

Spontaneous Recovery of Sudden Sensorineural Hearing Loss: Possible Association with Autoimmune Disorders

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Abstract

A 46-year-old white male diagnosed with systemic lupus erythematosus (SLE) and antiphospholipid syndrome (APS) was seen for audiological testing 15 minutes following a sudden onset hearing loss in the right ear. The test battery included pure-tone audiometry, word-recognition testing, speech-recognition threshold (SRT) testing, immittance testing, and distortion-product otoacoustic emissions (DPOAE) testing. Testing revealed a sensorineural hearing loss in the right ear. Shortly after testing, the patient indicated that his condition had improved. Testing was repeated, and the second round of tests revealed normal hearing in both ears. Four days later, a follow-up test again indicated normal hearing in both ears. Possible connections of this brief occurrence of idiopathic hearing loss with the patient's medical conditions are discussed. Specifically, symptoms were consistent with a transient ischemic attack (TIA) affecting his right cochlea in the stria vascularis region, resulting in a temporary, sensorineural hearing loss. No residual effects were observed clinically.

Key Words: Antiphospholipid antibody disorder, antiphospholipid syndrome, sensorineural hearing loss, systemic lupus erythematosus

Abbreviations: AAD = antiphospholipid antibody disorder; ANA = antinuclear antibodies; aPL = antiphospholipid antibodies; APS = antiphospholipid syndrome; CBC = complete blood count; CID = Central Institute for the Deaf; DPOAE = distortion-product otoacoustic emissions; IgG/IgM = immunoglobulin; OAE = otoacoustic emission; OHC = outer hair cell; PI-PB = performance intensity—phonetically balanced; PTA = pure-tone average; SLE = systemic lupus erythematosus; SNR = signal-to-noise ratio; SRT = speech-recognition threshold; TIA = transient ischemic attack

Sumario

Una mujer de 46 años de edad, diagnosticada con un lupus eritematoso sistémico (LES) y un síndrome antifosfolipídico (APS) fue evaluada audiológicamente 15 minutos después de sufrir de una pérdida súbita de audición en el oído derecho. La batería de pruebas incluyó audiometría de tonos puros, pruebas de reconocimiento de palabras, prueba de umbral de reconocimiento del lenguaje (SRT), impedanciometría y emisiones otoacústicas por productos de distorsión (DPOAE). La evaluación reveló una hipoacusia sensorineural en el oído izquierdo. Poco después de la evaluación, la paciente indicó que su condición había mejorado. Se repitieron las pruebas, y en la segunda ronda los estudios revelaron una audición normal en ambos oídos. Cuatro días después, una evaluación de seguimiento volvió a mostrar resultados normales en ambos oídos. Se discuten posibles conexiones de este breve

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episodio de hipoacusia idiopática en una paciente con las condiciones médicas descritas. Específicamente, se discuten los síntomas son consistentes con un ataque isquémico transitorio (ITA) que afectó la estría vascular de la cóclea derecha, produciendo una hipoacusia sensorineural temporal. No se observaron efectos residuales clínicamente.

Palabras Clave: Trastorno de anticuerpos anti-fosfolipídicos, síndrome anti-fosfolipídico, hipoacusia sensorineural, lupus eritematoso sistémico

Abreviaturas: AAD = trastorno anti-fosfolipídico; ANA = anticuerpo antinuclear; aPL = anticuerpo anti-fosfolipídico; APS = síndrome anti-fosfolipídico; CBC = conteo sanguíneo completo; CID = Instituto Central para el Sordo; DPOAE = emisiones otoacústicas por productos de distorsión; IgG/IgM = inmunoglobulina; OAE = emisión otoacústica; OHC = células ciliadas externas; PI-PB = intensidad-desempeño—balance fonético; PTA = promedio tonal puro; SLE = lupus eritematoso sistémico; SNR = tasa señal-ruído; SRT = umbral de reconocimiento del lenguaje; TIA = ataque isquémico transitorio

Systemic lupus erythematosus (SLE) is a chronic, inflammatory autoimmune disorder that typically affects multiple organs of the body. It results from the immune system's inability to distinguish between the antigens that attack the body and the body's own cells and tissues. Consequently, the immune system deploys antibodies against its own tissues. These "auto-antibodies" react with body tissues to form immune complexes that accumulate in tissues and organs causing inflammation. The reported prevalence of systemic lupus erythematosus in the United States is 40 to 50 cases per 100,000 (Lawrence et al, 1998) and is more prevalent in women than in men.

