

# CASE REPORT: BILATERAL SEQUENTIAL SUDDEN HEARING LOSS IN A 52 YEAR OLD MAN

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## ABSTRACT

This report discusses a case of sequential, bilateral sudden sensorineural hearing loss in a 52 year-old man within the span of one week. He had a medical history of hypertension, diabetes mellitus type 2, and non-alcoholic steatohepatitis. His presentation contrasts the more commonly seen idiopathic, unilateral sudden sensorineural hearing loss. There was minimal improvement to his hearing loss bilaterally following three inner ear dexamethasone injections, a course of oral prednisone, hyperbaric therapy sessions, and methotrexate.

## INTRODUCTION

Sudden sensorineural hearing loss (SSNHL) is a pathological process of abrupt hearing loss within a few minutes or over a few days. Symptoms that patients experience include ear fullness, dizziness, and tinnitus and are generally unilateral. SSNHL is defined by a 30 dB hearing loss of three consecutive frequencies. The incidence of SSNHL is estimated to be 5 to 20 cases per 100,000 persons per year and most commonly seen in the 40-50 age range. Most cases of sudden sensorineural hearing loss are idiopathic, though 10% have an identifiable underlying cause. Only 5% of SSNHL is bilateral and typically associated with an underlying autoimmune mediated etiology. Bilateral SSNHL can be further categorized into a simultaneous loss of hearing (within 3 days), or sequential (3 to 30 days). Bilateral SSNHL is associated with more severe hearing loss and poorer prognosis and therefore timing of treatment is imperative. Reported cases of bilateral sudden hearing loss have been associated with conditions such as myelodysplastic syndrome and urticarial vasculitis. Infectious, tumoral, ototoxic, autoimmune, traumatic, and neurologic etiologies have also been sources of sudden hearing loss.

## ACKNOWLEDGEMENTS/REFERENCES

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Thank you to Dr. Bakos for encouraging me to write and present this case.

## CASE REPORT

A 52 year-old male with past medical history of hypertension, diabetes mellitus 2, non-alcoholic steatohepatitis (NASH) and no recent history of sickness or trauma presented to clinic with a sudden left side hearing loss that was noticed about 2 weeks prior to the visit. Symptoms included intermittent tinnitus that has been worsening, but denied associated dizziness. He had no changes in his medications. Audiometry revealed left sided sensorineural hearing loss in the low frequencies but unchanged hearing loss in the higher frequencies compared to a prior audiogram from a year ago (Figure 1 and 2). The previous audiogram demonstrated bilateral severe sensorineural hearing loss in the higher frequencies attributed to gradual sensorineural hearing loss with tinnitus. Patient returned one week later presenting with a new onset sudden right sided hearing loss with associated tinnitus. Hearing loss reported in the lower frequencies of the right side, indicating a sequential pattern. Audiometry showed right ear hearing was worse than left at the level of severe sensorineural hearing loss (Figure 3). Workup included MRI brain and CT head that were unremarkable. Bloodwork done 4 weeks after the hearing loss of the second ear showed IgG EBV Antibody >600.0 (negative <18.0), IgG EBV nuclear antigen antibody >600.0 (negative <18.0) and IgG CMV 1.7 (negative <0.6) where subsequent course of antiviral therapy was started of valacyclovir 1g TID for 7 days. RF and ANA antibodies were negative, ESR 4 (normal 0-30) and CRP <1 (normal: 0-10). The patient was initially prescribed a course of 10mg oral prednisone, but there was no subjective improvement in hearing at his follow-up appointment. Therefore, he underwent bilateral chemical labyrinthotomy with 10 mg/cc dexamethasone. In addition, hyperbaric oxygen therapy was recommended. After 1 month from the incident, patient had persistent hearing loss and tinnitus with no improvement seen on the audiogram (Figure 4) despite 3 doses of chemical labyrinthotomy and 17 hyperbaric oxygen therapy sessions. He was recommended to consult for bilateral hearing aids. He went to an academic center for further evaluation and recommended methotrexate, which did not improve his overall audiogram, but his word recognition score demonstrated improvement.

