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Sudden Hearing Loss: A Ten-Year Outpatient Experience

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Abstract: The aim of this study was to determine the effects of various treatment modalities employed for patients with sudden sensorineural hearing loss (SHL). We retrospectively evaluated the records of patients treated in the sudden hearing loss section of the Otolaryngology Department at Clinic Hospital, School of Medicine, University of São Paulo, Brazil, between 1996 and 2006. Our study included patients with SHL of sudden onset (occurring over a 72-hour period) at equal to or greater than 30 dB at three consecutive frequencies. We divided patients into five groups by profile and treated them with dextran, dexamethasone, acyclovir, nicotinic acid, and papaverine hydrochloride (with or without vitamin A). We performed audiometry at baseline and on days 30, 90, 120, and 180 of treatment. We determined outcome as the difference between day-0 and day-180 pure-tone averages (PTAs). Among the 139 patients evaluated, baseline PTA was similar in all groups. We observed significant improvements in PTAs after 180 days of treatment and noted a significant linear correlation between time from SHL onset to initial visit and recovery. However, no significant difference was evident among the treatment groups. In the treatment of SHL, dextran provided no more benefit than did dexamethasone or acyclovir. Earlier initiation of treatment improves the prognosis for patients with SHL.

Key Words: acyclovir; dexamethasone; dextrans; hearing loss; sensorineural; sudden

Sudden sensorineural hearing loss (SHL) is defined as hearing loss equal to or greater than 30 dB at three or more consecutive frequencies, the onset of which takes place over a period of 3 days or fewer [1]. In most cases, it is severe, nonfluctuating, unilateral, and idiopathic [1,2]. Approximately one-third of cases are accompanied by complaints of tinnitus, dizziness, and ear fullness [2,3]. In the United States, the incidence is 5–20 cases per 100,000 habitants per year [1]. Worldwide, SHL accounts for approximately 1% of all cases of deafness, and 15,000 new cases occur annually [1,4]. Since SHL was first described 62 years ago [5], various etiologies have been proposed, including the infectious, traumatic, neoplastic, immune, ototoxic, vascular, ischemic, neurological, and metabolic. However, the etiology is identified in only 10–15% of cases [1].

Treatment is controversial and can involve anti-inflammatory drugs (steroids), vasodilators, antiviral agents, plasma expanders, diuretics, calcium channel antagonists, hyperbaric chamber use, carbogen, anticoagulants and, more recently, intratympanic corticosteroids [6–11]. Approximately one-third of affected patients improve spontaneously, most within the first 2 weeks of disease progression [1,3]. Risk factors associated with poor prognosis include time of disease progression prior to the initiation of treatment, extreme age (being very young or very old), severe initial hearing loss, concomitant vestibular symptoms, and a descending pure-tone audiometry curve [1,4,12].

The aim of this study was to observe the progression of SHL in patients who underwent different treatments at the SHL outpatient facility of our institution and to describe clinical and epidemiological aspects.

PATIENTS AND METHODS

This retrospective study analyzed the records of patients treated in the sudden hearing loss section of the Otolaryngology Department, Clinic Hospital School

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of Medicine, University of São Paulo, Brazil, between 1996 and 2006. The inclusion criterion was SHL of sudden onset over a 72-hour period, equal to or greater than 30 dB at three or more consecutive frequencies. The sample was composed of 139 patients, 73 (52.5%) female and 66 (47.5%) male. The mean age was 45.4 ± 15.8 years (range, 13–82 years). Patients were divided into five treatment groups on the basis of SHL duration and clinical profiles that indicated the use or absence of the current drugs prescribed.

Group I included patients who had a 5-day history of SHL and were hospitalized and treated with the plasma expander dextran (40,000 IU IV bid) for up to 10 days; dexamethasone (8 mg PO bid) for 10 days (followed by progressive weaning); acyclovir (200 mg PO tid) for 15 days; nicotinic acid (30 mg PO bid) for 30 days; papaverine hydrochloride (100 mg PO bid) for 30 days; and vitamin A (50,000 IU PO bid) for 30 days. Group II included patients who had a 0- to 15-day history of SHL and in whom the use of plasma expanders was contraindicated owing to systemic hypertension, cardiopathy, coagulation disorder, or renal failure. The treatment regimen was the same as that used in group I patients, minus dextran or minus dextran and acyclovir if the SHL onset was greater than 5 days. Group III included patients who had a 6- to 15-day history of SHL and who were hospitalized and treated with the same treatment used in group I minus acyclovir. Group IV included patients with a 16- to 30-day history of SHL and who were treated with the group I regimen minus dextran and acyclovir. Group V included patients with more than a 30-day history of SHL who were treated with nicotinic acid, papaverine hydrochloride, and vitamin A.

