Case Report

Low-Frequency Sensorineural Hearing Loss Associated With Iron-Deficiency Anemia

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Past studies have found an association between cochlear hydrops or early Meniere’s disease and acute low-frequency hearing loss (ALHL) without vertigo. However, its mechanism remains unclear in some ALHL cases. This report presents a case of ALHL associated with iron-deficiency anemia (IDA). The patient was a 49-year-old female who had previously been treated with betamethasone for sudden hearing loss in the right ear. Eight months later, the symptoms recurred and cochlear hydrops was diagnosed. Isosorbide and betamethasone were administered orally, and intravenous hydrocortisone tapering was added 1 week later, but these treatments were ineffective. At the same time and subsequently, iron sucrose was intravenously administered for IDA, and the patient’s hearing loss gradually resolved within 2 months. In view of the increase in serum hemoglobin levels after iron therapy, this might have been the most effective treatment in this case. The hearing loss could therefore be associated with the patient’s IDA.

KEYWORDS: Low-frequency sensorineural hearing loss, iron, anemia, ALHL

INTRODUCTION

Acute low-frequency hearing loss (ALHL) without vertigo has been defined as a distinct entity from idiopathic sudden sensorineural hearing loss (ISSNHL) and shows good prognosis.1 Although some studies have found an association with cochlear hydrops or early Meniere’s disease,2,3 its mechanism remains unclear in some ALHL cases. Iron-deficiency anemia (IDA) is common and accounts for approximately half of all anemia cases worldwide. The disease is caused by insufficient dietary intake and/or absorption of iron and/or loss of iron through bleeding of the intestinal, uterine, or urinary tracts.4 Although several studies have shown an association between sensorineural hearing loss (SNHL) and IDA,4,5 there are no existing reports on the relationship between ALHL in particular, and IDA. Here, we report a case of ALHL probably associated with IDA.

CASE PRESENTATION

The patient was a 49-year-old female who visited an ENT clinic for a loss of hearing in her right ear. Pure-tone audiometry (PTA) showed low-frequency SNHL in the right ear. Diagnosed as ISSNHL, oral betamethasone was administered at a daily dose of 6 mg, and the patient’s hearing had improved to normal 2 days later. After 8 months, the patient again experienced hearing loss and fullness in the right ear and visited another ENT clinic. She did not have vertigo, and both ear drums were normal. PTA again showed low-frequency SNHL in the right ear (Figure 1A). The patient was referred to a hospital, where prednisolone was intravenously administered on days 1 to 3 at a daily dose of 60 mg; a subsequent PTA showed no change in condition. Endolymphatic hydrops was suspected because of the recurrent low-frequency SNHL. The patient was treated with 60 mL of 70% isosorbide, an osmotic diuretic, daily, and tapering oral betamethasone with an initial dose of 3 mg decreasing to 1 mg over a period of 6 days. One week later (day 11), PTA showed no improvement. The patient was therefore treated with tapering intravenous hydrocortisone succinate as a higher steroid dose at 300 mg for 2 days, 200 mg for 3 days, and 100 mg for 3 days, but the hearing loss did not completely resolve (Figure 1B). A re-administration of isosorbide from days 25 to 39 was also ineffective, and the hearing level was seen to deteriorate on day 25 (Figure 1C).
The laboratory examination from day 1 showed microcytic anemia (serum hemoglobin (Hb): 8.3 g/dL; hematocrit: 26.7%; mean corpuscular volume: 75.4 fl). The serum iron level was 28 μg/dL, and the unsaturated iron-binding capacity was 349 μg/dL. These data were compatible with IDA. Considering that the patient had experienced nausea with oral iron tablets, intravenous iron sucrose was administered in 80 mg doses 13 times over 2 months. The total dose of iron sucrose was calculated based on the formula: [target Hb − actual Hb (g/dL)] × 2.4 × body weight (kg) + iron stores (mg). The iron sucrose injections were intermittently administered because of the patient’s schedule. On day 28, the serum Hb level had increased to 11.2 g/dL, near the cut-off value of 12 g/dL in non-pregnant adult women. On day 53, PTA showed a significant improvement, despite the cessation of steroid therapy. On day 67, the patient’s hearing level had resolved almost to normal (Figure 1D). Figure 2 illustrates the clinical course including laboratory data, therapies, and mean PTA thresholds at low frequencies (125, 250, and 500 Hz). In view of the increase in serum Hb after iron sucrose administration, iron therapy could be the most effective element in hearing loss management in this case.