## RESULTS

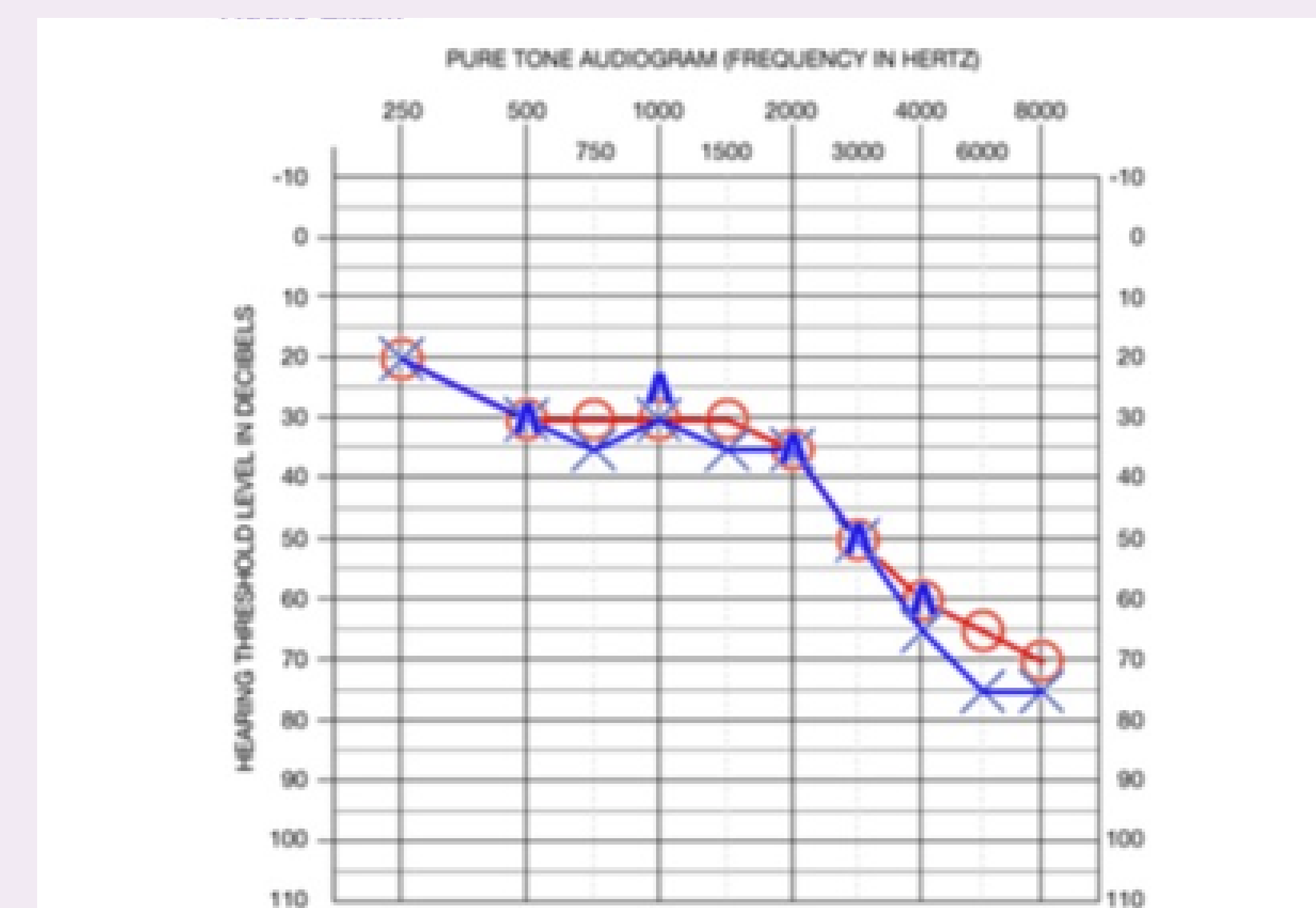


Figure 1: Baseline audiogram prior to SSNHL of left ear

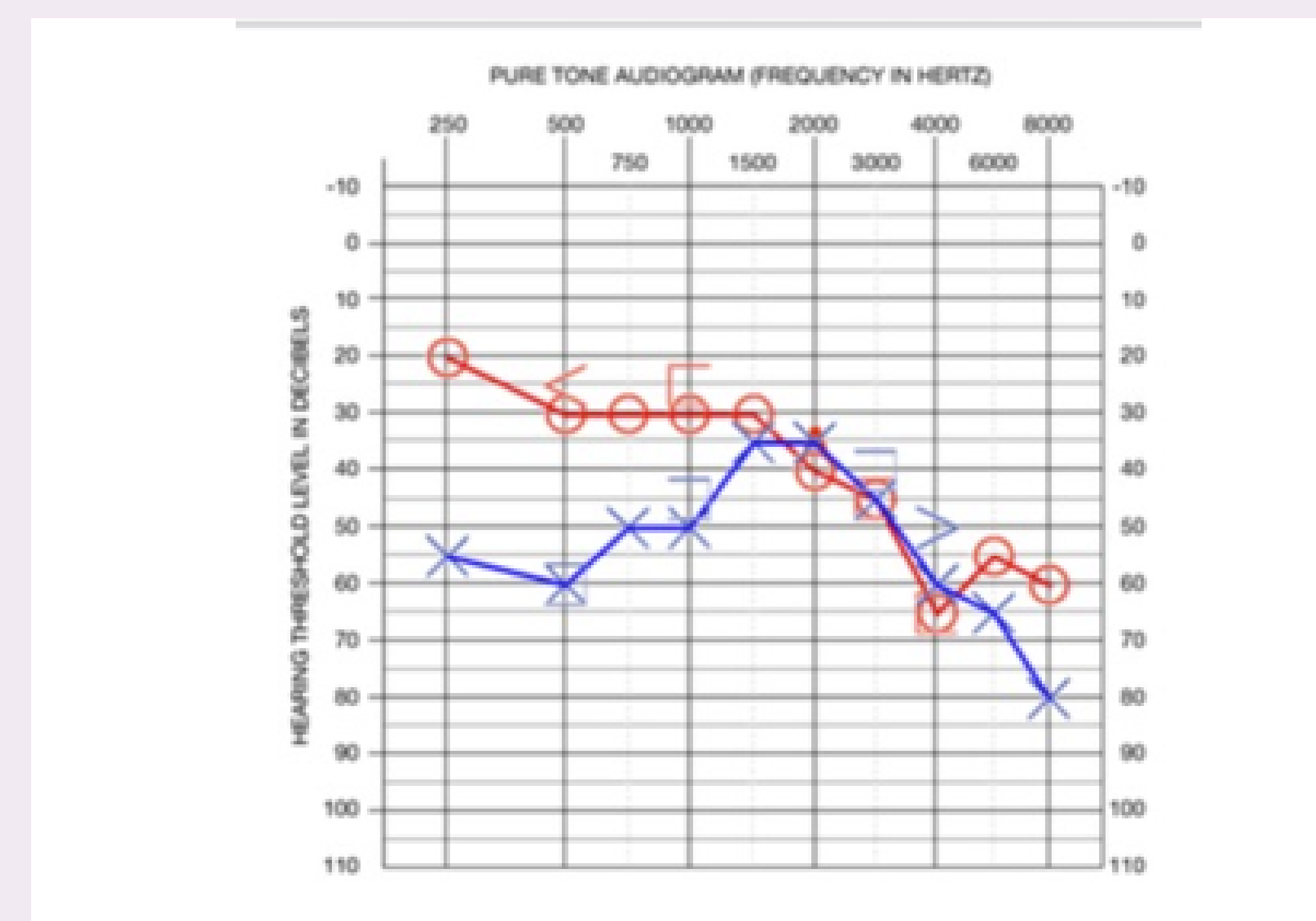


Figure 2: SSNHL of left ear

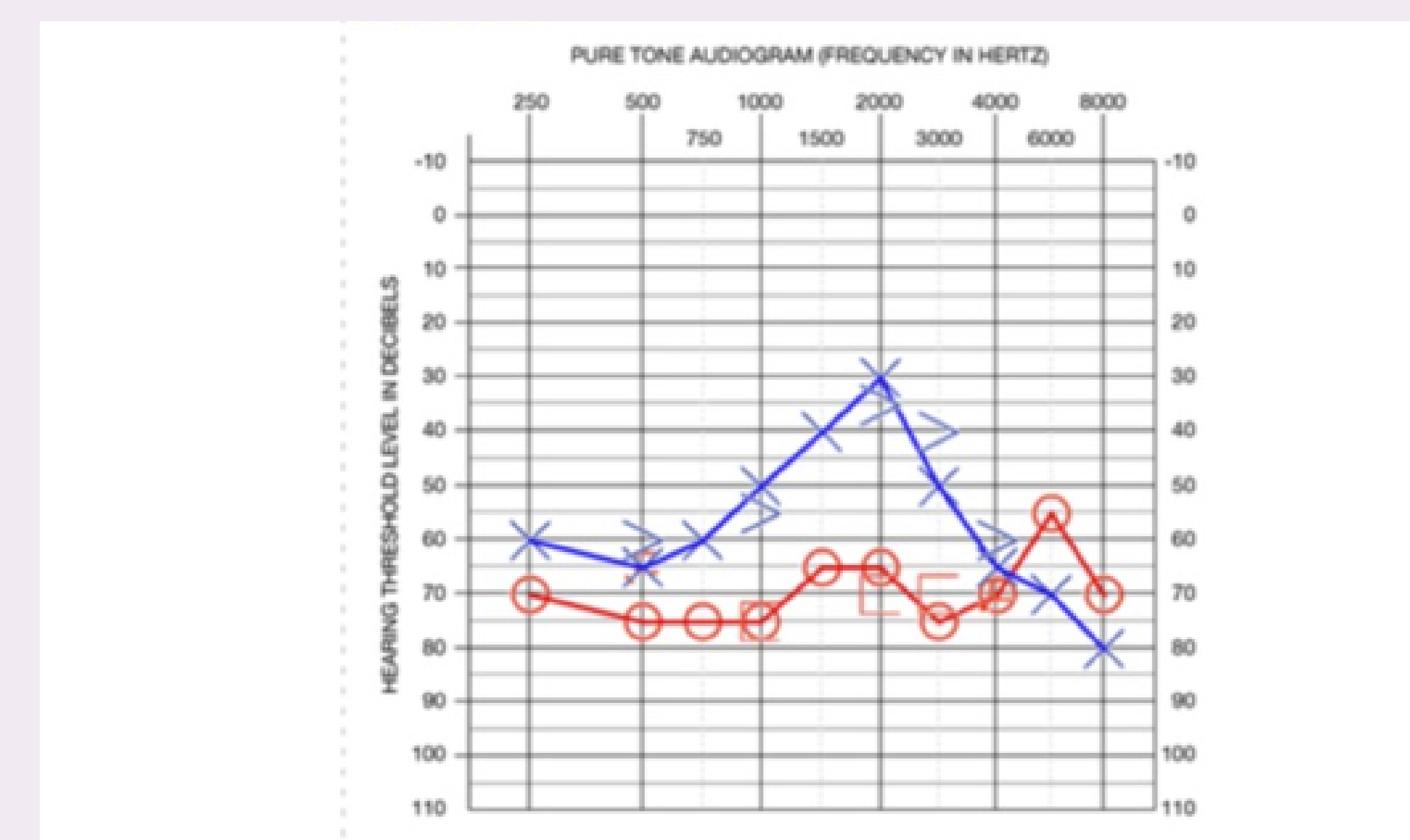


Figure 3: Sequential SSNHL of right ear one week after left ear SSNHL

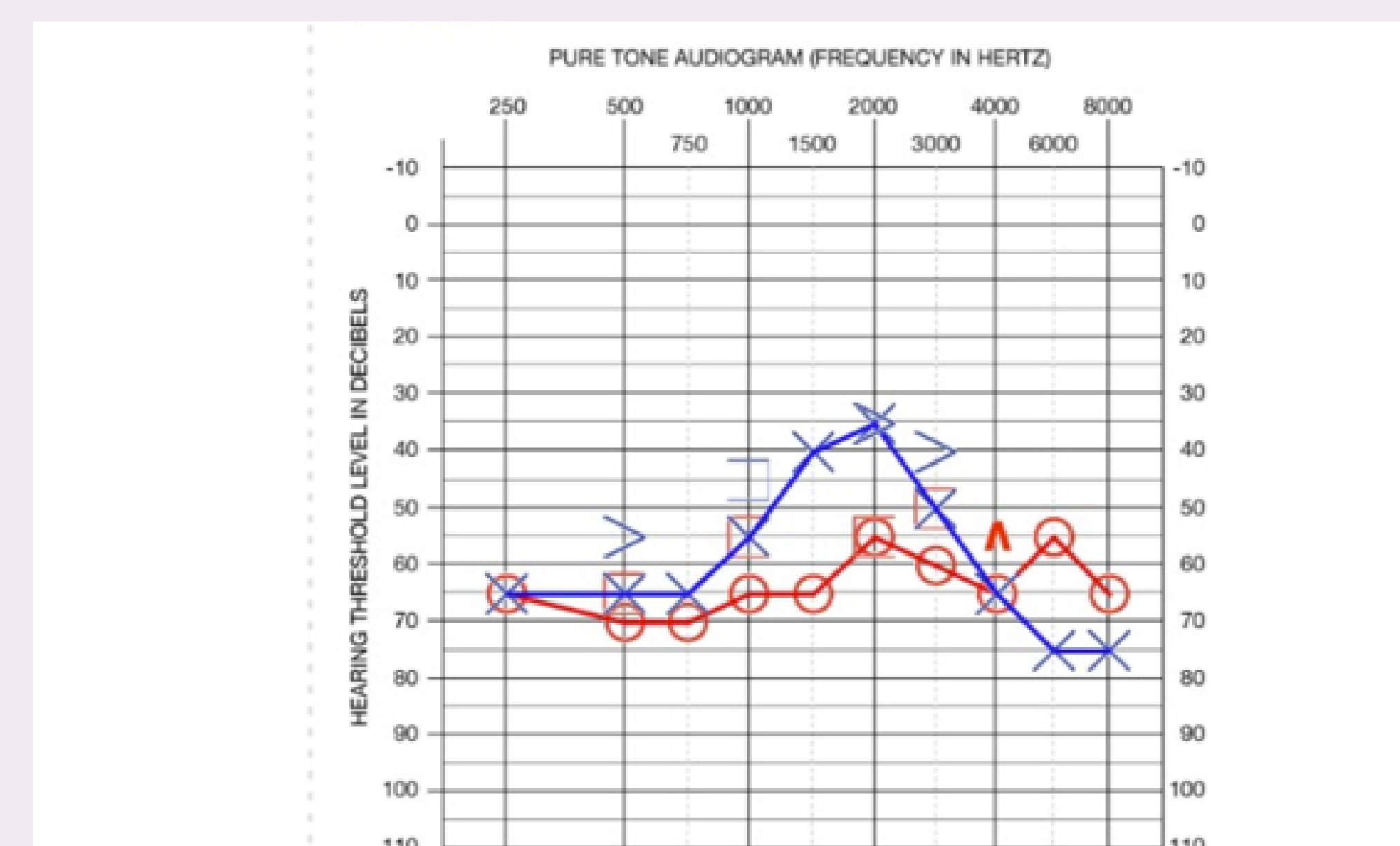


Figure 4: After 1 month from incident and multiple treatments, with persistent hearing loss

## DISCUSSION

Bilateral, sequential, idiopathic sudden sensorineural hearing loss is a rare patient presentation for hearing loss. Despite extensive treatment including: three courses of inner ear dexamethasone injections, oral prednisone, and hyperbaric oxygen therapy, our patient did not have improvement in their hearing. Bloodwork findings indicated a high count of IgG to EBV, indicating a primary or past infection of the virus. Alternatively, a high count IgG but a non-significant IgM value to CMV indicated