All patients underwent imaging tests (temporal bone magnetic resonance imaging or computed tomography) and complete blood cell counts. The laboratory exams included erythrocyte sedimentation rate, fasting glucose, cholesterol, triglycerides, free thyroxine and thyroid-stimulating hormone, circulating immunocomplex, complement, rheumatoid factor, and antinuclear factor. Serological tests were also conducted for herpes simplex virus type I, cytomegalovirus, syphilis, rubella, measles, Lyme disease, and the human immunodeficiency virus.

The patients underwent serial audiometric testing at baseline and after 30, 90, 120, and 180 days of treatment. Patients in groups I and III were hospitalized to receive intravenous dextran. For both groups, we performed pure-tone audiometry on alternate days. If no improvement occurred in the auditory threshold after 3 days, treatment was discontinued and the patient was discharged. The criteria for improvement were ≥10-dB increase at three consecutive frequencies; ≥15-dB increase at two consecutive frequencies; ≥20-dB increase at an isolated frequency; or ≥15% increase in the speech recognition index. If audiometric improvement occurred, the treatment regimen was maintained for up to 10 days. To document audiometric variation, we used the pure-tone average (PTA). The low-frequency PTA was determined by averaging the pure tones of the 250-, 500-, and 1,000-Hz frequencies, and the high-frequency PTA was based on those of the 2,000-, 4,000-, and 8,000-Hz frequencies. We determined audiometric evolution of the responses in the different treatment groups by subtracting baseline PTA from final PTA (after 180 days of treatment).

To analyze the statistical results, we used the Student's *t*-test and analysis of variance. The level of significance was set at $p < .05$ in two-tailed tests.

RESULTS

The SHL was unilateral in 129 patients (92.8%) and bilateral in 10 (7.2%). No statistically significant difference was evident in terms of gender or the side affected (Table 1; $p = .518$). Of the 139 patients evaluated, 77 (55.4%) were white, 35 (25.2%) were of African descent, 4 (2.9%) were of Asian descent, and 23 (16.5%) were of unknown ethnicity (information not provided in the medical chart). The mean time from SHL onset to the first medical visit was 17.2 ± 24.6 days (range, 0–120 days). The SHL was accompanied by airway infection in 25 cases (18%). Patients complained of dizziness in 75 cases (54%). Tinnitus was reported in 132 cases (95%). The metabolic disorders observed were lipid disorders in 33 cases (23.7%), glucose disorders in 15 cases (10.8%), and thyroid disorders in 12 cases (8.6%). Possible etiological factors for SHL were observed in 66 (47.5%) of the cases (Table 2).

Considering all the cases, we observed significant posttreatment reductions in low- and high-frequency PTA (Table 3). High-frequency PTA returned to normal values in 15 (14.7%) of the patients (Table 4), and low-frequency PTA did so in 28 (20.1%) (Table 5). At baseline, low- and high-frequency PTA were similar among the treatment groups (Table 6). No statistically significant difference was noted among the treatment groups in terms of audiometric progression determined by calculating the difference between the low- and high-frequency PTAs seen at baseline and after 180 days (Table 7).

Table 1. Gender Distribution of Laterality Among Patients with Sudden Sensorineural Hearing Loss

	Gender	
Laterality	**Male**	**Female**
Unilateral	60 (90.9%)	69 (94.5%)
Bilateral	6 (9.1%)	4 (5.5%)

Table 2. Etiology of Sudden Sensorineural Hearing Loss Among the Patients Studied

Etiology	No. of Patients (%)
Diabetes mellitus	15 (10.8)
Autoimmune disease	8 (5.8)
Acoustic trauma	6 (4.3)
Mumps (paramyxovirus)	5 (3.6)
Ototoxic drugs	5 (3.6)
Lyme disease	4 (2.9)
Ear malformation	4 (2.9)
Iatrogenic (otological surgery)	3 (2.2)
Syphilis	3 (2.2)
Head trauma	2 (1.4)
Anemia	2 (1.4)
Human immunodeficiency virus	2 (1.4)
Cytomegalovirus	1 (0.7)
Thalassemia	1 (0.7)
Thrombophilia	1 (0.7)
Oral contraceptive	1 (0.7)
Anticoagulant	1 (0.7)
Cochlear bleeding	1 (0.7)
Toxoplasmosis	1 (0.7)

Table 3. Comparison Between Baseline and Final Pure-Tone Averages

Frequencies	Baseline PTA (dB)	Final PTA (dB)	p[a]
Low	77.4 ± 27.1	55.8 ± 32.4	< .001
High	82.4 ± 30.3	65.7 ± 35.6	< .001

PTA = pure-tone average.
[a] Analysis of variance.
Note: Data presented as mean plus or minus the standard deviation.