Patients with SLE can manifest a myriad of symptoms relating to the body's inflammatory response, including painful joints, fever, butterfly rash, chest pain with deep breathing, hair loss, Raynaud's syndrome (a syndrome that causes arterial contraction in fingers and toes), photosensitivity, edema in legs or around eyes, mouth ulcers, swollen glands and extreme fatigue. Several investigations such as antinuclear antibody (ANA) test, complete blood count (CBC), urinalysis, chest X-rays, and neurological examination need to be performed to determine any coexisting conditions affecting the renal, neurologic, hematologic, or immunologic systems. The diagnosis of SLE is based on the results of these tests together with the clinical symptoms (Tan et al, 1982). The clinical manifestation of SLE is highly variable as it may develop insidiously and exist as a chronic

condition with the patient alternating between periods of total remissions and flare ups. In its more serious form, SLE may even result in death. Although no cure has been developed for the disease, the prognosis for SLE patients today is much better with the advent of new medications and treatment strategies (Luckmann and Sorensen, 1980).

A condition that commonly occurs in SLE patients is antiphospholipid syndrome (APS) or antiphospholipid antibody disorder (AAD). It is an autoimmune disorder that can occur by itself (primary APS) or in conjunction with another disorder like SLE (secondary APS). Antiphospholipid antibodies (aPL) are a heterogeneous group of autoantibodies that are deployed by the human body against its own tissues. In APS, these antibodies target negatively charged phospholipids, a lipid containing phosphorus which is a basic part of a cell membrane, and serum phospholipid-binding proteins that are proteins that assist in the transfer of phospholipids between cellular membranes. Although various theories have been proposed to explain how aPL causes clotting in a blood vessel, or thrombosis, the exact mechanism remains unknown. aPL has been known to interfere with fibrinolysis and anticoagulant pathways in the body besides affecting cells like platelets and monocytes (Cuadrado and Lopez-Pedrerá, 2003). aPL occurs in about 1% of the general population (Juby and Davis, 1998). However, not all patients with aPL develop symptoms of APS. Approximately 30–40% of SLE patients are aPL positive, and about 50% of them are likely to develop

thrombosis within 10–20 years (Petri, 2000; Shah et al, 1998).

Patients suffering from this disorder show symptoms related to the occurrence of thrombosis. Clinical criteria for diagnosis of APS include thrombosis (venous, arterial, or small vessel) or pregnancy morbidity whereas laboratory criteria include anticardiolipin antibody: immunoglobulins (IgG and/or IgM) or lupus anticoagulant (Wilson et al, 1999). At least one clinical and one laboratory criteria cited above have to be met in order to confirm the diagnosis of APS (Cuadrado and Lopez-Pedraza, 2003). The most sensitive test for aPL detection is a solid-phase immunoassay for aPL (Harris et al, 1983). Treatment usually involves oral anticoagulant therapy or aspirin in asymptomatic cases. The prognosis for APS depends on the occurrence of thrombosis and their localization in the body. Clinical features such as pulmonary hypertension, neurological involvement, ischemic heart disease, and renal impairment are indicative of poor prognosis (Amigo, 2001). Mortality rate in SLE patients is higher if they also suffer from APS (Cuadrado and Lopez-Pedraza, 2003).

