Relationship between distortion product otoacoustic emission signal-to-noise and hearing threshold change during methylprednisolone therapy for sudden deafness

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Abstract. Sudden deafness is an emergency case in audiology requiring immediate treatment. According to the consensus on diagnosis and treatment of sudden hearing loss 2010 in Madrid, Spain, steroid drugs are the treatment of choice for sudden deafness with an unknown etiology. Patients recovering from sudden deafness exhibit increasing otoacoustic emissions that begin prior to hearing improvement. In this study, we evaluated a new methylprednisolone regimen (1 mg/kg daily, tapering by 20 mg every 5 days) for sudden deafness using distortion product otoacoustic emissions (DPOAE) and pure tone audiometry. Pure tone audiometry and DPOAE evaluations were conducted on 22 patients before treatment and on day 15 after therapy. Hearing threshold improved at all measured frequencies during treatment (1500−12000 Hz). There were also significant changes in DPOAE signal-to-noise ratio (SNR) at 1500, 2000, and 8000 Hz. Moreover, there were significant associations between SNR change and hearing threshold change at 8000 Hz and 10000 Hz. Thus, this new methylprednisolone regimen is highly effective for sudden deafness.

1. Introduction
Sensorineural sudden deafness refers to hearing loss that usually occurs only in one ear and has an unknown cause (e.g., trauma or ear canal blockage). Deafness is defined by the United States National Institute for Deafness and Communication Disorders as sensorineural hearing loss of ≥30 dB, at least in three consecutive hearing frequencies in approximately 72 hours. The incidence of sudden-onset deafness is reported to range from 5−20 per 100,000 individuals per year and is the most prevalent in individuals in the age group of 40−54 years. Idiopathic sudden deafness or idiopathic sudden sensorineural hearing loss is the most common type, accounting for 85%−90% of all sudden-onset cases, with the vast majority affecting only one ear (96%−99%). Spontaneous healing is reported in only 32%−65% of cases.

The cause of sudden-onset deafness is unknown. Currently, there are several major etiological hypotheses for sudden deafness, including viral infections, vascular disorders (thromboembolism, vasoconstriction, hypertension, and blood hyperviscosity), intracochlear membrane rupture, and autoimmune disease of the inner ear [1-9]. Based on the registry of the Neurotology Department of...
Otolaryngology, Cipto Mangunkusumo Hospital, 50 new cases of deafness with onset in <1 month were examined during from March 2011 to March 2012.

Sudden deafness is diagnosed based on anamnesis, ENT physical examination, audiology, laboratory testing, and other investigations. In addition to the diagnosis itself, these examinations also aim to identify the cause. Initial otoscopy should determine the presence of external ear abnormalities, such as serum impaction, and abnormalities in the middle ear, such as effusion, infection, tympanic membrane perforation, and the presence of mass. Pure tone audiometry provides the evidence of mild to severe sensorineural deafness and can also be performed as a treatment evaluation [1,3,10,11].

Otoacoustic emission (OAE) is a low-intensity sound that is measurable at the outer ear and reflects the activity of cochlear outer hair cells. These emissions can spontaneously occur because of internal sounds in the cochlea (spontaneous otoacoustic emission, SOAE) but are more often evoked by external acoustic stimulation (evoked otoacoustic emission, EOAE). In the clinic, EOAE is analyzed and interpreted at specific stimulus frequencies or within a limited frequency range; therefore, it is possible that an OAE check yields a normal value at one stimulus frequency and abnormal or undetectable emissions at another. OAEs evoked by high-frequency sounds are more specific and sensitive than those evoked at low to medium frequencies. Permanent damage to outer hair cells cause persistent hearing and EOAE loss at specific frequencies. There are two types of EOAEs used clinically, namely transient evoked otoacoustic emissions (TEOAEs) and distortion product otoacoustic emissions (DPOAEs). The latter are intermodulation distortion responses generated by the ear in response to two pure tones simultaneously occurring at different frequencies and intensities. These emissions can be recorded at a frequency interval of 1–12 kHz, which is a greater range and peak frequency than that possible using TEOAE. The measurement of DPOAE uses a two-tone pure stimulus at two frequencies (f1 and f2, where f2 > f1) and two intensities (L1 and L2). The frequency ratio (f2/f1) is 1/2, and the intensity ratio (L1/L2) is often 65/55 dB SPL. The assessment of DPOAE yields signal-to-noise ratios (SNRs) by calculating the difference between OAE intensity and the floor noise level. At a given frequency, SNR ≥ 6 is considered normal (Pass Criterion), whereas SNR < 6 is considered an indication for medical referral (Refer Criterion) [12–18].