Table 4. Etiology of Sudden Sensorineural Hearing Loss in Patients Presenting with Normalization of the PTA at High Frequencies

Patient	Time (days)	Etiology	Baseline PTA (dB)	Final PTA (dB)
1	3	Autoimmune	60	20
2	1	Cytomegalovirus	91.7	15
3	7	Oral contraceptive	63.3	25
4	7	Diabetes mellitus	46.6	25
5	10	Diabetes mellitus	60	10
6	7	ND	73.3	11.6
7	1	ND	70	15
8	10	ND	68.3	20
9	1	ND	46.6	11.6
10	7	ND	40	20
11	30	ND	35	15
12	3	ND	110	23.3
13	2	ND	36.7	15
14	3	Toxoplasmosis	61.6	18.3
15	3	Thrombophilia	90	10

ND = not determined; PTA = pure-tone average.

Table 5. Etiology of Sudden Sensorineural Hearing Loss in Patients Presenting with Normalization of the PTA at Low Frequencies

Patient	Time (days)	Etiology	Baseline PTA (dB)	Final PTA (dB)
1	3	Autoimmune	60	20
2	8	Mumps	97.5	17.5
3	1	Cytomegalovirus	68.3	15
4	7	Oral contraceptive	70	20
5	4	Diabetes mellitus	60	15
6	7	Diabetes mellitus	48.3	25
7	10	Diabetes mellitus	60	10
8	6	Diabetes mellitus	75	20
9	8	Diabetes mellitus	75	20
10	7	Lyme disease	41.7	25
11	30	ND	60	15
12	22	ND	41.6	16.6
13	2	ND	30	20
14	3	ND	32.5	20
15	2	ND	61.6	25
16	1	ND	31.6	15
17	3	ND	38.3	20
18	3	ND	71.6	10
19	10	ND	58.3	20
20	1	ND	70	13.3
21	2	ND	60	21.6
22	9	ND	40	20
23	10	ND	33.3	20
24	3	ND	110	21.6
25	2	ND	65	15
26	7	Head trauma	33.3	21.6
27	3	Toxoplasmosis	73.3	23.3
28	3	Thrombophilia	85	22.5

ND = not determined; PTA = pure-tone average.

We observed a significant inverse linear correlation between time from SHL onset to the first medical visit and the audiometric progression (baseline PTA minus final PTA: ΔPTA). Therefore, a longer time to the first medical visit resulted in minor ΔPTA. The Spearman correlation coefficient was $r = -0.45$ ($p < .001$; 95% confidence interval [CI] = −0.58 to −0.29) for the low-frequency PTA (Fig. 1) as compared with $r = -0.42$ ($p < .001$; 95% CI = −0.55 to −0.25) for the high-frequency PTA (Fig. 2). At baseline, mean low-frequency and high-frequency PTAs were similar for both genders. A borderline significant gender-based difference appeared in terms of the low-frequency and high-frequency ΔPTA values (Table 8).

DISCUSSION

Estimates have shown that among patients with SHL, the mean age ranges from 43 to 53 years, the gender distribution is equivalent, and the hearing loss is unilateral in more than 95% of cases [2,4,11,13,14], similar to what

Table 6. Baseline PTAs in the Groups Studied

	Baseline PTA (dB)					
Frequencies	**Group I**	**Group II**	**Group III**	**Group IV**	**Group V**	***p*[a]**
Low	79.9 ± 30.5	76.1 ± 26.1	78.8 ± 28.3	75.9 ± 26.3	78.4 ± 27	.945
High	88.8 ± 31.2	80 ± 33.5	79.1 ± 25.1	79 ± 36	86.9 ± 28	.275

PTA = pure-tone average.
[a] Analysis of variance.
Note: Data presented as mean plus or minus the standard deviation.