The prevalence of auditory symptoms in SLE has generated limited interest among researchers. Hamblin et al (1982) reported that a patient with SLE and severe sensorineural hearing loss experienced auditory improvements with plasma exchange, suggesting a vascular mechanism to the hearing loss. Caldarelli et al (1986) posited vasculitis, an inflammation of blood vessels resulting from immune complexes depositing on the vascular wall, as the etiology for a patient with severe, bilateral, sensorineural hearing loss who was later diagnosed with SLE. In a study of 30 SLE patients, Bowman et al (1986) suggested an autoimmune mechanism likely explained the sensorineural hearing loss exhibited by two patients with otherwise unexplained hearing loss. Andonopoulos et al (1995) reported that a significant number of SLE patients who exhibited no auditory symptoms had subclinical sensorineural hearing loss due to autoimmune pathogenesis. Although their patients with SLE had significantly higher thresholds than the normal group, they could not correlate this finding with specific physiologic changes. Andonopoulos et al (1995) discussed immunologic theories and vasculitis as possible causes. Hisashi et al (1993) were the first to suggest a link between

sensorineural hearing loss in patients with SLE and aPL. They postulated that aPL induce thrombosis in the peripheral auditory system in patients with SLE resulting in sensorineural hearing impairment. Compadretti et al (2005) report findings in a patient that support their hypothesis.

CASE HISTORY

Our patient first sought medical intervention for recurrent pain in his right knee at 36 years of age. It was diagnosed as deep vein thrombosis involving the right popliteal fossa and was treated with warfarin, an anticoagulant. At the time of the initial diagnosis, he also presented with a history of arthritic type pain and joint stiffness treated via ibuprofen. Four years later at 40 years of age, he injured his hip in a fall, and blood reports taken during the treatment for his injury revealed an abnormally low platelet count (thrombocytopenia). Detailed investigations revealed the presence of circulating lupus anticoagulant and positive aPL, IgG type, suggestive of APS. Over the next five years, he was treated with a regimen of warfarin and prednisone to control hypercoagulability, an abnormal increase in the ability to form blood clots, and the low-platelet count, respectively. Subsequently, he underwent splenectomy. In people with APS, the removal of the spleen usually has the effect of increasing the platelet count (Galindo et al, 1999). He was weaned off prednisone over the next four months as his platelet count improved from the surgery. At this time, he tested positive for ANA (1:320; normal \leq 1:20) and aPL ($>$ 150; normal \leq 20 GPL). ANA are antibodies found in individuals with SLE and are therefore necessary but not sufficient to diagnose a person with SLE. The patient was diagnosed with APS secondary to SLE based on his clinical presentation, case history, and laboratory tests.

Approximately one year later at 46 years of age, the patient experienced an episode of hearing loss in his right ear for which he sought diagnosis. He reported having periodic audiological evaluations in order to monitor his hearing related to occupational concerns. He had no concerns about his hearing until this particular event. The patient works in a field requiring acute attention to sounds and has a strong occupational interest in protecting his

hearing. History of noise exposure was denied, and the patient reported wearing earplugs any time he expects even moderate amounts of noise exposure (e.g., mowing the lawn.) Our patient sought audiological testing five minutes after suffering a significant event without provocation. He complained of significant hearing loss, a plugged feeling, and distortion in his right ear with no change in his left ear. Dizziness was denied. An audiological evaluation was carried out within 15 minutes of the incident. At the time of the episode, the patient reported that he was currently taking warfarin and losartan (for hypertension) as prescribed and acetaminophen as needed for headaches.

METHOD

Pure-Tone Audiometry

Pure-tone audiometry was carried out (Grason-Stadler GSI 61 with Etymotic ER-3a) in a sound-treated booth (Interacoustics Corporation). Conventional pure-tone air- and bone-conduction threshold techniques were used to establish thresholds. Testing was carried out for octave frequencies from 250 Hz to 8000 Hz and interoctave frequencies of 3000 Hz and 6000 Hz. Thresholds measured at 25 dB HL or better were considered normal.