an acute infection. There was no change to improvement of hearing loss due to prophylactic course of antiviral therapy. Autoimmune labwork was within the normal range, which negates an autoimmune etiology to the hearing loss. There may have been a confounding factor in that bloodwork was taken over 4 weeks after the latest incidence of sudden hearing loss and after steroid treatment where an opportune time window may have been missed to measuring inflammation markers.

A study recorded in *Disease markers* looked that how incidence of successive bilateral SSNHL could be predicted by measuring certain blood inflammatory metabolic parameters defined by bilateral hearing loss that occurred at least 1 year apart. In this retrospective study, these patients with successive bilateral SSNHL had higher rates of low HDL, high LDL, diabetes, fibrinogen measurements, whilst interestingly there was no associated significance in patterns of hypertension. In addition, therapeutic efficacy was lower in these patients than those with unilateral sudden hearing loss. It is thought that systemic stress could cause a chronic inflammation that increased risk for successive bilateral SSNHL.

The patient in our case report had a medical history of NASH that can result in higher LDL and lower HDL levels, as well as with diabetes mellitus. Etiologies considered in this pathology include microcirculatory failure which can be metabolic disorder associated, and prothrombotic susceptibility that leads to ischemic changes in the inner ear leading to loss of hearing.

Bilateral sudden hearing loss and tinnitus relating to salicylate intoxication, myelodysplastic syndrome, and urticarial vasculitis have also been documented.

## CONCLUSION

For our patient, he fit the age demographic as well as had a systemic medical history of inflammatory states such as hypertension, diabetes mellitus, and NASH that could contribute to lipid abnormalities. Bilateral SSNHL also is associated with poorer therapeutic efficacy, which is consistent with this patient's outcome to treatment, even if they were administered in a timely manner.

However, with blood work, there was no indications of active inflammation indicated by acute phase proteins CRP or ESR. There was also no lipid panel available and could not verify specific lipid levels, although a diagnosis of NASH would point to elevated LDL and lower levels of HDL. Thus, direct etiology of this patient's bilateral sequential sensorineural hearing loss was not known.

This case report can be an addition to the less reported cases of sudden bilateral sensorineural hearing loss. Even without a defined cause of this patient's hearing loss, his medical history is consistent with a chronic inflammatory state that has been proposed as an increased risk of bilateral SSNHL by previous authors.