re-

ported that sensorineural hearing loss is more prominent in those with high levels of cholesterol, triglyceride, and low LDL and in patients with low HDL concentration [18–23]. However, the incidence of hyperlipidemia among a selected group of patients with neurosensory hearing loss varies.

Spencer [19] found that 42.3% of 300 patients with different symptoms of inner-ear pathology had clearly defined hyperlipoproteinemia. Cunningham and Goetinger [24] reported a low correlation. They selected a group of subjects aged between 20 and 50 years and compared auditory thresholds at high frequencies and serum lipid levels. Lowry and Isaacson [12] reported 20% hyperlipoproteinemia among 100 patients with bilateral hearing loss. Pruszewicz et al. [25] reviewed the possible etiology in 42 patients with sudden hearing loss and found that 55% had raised cholesterol level. In a survey of 44 patients with cochlear-type hearing loss, Booth [26] found hyperlipidemia in 28% of the men and 10% of the women. By contrast, the study by Pulec et al. [27] showed that this rate is about 5%.

The effect of hyperlipidemia on auditory perception has been studied clinically and experimentally. Ben-David et al. [28] compared the auditory brainstem responses in hyperlipidemic and normolipidemic subjects and reported latency prolongation in the hyperlipidemic patients. Saito et al. [29] showed delay in auditory brainstem wave latencies in 40% of guinea pigs fed a high-fat diet. Morizono and Paparella [30], using cholesterol-fed rabbits, showed increased auditory dysfunction with time at all frequencies. Preyer et al. [31] compared distortion product otoacoustic emission input-output functions in 20 patients with familial hypercholesterolemia without hearing loss against 20 healthy subjects and found reduced responses in the high-frequency region of the hyperlipidemic patients. Erdem et al. [32] reported similar results with distortion product otoacoustic emission at 4 Hz.

The exact pathological mechanism for the hyperlipidemia-induced hearing loss remains obscure. Increased blood viscosity and atherosclerosis of the cochlear vessels reduce the blood perfusion of the cochlea and promote hearing impairment [6,8,19,20,23,24]. Vascular mechanisms are not solely responsible for the auditory dysfunction. Lipidosis of the inner ear has been postulated by Nguyen and Brownell [33] as an alternate mechanism. The latter authors showed that the lateral wall of outer hair cells from guinea pig cochlea incorporates water-soluble cholesterol. This uptake of cholesterol is accompanied by an increased stiffness of the cells, which may impair the cells' electromotile response.

An interactive relation between noise and hyperlipidemia has been proposed. Noise as a stress factor has been shown to increase the serum cholesterol level [34].

Noise and metabolic disturbances have a synergistic effect on the development of tinnitus and hearing loss. Insufficient perfusion of the cochlea due to increased blood viscosity and microthrombosis related to hyperlipidemia has been claimed to cause susceptibility to noise-induced hearing loss [35]. Axelsson and Lindgren [34] matched 78 men at age 50 years with high serum cholesterol levels to 75 randomly selected 50-year-old men and found significant correlation between noise exposure and high cholesterol level. Sikora et al. [36] exposed chinchillas fed a high-fat diet to 105–114 dB of noise and observed more severe noise-induced high-frequency hearing loss in the hyperlipidemic group.

In his study of 64 rats, Pillsbury [37] exposed half to loud noise and fed half of each subgroup a fat diet and found elevated auditory brainstem response thresholds in the hyperlipidemic animals. Tami et al. [38] demonstrated in their study of 11 rabbits that the cochlear changes observed after noise exposure were comparable between the normal and hypercholesterolemic groups and that pathophysiology is related to cholesterol-induced chronic vascular changes and not solely to hypercholesterolemia. We found in our study that the incidence of hyperlipidemia among patients with noise-induced tinnitus and hearing loss was 35%.

Studies investigating the restorative possibility of a low-cholesterol diet and antihyperlipidemic therapy on hearing loss and tinnitus are very few. Rosen et al. [39] were the first to report that restoration of hearing after loss and relief of tinnitus is possible by changing the eating habits of patients with an elevated blood lipid level. Henry [40] and Sweet et al. [41] examined the dietary restriction of cholesterol and fat intake in mice. Enhancement of cochlear action potentials and auditory brainstem responses has been demonstrated after limited intake of cholesterol. Strome et al. [22] reported follow-up of three children with high serum cholesterol and sensorineural hearing loss who achieved improved hearing thresholds by reducing dietary cholesterol.

LDL apheresis has been reported to be much more effective as compared with standard prednisolone, dextran, and pentoxifylline therapy in patients with sudden hearing loss [9]. Pulec et al. [27] reported symptomatic relief in 83% of patients and an improvement of an average 15 dB through all frequencies in some of those after a low-cholesterol diet and vasodilator agents within a period of 5 months. Kojima et al. [42] studied 12 patients with unilateral sudden hearing loss and hyperlipidemia. However, patients were at the chronic phase, more than 1 month from the onset of the hearing loss. These authors observed hearing improvement of more than 10 dB in 9 of the 12 patients after antihyperlipidemic therapy.

In our study, changes in tinnitus scores before and after therapy in the responsive group were significant

as compared with those in the unresponsive group. When patients were required to qualify the severity of their tinnitus, 35% of the patients in the responsive group rated their tinnitus as decreased, and 20% of patients reported no tinnitus. In the unresponsive group, 9% of patients rated their tinnitus as decreased, and 1 patient reported no tinnitus (2%; $p < .05$). The difference of average air-conduction pure-tone thresholds for 500-, 1,000-, and 2,000-Hz frequencies before and after therapy between responsive and unresponsive groups was not significant. However, significant improvement was found at high-frequency (4,000- and 8,000-) thresholds in subjects who benefited from the antihyperlipidemic treatment as compared to their thresholds before therapy and to the thresholds of those who had no benefit from the treatment ($p < .05$).

Tinnitus and hearing loss are frequently encountered after an acute acoustic and blast trauma or chronic noise exposure. Even though other factors making the patients susceptible to noise-induced hearing loss should be considered, on the basis of present results it can be concluded that the incidence of hyperlipidemia is high among patients with noise-induced hearing loss and that significant improvement in lowered intensity of tinnitus and in average hearing thresholds at higher frequencies can be achieved after lowering patients' serum lipid level [43].

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