Speech Audiometry

Speech-recognition thresholds (SRT) were obtained using spondee words in the same sound-treated chamber. Pure-tone average (PTA) and SRT within ± 6 dB were considered to be in agreement. Word-recognition scores were obtained using the Central Institute for the Deaf (CID) W-22 word list presented at 60 dB HL (unless otherwise noted) from a recording using contralateral masking. The performance intensity-phonemically balanced (PI-PB) function was obtained by presenting the words at 70, 80, and 90 dB HL with contralateral masking.

Immittance Audiometry

Tympanograms and acoustic reflexes

were derived in a conventional manner (Grason Stadler, GSI Tymstar Middle Ear Analyzer). Acoustic reflexes were measured at 500 Hz, 1000 Hz, 2000 Hz, and 4000 Hz both ipsilaterally and contralaterally.

Otoacoustic Emissions

Distortion-product otoacoustic emissions (DPOAE) were measured using the screening protocol (Biologic Systems Corp., Scout Otoacoustic Emissions System v.3.45). Levels for L1 and L2 were 65 and 55 dB SPL, respectively, with an F1 to F2 ratio of 0.8. F2 frequencies ranged from approximately 2000 Hz to 5000 Hz, bilaterally. A minimum 6 dB signal-to-noise ratio (SNR) was used to determine a normal otoacoustic emission (OAE). For a given F1 and F2, the response was considered to be generated at the F2 place (Kummer et al, 1995).

RESULTS

Four sets of audiometric results were collected: pre-episode, during the episode, within 15 minutes after the episode, and a four-day follow-up. In all cases, all the tests listed above were performed with two exceptions. First, DPOAEs for the unaffected ear were not measured during the recovery phase of the episode. Second, the PI-PB function was measured only during the episode.

The patient had normal hearing at the time of his pre-episode hearing test. These thresholds, shown in Figure 1, were measured three months prior to his episode. The PTA and SRT were in agreement. Immittance audiometry revealed type-A tympanograms and normal acoustic reflexes for all frequencies tested bilaterally. Word-recognition scores were measured at 40 dB HL and were 100% for both ears.

An audiometric evaluation was performed shortly after the episode began. Figure 2 shows the thresholds obtained during his episode. The right ear had normal to moderate hearing loss with a sensorineural notch at 4000 Hz while the left ear exhibited normal hearing at all frequencies tested. PTA and SRT were in agreement. Word-recognition scores were 100% and 76% for the left and right ears, respectively. Roll over was absent. Immittance audiometry revealed

type A tympanograms bilaterally. Acoustic reflexes were within normal limits bilaterally for all frequencies tested. While left ear DPOAE results were normal, right ear results were abnormal as shown in Figure 3. For 2953, 3891, and 4875 Hz F2 frequencies, the SNR was at or below 2.1 dB. The only F2 frequency that revealed a normal OAE was 1969 Hz, which was measured at 17.8 dB SNR.

After our audiological assessment was complete, the patient reported that the plugged up feeling and distortion in his right ear had disappeared. The patient's hearing was reevaluated immediately, repeating pure-tone and speech audiometry. DPOAE testing was repeated in the right ear only. Pure-tone audiometry revealed hearing sensitivity within normal limits bilaterally as shown in Figure 2. PTA and SRT were in agreement. Word-recognition scores were 100% in both ears. Figure 3 shows the right-ear DPOAE results, which were normal. Of the four frequencies tested in the right ear, the minimum SNR was 8.1 dB.

A follow-up evaluation was performed four days after the episode. Figure 4 shows pure-tone thresholds from this evaluation. PTA and SRT were in agreement. Immittance results were also normal in both ears. Finally, as shown in Figure 3, DPOAEs were present at all frequencies tested.