Table 7. Differences Between Baseline and Final PTAs in the Groups Studied

	ΔPTA (dB)					
Frequencies	**Group I**	**Group II**	**Group III**	**Group IV**	**Group V**	***p*[a]**
High	18.9 ± 20.6	29.6 ± 28.3	19.1 ± 19.9	11.8 ± 14.1	13 ± 18.6	.069
Low	25.7 ± 4.5	30.6 ± 31.2	23.1 ± 22.3	14.8 ± 14.9	20.4 ± 20.8	.237

PTA = pure-tone average; ΔPTA = baseline PTA minus final PTA.
[a] Analysis of variance.
Note: Data presented as mean plus or minus the standard deviation.

we observed in our study. The only epidemiological study of SHL published to date was performed in Japan in three different years and different decades [13]. The authors found that the time from the onset of SHL to the first medical visit was 11.6 ± 12.1 days in 1974, 9.1 ± 9.8 days in 1987, and 8.1 ± 9.1 days in 1993. Owing to differences in design and population, we cannot directly compare our sample with theirs. However, the fact that the time to first medical visit was longer in our study might be attributed to patients' lack of knowledge concerning the symptoms or difficulty in patients' gaining access to treatment.

Approximately 25–40% of patients with SHL present with concomitant upper-airway infection [11]. However, other authors [2] observed that, unlike our finding, only 11% of their SHL patients presented with concomitant upper-airway infection. Nevertheless, none of those authors compared their results with the prevalence observed in epidemiological studies of upper-airway infection in the corresponding populations. Although Nakashima et al. [13] did not investigate the prevalence of upper-airway infection in SHL, they found no correlation between SHL incidence and seasonality. Possibly these discrepancies in the prevalence of upper-airway

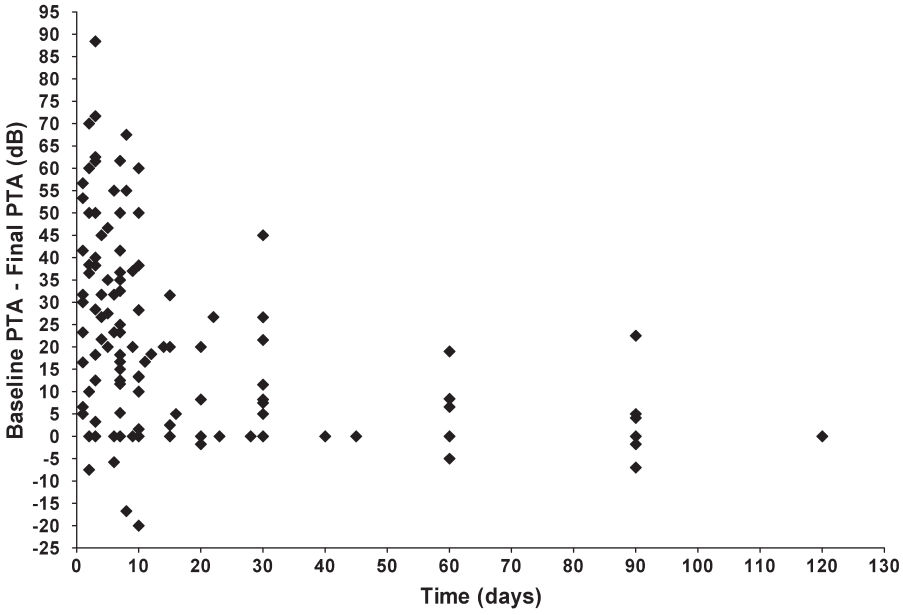

Figure 1. Correlation between onset of treatment and progression at low frequencies.