The management of sudden deafness greatly varies because of the large number of predisposing factors and possible etiologies. Shotgun therapy is the most common because the cause of sudden deafness is usually not immediately known at diagnosis. Several therapeutic regimens, such as vasodilators (oral papaverine, histamine infusion, oral nicotinic acid, and carbogen inhalation therapy), anticoagulant drugs, low molecular weight dextran, corticosteroids, diuretics, vitamins, minerals, and sedative drugs, have been reported to provide effective results. The 3rd Madrid Congress attended by experts from various countries created a Consensus on Diagnosis and Treatment of Sudden Hearing Loss that established corticosteroids as the first-line therapy. The choice of prednisone or methylprednisolone therapy is at the discretion of the treating clinician. Both drugs are corticosteroids with medium or intermediate working time and half-life of 12–36 h. Steroids may suppress damage to the inner ear from cytotoxic immune response and/or increase microvascular flow in the cochlea, thereby preventing the onset of endolymphatic hydrops. Based on these actions, it is also speculated that anti-inflammatory effects of corticosteroids contribute to healing from sudden deafness [1,9,11,19,20]. The therapeutic response is classified according to changes in the hearing threshold as follows: (1) healed if the improvement (threshold reduction) is >30 dB at five frequencies; (2) very good if the hearing threshold is <30 dB at 250, 500, 1000, and 2000 Hz and below 25 dB at 4000 Hz; (3) good if the threshold reduction is 10–30 dB at five frequencies; and (4) no improvement if hearing threshold decreases by <10 dB at five frequencies [11].

In this study, we examined the efficacy of a modified methylprednisolone regimen and also tested the prognostic value of DPOAE SNR by assessing the relationship with changes in hearing threshold during therapy (i.e., therapeutic response).
2. Methods
This study used a pre–post analytic pre-experimental design to assess the relationship between DPOAE SNR changes and hearing threshold changes as measured by pure tone audiometry during the administration of methylprednisolone (1 mg/kg daily in the morning after meals, tapering by 20 mg every 5 days). The study protocol was approved by the Health Research Ethics Committee, Faculty of Medicine, Universitas Indonesia-Cipto Mangunkusumo Hospital. The study included 22 patients treated at the Neurotology Department of Otolaryngology, Cipto Mangunkusumo Hospital, Jakarta, Indonesia. Admission criterion was sudden deafness with an onset of ≤1 month based on anamnesis, physical examination, tuner, and pure tone audiometry. Exclusion criteria were abrupt hearing loss, known causes such as trauma and malignancy; abnormalities in the ear canal or middle ear detected by otoscopy, tuning, and tympanometry; and contraindications to methylprednisolone therapy. Subjects were enrolled as candidates by consecutive sampling from July 2012 to February 2013. Patients underwent pure tone audiometry and DPOAE tests before treatment and on the 15th day of therapy. Audiometric examination was performed at 11 frequencies (250, 500, 1000, 1500, 2000, 3000, 4000, 6000, 8000, 10000, and 12000 Hz) and DPOAE at nine frequencies (500, 2000, 3000, 4000, 6000, 8000, 10000, and 12000 Hz). Changes in SNR and hearing thresholds after therapy were assessed using paired t-tests if the data were normally distributed both before and after treatment or using Wilcoxon signed rank test if one or both data sets were not normally distributed.

3. Results

Table 1. Clinicodemographic characteristics of patients with sudden deafness (n = 22)