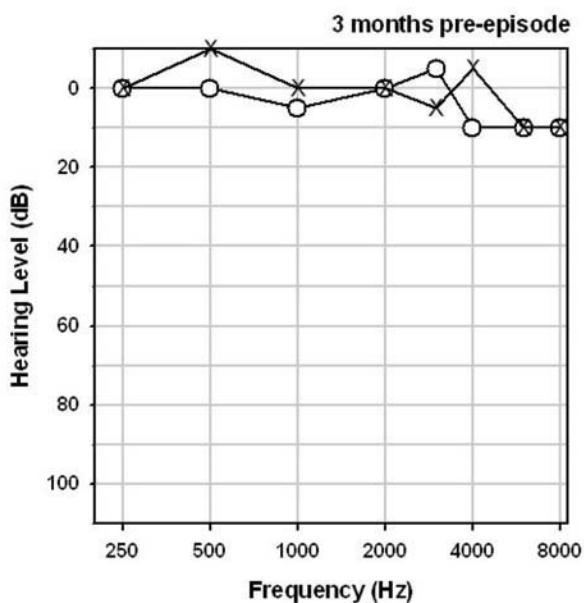


Figure 1. Audiometric thresholds measured three months before the episode.

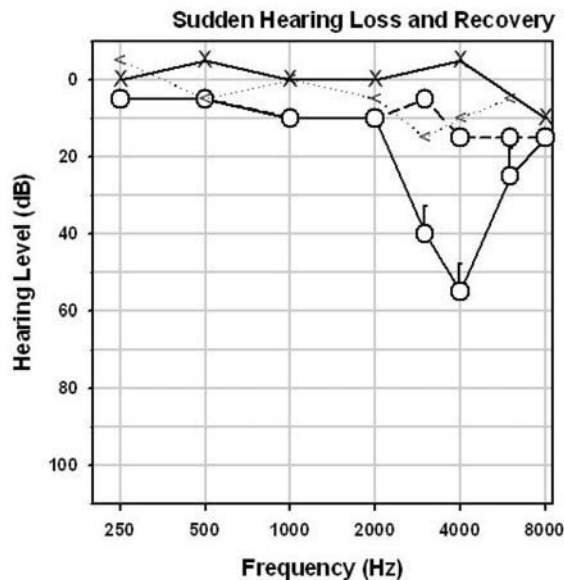


Figure 2. Audiometric thresholds measured during the sudden hearing loss and after the recovery of hearing. Thresholds measured in the right ear at the time of the sudden hearing loss, represented by the solid line and open circles. After the hearing loss was reported to have disappeared, air and bone conduction was repeated within 15 minutes of the first audiogram. The repeated right-ear thresholds are represented by the dashed line and open circles

DISCUSSION

Test results and patient report suggest a transient sensorineural hearing loss in the right ear. The time-course of events, from the initial symptoms, to audiometric testing, to the reevaluation, suggested that the episode lasted between 20 and 40 minutes. Given the patient's occupational interest in protecting his hearing and avoiding noise exposure, and the brevity of the episode, it is highly unlikely that this hearing loss was caused by noise exposure. It is likely that the hearing loss was cochlear in origin as the DPOAEs were absent when behavioral thresholds were abnormal and present when normal behavioral thresholds were restored (Brownell, 1990).

As reviewed, the patient currently suffers from SLE with a secondary APS. Given the rarity of a short-term hearing loss in general that is not noise induced, it is plausible that one of his medical conditions is involved. Therefore, SLE and APS with regards to their possible impact on hearing will be discussed.

Auditory symptoms in SLE without a diagnosis of APS have been reported in a number of studies (Hamblin et al, 1982;