DISCUSSION

The clinical features of and treatments for ALHL have been discussed in Japan and Korea. A recurrence of hearing loss, and subsequent progression to Meniere’s disease, can suggest a pathophysiology of cochlear endolymphatic hydrops and early-stage Meniere’s. In terms of ALHL treatment, the efficacy of steroids is controversial. Morita et al. showed that recovery rates with a combination of steroids and diuretics were significantly better (91.3%) than those of the control group (66.7%), and of steroids (75.5%) or diuretics (75%) alone. Other studies have also shown significantly high recovery rates of around 80% with combined oral steroid and diuretic treatment. In the present case, a combination of steroid and diuretic as well as an intravenous steroid were ineffective. However, iron therapy for IDA was possibly effective 2 months later, since neither steroid nor diuretic was administered for a month preceding hearing loss.

Figure 1. Pure-tone audiometry from the onset of hearing loss: (A) Day 1; (B) Day 18; (C) Day 39; (D) Day 67.

Figure 2. The clinical course: Circles with continuous lines indicate mean PTA thresholds at 125, 250, and 500 Hz (left-sided ordinate); Triangles with continuous lines indicate serum Hb levels (right-sided ordinate); Arrow with dotted line indicates predictive increase to target Hb; Thick arrows indicate days when iron sucrose was administered intravenously; Thick black bars indicate when isosorbide was administered; Open bars indicate the periods and doses of each steroid therapy (PSL, prednisolone; B, betamethasone; HYD, hydrocortisone succinate).
Some researchers have described the association between ISSNHL and IDA, with a recent American survey of adults indicating a significant relationship between IDA and SNHL with increased odds of 1.82. A vascular mechanism has been proposed to explain the hearing loss due to IDA in that the cochlea is highly susceptible to ischemic damage, since only the labyrinthine artery supplies blood to this area. Another hypothesis is reactive thrombocytosis associated with IDA. Our patient presented with fluctuation and recurrence of hearing loss, and therefore it is unlikely that her hearing loss was associated with any stroke-like vascular mechanism. On the other hand, animal studies have shown that iron deficiency can influence cochlear changes by itself, including strial atrophy and a reduction of spiral ganglion cells, which can permanently impact the stereocilia of the inner and outer hair cells. However, our patient’s hearing loss was not irreversible. Elsewhere, Sun et al. reported significant improvements in clinical results using iron therapy in ISSNHL patients, supporting the possible efficacy of iron sucrose treatment in our patient.

No studies have shown the lesion sites or impaired frequencies in patients with IDA-associated SNHL. However, many studies have shown bilateral and unilateral SNHL at various frequencies in patients with sickle cell anemia (SCA). Onakoya et al. reported both bilateral (62%) and unilateral (left ear, 18%; right ear, 20%) SNHL in patients with SCA. While high-frequency SNHL was observed in most patients, low-frequency SNHL was observed in some patients, particularly in the right ear (5.6%). Low-frequency SNHL may be caused by diffuse damage to the cochlear organs rather than to the basal turn alone. These studies suggest that anemia, including IDA, can cause low-frequency SNHL only unilaterally, similar to Meniere’s disease.

IDA is highly prevalent in women of child-bearing age because of insufficient iron consumption to compensate for blood loss through menstruation. ALHL is also significantly prevalent in young female individuals. The shared etiology of both conditions supports the possibility that IDA is a key factor in our patient’s ALHL.

CONCLUSION
The clinical course of this patient suggests a possible association between ALHL and IDA. Serum Hb and iron levels should be examined for this type of SNHL, particularly in premenopausal females.

Informed Consent: Verbal informed consent was obtained from the patient.

REFERENCES