| Characteristic                     | N   | %   |
|-----------------------------------|-----|-----|
| **Age**                           |     |     |
| 18–40 years                       | 8   | 36.4|
| 41–60 years                       | 10  | 45.5|
| >60 years                         | 4   | 18.1|
| **Sex**                           |     |     |
| Male                              | 13  | 59.1|
| Female                            | 9   | 40.9|
| **Onset of sudden deafness**      |     |     |
| ≤7 days                           | 6   | 27.3|
| 8–14 days                         | 4   | 18.2|
| 15–21 days                        | 7   | 31.8|
| ≥22 days to 1 month               | 5   | 22.7|
| **Unilateral**                    | 21  | 95.5|
| **Bilateral**                     | 1   | 4.5 |
| **Degree of deafness**            |     |     |
| 0–25 dB                           | 5   | 21.7|
| 26–40 dB                          | 3   | 13.0|
| 41–55 dB                          | 2   | 8.7 |
| 56–70 dB                          | 7   | 30.4|
| 71–90 dB                          | 2   | 8.7 |
| >90 dB                            | 4   | 17.4|
| **Predisposing Factors**          |     |     |
| DM/Hypertension/Dyslipidemia      | 8   | 36.4|
| DM + Hypertension/Dyslipidemia    | 7   | 31.8|
| Others                            | 7   | 31.8|
Data are presented in Table 1 were on the basis of the analysis of 23 ears (one of the 22 patients had bilateral hearing loss). Predisposing factors for sudden deafness in this patient cohort included diabetes mellitus, hypertension, dyslipidemia, or combination of those disease mentioned before, as well as less common predisposing factors, such as blood clots and history of renal failure in 31.8% of the patients undergoing hemodialysis.

Table 2 shows the distribution of mean SNR (±SD) or median (interquartile range) of DPOAE SNR before therapy and after 15 days of therapy. The biggest change was at 2000 Hz, followed by 1500 Hz and 8000 Hz. Figure 1 shows the SNR value bar chart prior to therapy and after 15 days of therapy according to the “Pass” and “Refer” criteria, where SNR ≥ 6 is deemed a “Pass” and SNR < 6 as “Refer”. Changes in the proportions of Pass and Refer were observed at 1500, 2000, 8000, 10000, and 12000 Hz, with the largest change (six patients shifting from Refer to Pass) at 8000 Hz. No changes were observed at 4000 and 6000 Hz.

Table 2. Mean or median SNR at each tested frequency before therapy and after 15 days of methylprednisolone therapy

| Frequency (Hz) | Median value Before therapy | Median value After therapy |
|---------------|-----------------------------|----------------------------|
| 1500          | 3 (0–28)**                  | 6 (0–28)**                 |
| 2000          | 6 (0–28)**                  | 10.48 ± 8.22*              |
| 3000          | 6 (0–20)**                  | 7 (0–20)*                  |
| 4000          | 0 (–3–12)**                 | 0 (0–19)*                  |
| 6000          | 1 (0–17)**                  | 1 (0–29)*                  |
| 8000          | 4 (0–16)**                  | 6 (1–35)*                  |
| 10000         | 5.43 ± 2.90*                | 6 (0–26)*                  |
| 12000         | 4.17 ± 3.22*                | 5.65 ± 3.66**              |

*normally distributed data; **non-normally distributed data.

Figure 1. Stem distribution of SNR values expressed as Refer and Pass on the 1st and 15th day of therapy.
Table 3 shows the distribution of mean (±SD) or median (IQR) hearing thresholds from pure tone audiometric examination before and after therapy. The distribution before therapy indicated that some subjects still demonstrated normal hearing (threshold ≤ 25 dB) at 250, 500, 1000, and 1500 Hz, whereas threshold was ≥25 dB at all other tested frequencies.

Table 3. Mean or median hearing threshold at each tested frequency before treatment and after 15 days of methylprednisolone therapy

| Frequency (Hz) | Median value Before therapy (dB) | Median value After therapy (dB) |
|---------------|----------------------------------|---------------------------------|
| 250           | 46.74 ± 30.36*                   | 20 (5–95)                       |
| 500           | 48.91 ± 30.52*                   | 25 (5–105)                      |
| 1000          | 51.74 ± 31.93*                   | 35 (5–105)                      |
| 1500          | 53.48 ± 30.32*                   | 40 (5–115)**                    |
| 2000          | 56.30 ± 32.09*                   | 43.04 ± 33.90*                  |
| 3000          | 60 (20–115)**                    | 43.26 ± 32.67*                  |
| 4000          | 65 ± 32.29*                      | 51.74 ± 32.28*                  |
| 6000          | 75 (25–105)**                    | 70 (0–105)**                    |
| 8000          | 85 (25–105)**                    | 75 (5–105)**                    |
| 10000         | 90 (30–95)**                     | 80 (10–95)**                    |
| 12000         | 85 (45–85)**                     | 85 (5–85)**                     |

*normally distributed data; **non-normally distributed data.

A change in the mean or median hearing threshold was visible at all frequencies, except at 12000 Hz. Although there was no change in the median threshold at 12000 Hz, the minimum value was reduced from 45 dB to 5 dB. The biggest change, from 46.74 ± 30.36 dB to 20 (5–95) dB, was observed at 250 Hz. Figure 2 presents a bar chart of subject distribution according to threshold improvement of ≥10 dB after 15 days of therapy. The greatest hearing improvements were at 2000, 3000, and 6000 Hz (greatest difference between the number of improved and non-improved patients), whereas the lowest were at 8000, 10000, and 12000 Hz.