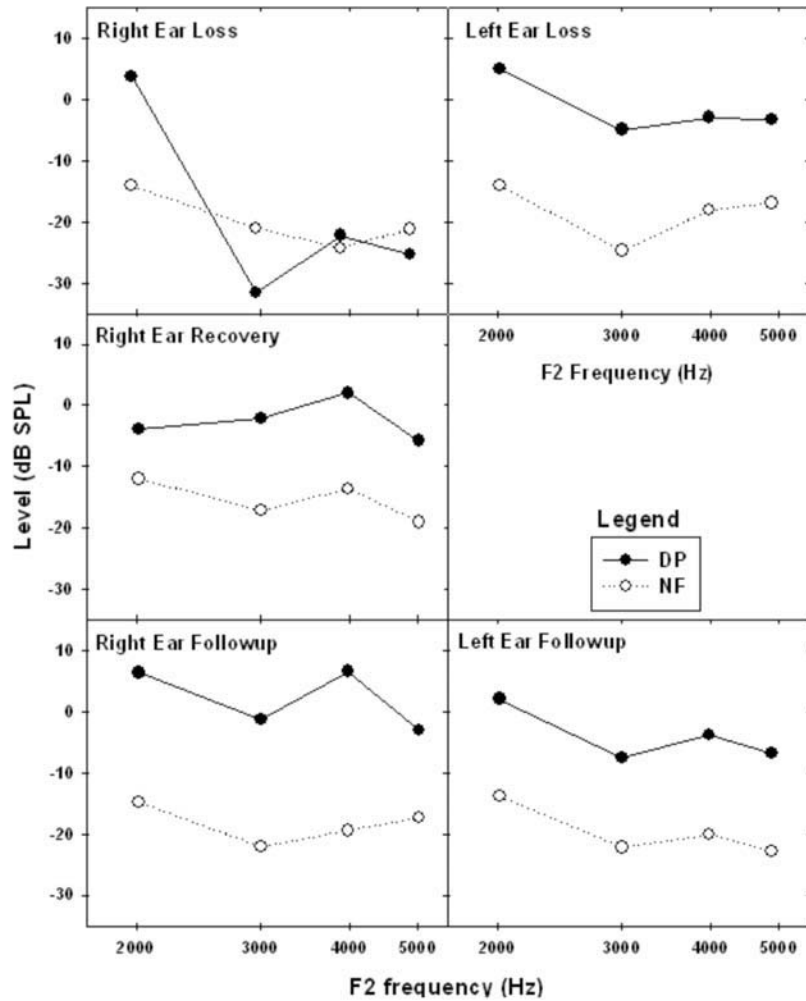


Figure 3. Each panel shows a distortion-product otoacoustic emission test result. The tests were performed during, immediately after, and four days after the episode. DP = distortion product; NF = noise floor.

Bowman et al, 1986; Caldarelli et al, 1986; Andonopoulos et al, 1995; Sperling et al, 1998; Peeva and Barland, 2001; Kastanioudakis et al, 2002). Most of these studies did not control for aPL levels that are found in APS. A greater proportion of SLE patients have sensorineural hearing loss than in the general population (Kastanioudakis et al, 2002; Kochkin, 2005).

One vascular mechanism suspected to cause hearing loss in SLE patients involves antigens and/or genetic material in the bloodstream (Harris et al, 1984; Sone et al, 1999). Vasculitis is an ischemic condition caused by an inability of these materials to pass through blood vessels. In instances of hearing loss, blood vessels feeding the small vascular region of the inner ear, namely the stria vascularis, are affected (Harris et al, 1984; Sone et al, 1999). While the reported studies

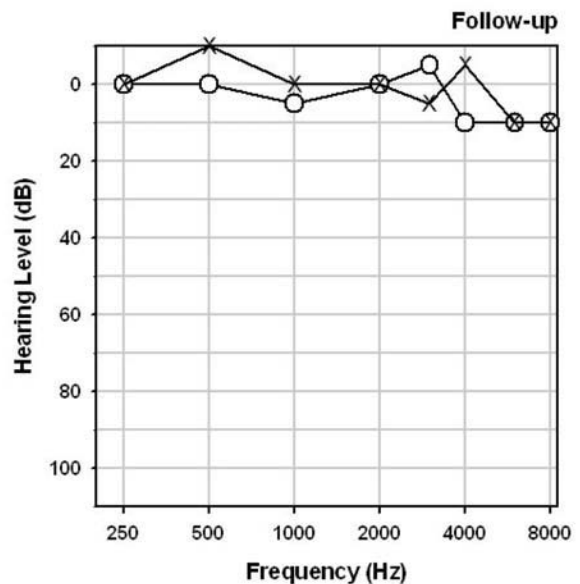


Figure 4. Audiometric thresholds measured four days after the episode.

of SLE and hearing loss discussed debris in the blood vessels or vasculitis as the immediate cause of the hearing loss, there are no documented reports of a spontaneous recovery.