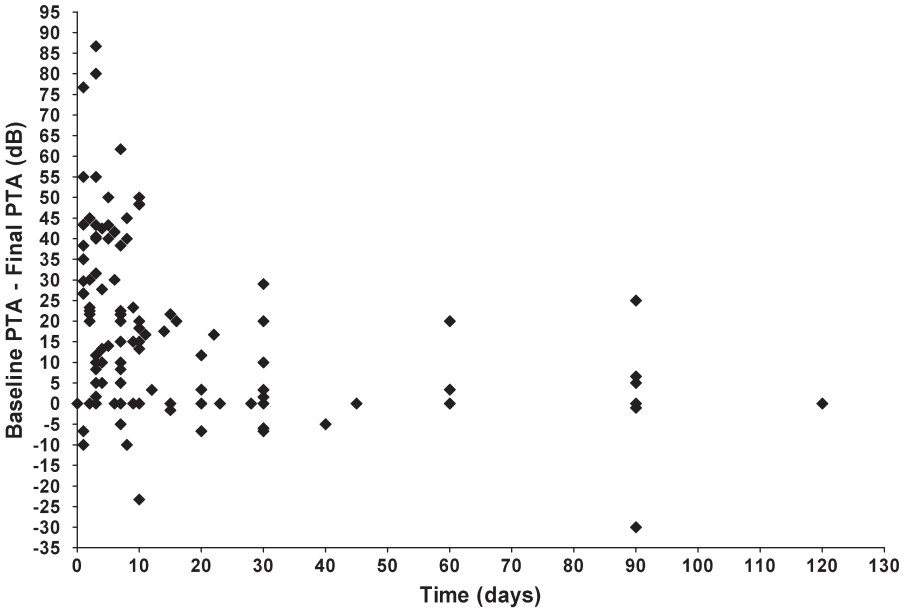

Figure 2. Correlation between onset of treatment and progression at high frequencies.

infection accompanying SHL are the result of geographical differences.

According to Rauch [11], vestibular symptoms were identified in 28–57% of cases, as compared with 32–40% reported by Nakashima et al. [13], 45% reported by Psifidis et al. [2], and 56% reported by Byl [4], all of which are in agreement with our findings. Concomitant tinnitus was reported by Psifidis [2] in 68% of cases and by Byl [4] in 74%. In our sample, tinnitus was much more prevalent (95% of cases) and was often the main reason for the disturbance, leading us to recommend that future clinical trials investigate therapeutic possibilities in preventing and managing this symptom in SHL patients.

In Brazil, Bittar et al. [15] observed that hypercholesterolemia (especially in terms of low-density lipoprotein levels), diabetes mellitus, and elevated thyroid hormone levels were more prevalent among the SHL patients presenting with vestibular complaints than in the general population. However, those authors found that prevalence of these disorders among patients with SHL was lower than that reported for patients with dizziness [15], which is in agreement with the findings by Cadoni et al.

Table 8. Gender Distribution of PTA

	Low-Frequency PTA (dB)	High-Frequency PTA (dB)
Female	25.2 ± 22.8	20.3 ± 21.7
Male	17.5 ± 20.2	12.3 ± 17.8
	$p = .071$[a]	$p = .053$[a]

PTA = pure-tone average.
[a] Analysis of variance.
Note: Data presented as mean plus or minus the standard deviation.

[16], who reported that metabolic risk factors were not highly prevalent among patients with SHL.

The most common etiological factors identified for SHL were those that are not the most frequent but can be directly identified through clinical history, blood samples, or imaging studies, including autoimmune diseases, acoustic trauma, ototoxic drugs, Lyme disease, and ear malformations. The autoimmune diseases that can lead to SHL are autoimmune inner-ear disease and immune-mediated systemic disease leading to a secondary inner-ear injury. Serological testing could be used to detect systemic evidence of immune dysfunction. However, despite advances, serological diagnosis remains difficult, as most tests are nonspecific and have a low accuracy rate [17]. In our sample, diagnoses were made by determining erythrocyte sedimentation rates and levels of antinuclear factor and rheumatoid factor. In two patients, the diagnoses were made based on a clinical history of vasculitis (Churg-Strauss syndrome) and thrombophilia, respectively.

The etiology of SHL has been hypothesized to be viral. One piece of evidence supporting this hypothesis is the fact that various authors have reported among patients with SHL seropositivity for a number of viruses, including herpes simplex, varicella zoster, cytomegalovirus, influenza, parainfluenza, rubella, and mumps [9, 18–20]. Veltri et al. [18] found that 65% of the SHL patients presented such seropositivity: 26% for influenza A3 or B; 16% for rubella; 8% for herpes; 8% for mumps; and 7% for cytomegalovirus. Stokroos et al. [9] found serological evidence of viral infection in 11.6% of the patients studied. Yoshida et al. [20] observed no statistically significant difference between patients with SHL and

controls in terms of seropositivity. Pitkaranta and Julkunen [21] investigated the protein MxA (a specific marker of systemic viral infection) in patients with SHL and found evidence of viral etiology in 6.5% of the cases. We found mumps (paramyxovirus) to be the most common etiology, although we did not test for influenza or parainfluenza. These discrepancies can be attributed to the difficulty of making the viral diagnosis, given that the time to the first medical visit (interval between the onset of SHL and blood collection) varied from study to study, which can influence seropositivity. However, we can assume that viral etiology is less relevant than it was in the past. Of note in our study and in a previous study [22] is that some of the patients presented seropositivity for the bacterium that causes Lyme disease.