![Figure 2](image-url)
Table 4 presents the statistical evaluation of threshold changes after 15 days of therapy relative to baseline. There were significant changes at all tested frequencies (p < 0.05).

| Frequency | Changes in hearing threshold after methylprednisolone therapy |
|-----------|---------------------------------------------------------------|
| 1500 Hz   | Hearing threshold before therapy 23 53.48 ± 30.32 | <0.001 |
| 2000 Hz   | Hearing threshold before therapy 23 56.30 ± 32.09 | <0.001 |
| 3000 Hz   | Hearing threshold before therapy 23 60 (20–115) | <0.001 |
| 4000 Hz   | Hearing threshold before therapy 23 65.00 ± 32.29 | <0.001 |
| 6000 Hz   | Hearing threshold before therapy 23 75 (25–105) | <0.001 |
| 8000 Hz   | Hearing threshold before therapy 23 85 (25–105) | <0.001 |
| 10000 Hz  | Hearing threshold before therapy 23 90 (30–95) | 0.001 |
| 12000 Hz  | Hearing threshold before therapy 23 85 (45–85) | 0.012 |

a : paired t-test ; b : Wilcoxon signed rank test ; p<0.05 (Statistically Significant)

Table 5 presents the change in SNR values after 15 days of methylprednisolone therapy. There were significant changes at 1500, 2000, and 8000 Hz (p < 0.05).

| Frequency | Changes in DPOAE signal-to-noise ratio after methylprednisolone therapy (1 mg/kg BW) |
|-----------|--------------------------------------------------------------------------------------|
| 1500 Hz   | DPOAE value before therapy 23 3 (0–28) | 0.01 |
| 2000 Hz   | DPOAE value before therapy 23 6 (0–28) | 0.005 |
| 3000 Hz   | DPOAE value before therapy 23 6 (0–20) | 0.23 |
| 4000 Hz   | DPOAE value before therapy 23 0 (–3–12) | 0.23 |
| 6000 Hz   | DPOAE value before therapy 23 1 (0–17) | 0.28 |
| 8000 Hz   | DPOAE value before therapy 23 4 (0–16) | 0.001 |
| 10000 Hz  | DPOAE value before therapy 23 5.43 ± 2.90 | 0.24 |
| 12000 Hz  | DPOAE value before therapy 23 4.17 ± 3.22 | 0.054 |

a : paired t-test ; b : Wilcoxon signed rank test ; p<0.05 (Statistically Significant)
Finally, we examined the relationship between threshold change and SNR change after therapy (Table 6). At frequencies of 8000 and 10000 Hz, most patients exhibiting a change in SNR also exhibited a change in hearing threshold.

**Table 6. Relationship of SNR changes with changes in hearing threshold**

| Frequency     | n     | Median (SD) | p     |
|---------------|-------|-------------|-------|
| 1500 Hz       | 8     | 8.2 ± 10.5  | 0.74<sup>a</sup> |
|               | 15    | 7.4 ± 6.0   |       |
| 2000 Hz       | 7     | 4 (1–29)    | 0.25<sup>b</sup> |
|               | 16    | 11 (1–23)   |       |
| 3000 Hz       | 7     | 4.9 ± 6.3   | 0.26<sup>a</sup> |
|               | 16    | 8.5 ± 6.7   |       |
| 4000 Hz       | 9     | 0 (0–13)    | 0.88<sup>b</sup> |
|               | 14    | 0 (0–19)    |       |
| 6000 Hz       | 7     | 0 (0–11)    | 0.28<sup>b</sup> |
|               | 16    | 1 (0–29)    |       |
| 8000 Hz       | 11    | 5.0 ± 2.1   | 0.03<sup>a</sup> |
|               | 12    | 11.7 ± 10.3 |       |
| 10000 Hz      | 8     | 3.4 ± 1.9   | 0.003<sup>a</sup> |
|               | 15    | 10.1 ± 6.4  |       |
| 12000 Hz      | 17    | 5.0 ± 2.7   | 0.47<sup>a</sup> |
|               | 6     | 6.9 ± 5.5   |       |