SLE is known to manifest itself with temporary symptoms or flare-ups. The time-course for these flare-ups varies in length from days, to weeks, to even months. Our patient reported several symptoms lasting from 20 to 40 minutes which is inconsistent with the typical hearing loss caused by SLE. Therefore, it is unlikely that SLE was the primary cause for our patient's temporary hearing loss.

Recall that the medical diagnosis of APS involves a combination of at least one positive laboratory and one clinical criterion. The laboratory diagnosis for APS includes a medium or high titer of an anticardiolipin antibody as well certain clinical criteria (e.g., vascular thrombosis, miscarried pregnancy) (Cuadrado and Lopez-Pedraza, 2003). It is possible for patients to present with significant levels of aPL without being diagnosed with APS. Therefore, the discussion will be limited to increased aPL regardless of the APS diagnosis for documented occurrences of hearing loss.

There are several documented reports of hearing loss in persons with raised aPL levels with or without SLE and/or APS (Asherson et al, 1987; Vyse et al, 1994; Toubi et al, 1995; Naarendorp and Spiera, 1998; Shah et al, 1998; Green and Miller, 2001). Symptoms associated with APS often last for a short period of time and include transient sensory, cognitive, balance, and memory disruptions with or without headaches, motor weakness, and muscle pain. The mechanism for this transient, localized symptom is thought to be transient ischemic attacks (TIA), a temporary interruption in blood flow due to some obstruction in a blood vessel. The presence of anticardiolipin antibodies associated with APS has been correlated with thrombosis which can cause TIAs. (Harris et al, 1983; Harris et al, 1984; Harris et al, 1985; Hisashi et al, 1993). TIAs typically last 2–15 minutes but do not last longer than 24 hours (Whisnant et al, 1990). This fits well with the time-course of our patient's 20 to 40 minute episode.

The stria vascularis has fine vascular structure and could easily be affected by a TIA. An attack on the stria vascularis would cause a decrease in the endolymph ionization. A decrease of this sort would lead to a shortage of metabolic resources required for normal outer hair-cell function and, therefore, normal auditory

thresholds. This would manifest as a sensorineural hearing loss. This does not necessarily cause trauma to the outer hair cells (OHCs). Therefore, if the endolymph ionization returned to normal, OHC function and, therefore, auditory thresholds would return to the condition prior to the attack.

In our search, we found no documented reports of hearing loss caused by TIAs. This is not surprising, however, given the short time-course of TIAs. Also, people suffering from TIAs may be less likely to report a hearing loss similar to that of our patient. This may be because a hearing loss lasting less than an hour would not be as disturbing to a patient as a loss of sight or cognitive function. Our patient was able to detect the subtle change in his hearing immediately and approached us for intervention. The rapid progression of his hearing loss and an equally rapid recovery seems to rule out vasculitis as the underlying mechanism. Also, the role of APS in this patient cannot be ignored given the transient nature of hearing loss associated with this condition. If our patient's medical condition were involved, one can speculate the hearing loss probably resulted from a TIA affecting the cochlea. This hypothesis supports the findings of Hisashi et al (1993) and other researchers who found an association between sensorineural hearing loss in SLE patients and aPL. The patient's hearing was reevaluated four days later, and all reports were normal. This is a rare case considering the complete and immediate nature of recovery exhibited by the auditory system. Given that the audiometric test results returned to pre-episode performance, any residual damage is subclinical.

In the strictest sense, this is a case of idiopathic hearing loss with a quick, spontaneous recovery. However, the etiology may be related to the patient's medical conditions. If this were the case, the increase in aPL in his blood, the time-course of TIAs, and the time-course of this hearing loss together suggest a TIA in a blood vessel serving the stria vascularis, thereby temporarily offsetting the ion balance in the endolymph near the region effected.

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