Also, some evidence supports the idea that SHL has a vascular etiology. Studies involving patients with SHL have shown evidence of sudden-onset cochlear ischemia and an association between SHL and systemic vascular diseases [23]. In studies involving animal models, such association has also been demonstrated [23]. Blood circulation in the inner ear is of the terminal type, the cochlear artery running from the base to the apex of the cochlea. Theoretically, this anatomical presentation predisposes to hypoxia, spasms, thrombi, and hemorrhage, especially at the cochlear apex, primarily affecting the response to low frequencies [23]. In fact, vascular etiology is a very promising theory, but confirming and explaining it is difficult.

In our study, the etiology was vascular in 4.3% of the cases of SHL, one of which was very severe. In that case, SHL was detected after an episode of aplastic anemia (severe erythrocytopenia and thrombocytopenia) resulting from prolonged use of a nonsteroidal antiinflammatory drug (potassium diclofenac used daily for more than 1 year to treat acne). Intracochlear hemorrhage can be revealed through the use of magnetic resonance imaging, showing the importance of this procedure in making the diagnosis [24].

Considering that the groups were homogenous in terms of initial PTAs and analyzing all cases seen, we observed significant reduction in PTA, with mean improvement of 21.6 dB in the low-frequency PTA and 16.7 dB in the high-frequency PTA. Despite the lack of a control group, the findings of our study can be compared with those presented in the literature. Chen et al. [3] showed that spontaneous improvement (a mean 15-dB improvement in PTA) occurred in 32% of the control cases of SHL, lower than that observed in our study. The authors estimated that audiometric recovery resulting from treatment translated to an increase in mean PTA of approximately 25 dB, similar to what we observed for low-frequency PTA.

When we analyzed the cases in which PTA normalized, we observed that in most cases baseline PTA was moderate and the etiology was unknown. Among the remaining cases, the most common etiology was diabetes mellitus, followed by viral causes. We cannot state with confidence that the likelihood of complete recovery is greater in such cases of SHL, as other factors such as gender and age at SHL onset might exert some influence. To our knowledge, no previous studies have attempted to identify the etiologies that are most often associated with audiometric normalization in patients with SHL. However, we agree with Mattox and Simmons [25], who reported a fundamental difference between hearing losses at low frequencies and those affecting high frequencies, stating that low-frequency losses are more easily reversed. This was also observed in our study in which low-frequency PTA normalized in 28 cases as compared with 15 cases of high-frequency PTA normalization.

Among the five treatment groups, the differences in ΔPTA were not statistically significant at any frequency. The improvement in PTA was less pronounced in patients in groups IV and V (who started treatment at least 15 days after the onset of SHL) than in the other patients, although the difference was not statistically significant. In groups I, II, and III, treatment was initiated within the first 15 days after the onset of SHL. However, the greatest improvement in PTA was observed in group II patients, who were the only ones not treated by dextran. Although some underlying diseases can impair inner-ear microcirculation and hinder clinical improvement of SHL, the comorbidities presented by patients in group II were not indicators of poor prognosis. In our study, dextran was not found to act as an adjuvant to dexamethasone or acyclovir in the treatment of SHL, as the patients presenting the best response to treatment were those in the group not treated by dextran.

For low-frequency and high-frequency PTA, we observed an inverse linear correlation between time from onset to treatment and audiometric response presented by the patients. This finding is in agreement with those of other studies showing that earlier initiation of treatment improves prognosis [1,4,25]. We observed a gender-based difference in ΔPTA, with a tendency toward statistical significance, suggesting that the SHL prognosis is poorer for men. A recent study demonstrated a statistically significant association between less recovery of hearing loss and male gender [16]. The better prognosis in women merits further analyses, given a possible bias that might be related to indicators of good prognosis.

CONCLUSIONS

On the basis of our analysis, we can infer that plasma expanders do not improve the efficacy of the corticosteroid-acyclovir combination used in managing SHL in patients. We can also conclude that prompt treatment leads

to favorable evolution. Further studies are warranted to specifically address tinnitus and SHL.

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