<sup>*p < 0.05, paired t-test, **p < 0.05, Mann–Whitney U test</sup>

<sup>a : paired t-test ; b : Mann–Whitney U test ; p<0.05 (Statistically Significant)</sup>

4. Discussion
This study demonstrated a significant improvement in hearing (lower threshold) over a broad frequency range (1500–12000 Hz) using a new methylprednisolone regimen. Furthermore, there was a significant association between the changes in hearing threshold and DPOAE SNR at 8000 and 10000 Hz. At these two frequencies, the majority of patients exhibiting a change in threshold also exhibited a change in SNR, suggesting that DPOAE predicts clinical outcomes at these frequencies.
Major predisposing factors for sudden deafness in this study were diabetes, hypertension, dyslipidemia, blood clotting factor disorders, and renal failure. It has been suggested that such predisposing factors cause secondary cochlear damage through anoxia or hypoxia [21]. In a study on 106 patients with sudden deafness, Orita et al. [7] found significantly greater hearing loss in hyperglycemia patients than normoglycemic patients (p < 0.05), but no difference in the severity between hypercholesterolemia patients and those with normal serum cholesterol. Hemorrhagic improvement did not significantly differ between the groups. Lin et al [8] conducted a 4-year cohort study from 2000 to 2004 to evaluate the incidence of sudden deafness and found a significantly higher incidence in diabetics than in non-diabetics (p < 0.001). The present results are also consistent with a major etiological contribution by diabetes, although the sample was of insufficient size to assess whether such patients had more severe hearing loss.

In the present study, the biggest change in the median or mean value of SNR after 15 days of methylprednisolone therapy was found at 2000 Hz, followed by at 1500 and 8000 Hz, whereas no changes were observed at 4000 and 6000 Hz. The absence of improvement at 4000 and 6000 Hz may reflect a greater impairment of cochlear cells sensitive to these frequencies. At 4000 and 6000 Hz, only four subjects were rated as Pass (SNR ≥ 6) before therapy, suggesting more severe damage in cochlear cells with sensitivities within this frequency range. Alternatively, greater improvement is expected at frequencies with more Pass patients before treatment, indicative of less severe damage. Indeed, a substantially larger number of patients were in the “Refer” category (SNR < 6) before treatment at frequencies ≥ 4000 Hz than at 250–3000 Hz. This can be explained by the greater sensitivity of high-frequency responsive outer hair cells at the basal cochlea to immune reaction-induced oxidative stress due to lower endogenous antioxidant capacity than that of low-frequency responsive cells at the cochlear apex. This greater susceptibility to free radical injury is the basis for the well documented progression of hair cell damage from high- to low-frequency response type [22]. In this study, we found that the mean and median thresholds were near normal (≤ 25 dB) before therapy at 250, 500, 1000, and 1500 Hz but were ≥25 dB at higher frequencies. In other words, basal outer hair cells sensitive to high-frequency sound (6000, 8000, 10000, and 12000 Hz) were more extensively impaired or injured than cells nearer to the apex prior to therapy, and therefore, were less responsive to treatment [22]. Indeed, the greatest decrease in mean threshold after therapy was observed at 250 Hz [from 46.74 ± 30.36 dB to 20 (5–95) dB], which could be attributed to the greater protection against cytotoxic inflammation conferred by antioxidants in low-frequency responsive hair cells. Mean or median hearing threshold values after 15 days of methylprednisolone therapy significantly improved at all frequencies, except at 12000 Hz, although even at this high frequency, 26.1% of patients showed improvement.

Dispenza et al. [9] reported 21 cases of sudden-onset deafness treated for 10 days with 60 mg daily oral prednisone, tapering at 14 days, and found a threshold decrease of >10 dB in 81% of the cases. Similarly, Rauch [4] studied 121 sudden-onset deafness cases over 5 years and found an average threshold decrease of 30.7 dB after 14 days of prednisone therapy (60 mg/day with tapering). Collectively, these results indicate that immunosuppression by corticosteroids accelerates the healing process. Statistical analysis revealed a significant change in threshold after 1 mg/kg daily methylprednisolone at all tested frequencies (p < 0.05). Enache and Sarafoleanu [6] conducted a study on 47 sudden deafness cases in the age range of 50–70 years treated with the non-steroidal anti-inflammatory pentoxifylline at 200 mg/day, cortisone at 300–400 mg/day, vitamin B1 and B6, calcium, and plasma expander at 500 mg/day. Twenty patients recovered, 18 demonstrated permanent hearing loss of 10–40 dB, and nine did not improve. Notably, patients who did not experience a threshold improvement had a greater degree of deafness at onset. Thus, Enache and Sarafoleanu [6] concluded that severe deafness at onset is suggestive of irreversible damage (that cannot be improved by therapy). Moreover, other studies have concluded that the prognosis of threshold recovery is influenced not only by the severity of deafness at onset but also by age, presence of vertigo, delay between onset and treatment, audiogram pattern, DPOAE amplitude, and accompanying predisposing
factors [14-23,24]. In the present study, some subjects with a high degree of deafness at onset showed a hearing loss of >10 dB even after therapy.

Another possible factor influencing recovery is the route of administration. However, Dispenza et al. [9] found no significant difference in threshold improvement using intratympanic injection versus oral prednisone (60 mg with tapering in 14 days). Thus, oral steroid therapy is the primary choice because intratympanic steroid injection can cause the perforation of the tympanic membrane, pain (both during and after injection), vertigo, and deafness. Early intervention likely reduces the deleterious effects of the cytotoxic immune response and increases microvascular flow in the cochlea, thereby preventing endolymphatic hydrops [9-14-25].

Based on the Consensus on Diagnosis and Treatment of Sudden Hearing Loss issued at the 3rd Madrid Congress, experts agree that the treatment of choice for sudden deafness is oral systemic corticosteroids with additional intratympanic corticosteroids if systemic administration does not lead to improvement. The choice of therapy may be prednisone or methylprednisolone at 1 mg/kg once daily after breakfast, tapering by 20 mg every 5 days. This consensus was the basis for the selection of 1 mg/kg daily methylprednisolone therapy in this study and the evaluation of therapeutic results on the 15th day. For patients with predisposing diabetes mellitus, steroids were administered after obtaining consent from the relevant department and additional efforts were made to regularly monitor the blood sugar levels.

SNR value also significantly changed by therapy at 1500, 2000, and 8000 Hz. Several factors, such as age, sex, environmental noise, genetics, spontaneous emission levels, body temperature, and affected ear (left or right), have been demonstrated to influence DPOAE values [13]. Thus, heterogeneity among subjects may have obscured differences at other frequencies. OAE response reflects preserved cochlear function, and predisposing factors that accompany sudden deafness contribute to cochlear damage. Dyslipidemia, hypertension, and diabetes lead to atherosclerosis through endothelial activation, resulting in a circulatory deficit, ensuing hypoxia or anoxia in the cochlea, and hair cell damage. Damage to specific hair cell populations caused by these conditions may explain the small change in DPAO amplitude after treatment at certain frequencies [13-21]. Park et al. evaluated DPOAE and audiometry values on the 1st, 7th, and 14th day of steroid therapy in 40 patients with sudden deafness and found that higher DPAOE values at onset predicted better prognosis but that the absence of DPOAE values at onset did not necessarily indicate poor prognosis. Further, 75% of patients who experienced threshold recovery also showed an increase in DPOAE values. This incidence is expected because mild to moderate degrees of deafness are generally associated with major damage to neural pathways, whereas patients with severe-to-very severe deafness exhibit both nerve and cochlear damage.

There were no significant relationships between SNR and threshold changes at the majority of tested frequencies (1500, 2000, 3000, 4000, 6000, and 12000 Hz), whereas significant associations were found at 8000 and 10000 Hz (p < 0.05). In the aforementioned study by Park et al. [5], it was concluded that the change in DPAOE SNR does not correlate with hearing impairment if mild to moderate at onset but that DPOAE SNR values are important predictors of threshold improvement if the degree of deafness at onset is severe-to-very severe. This conclusion requires further verification to ascertain the true location of the lesion in light-to-moderate and severe-to-very severe deafness. In this study, we found a similar pattern in that there was a significant relationship between SNR and hearing threshold changes at frequencies showing fairly large initial increases in threshold (8000 and 10000 Hz) indicative of severe and very severe damage. Alternatively, at lower frequencies with no such relationship, audiometric values were almost equally divided between mild, moderate, and severe. Currently, there is no well substantiated explanation for this frequency-specific relationship. Nonetheless, changes in DPOAE SNR values may be used to evaluate prognosis for hearing recovery at specific frequencies.
5. Conclusion

This study described a new methylprednisolone regimen with good efficacy against sudden deafness over a broad range of frequencies. The relationship between treatment-induced change in hearing threshold and DPAEA SNR may help predict treatment outcomes at certain hearing frequencies.

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