

Sildenafil-Associated Sudden Hearing Loss: A Case Report

Nagła utrata słuchu związana z sildenafiliem: opis przypadku

Authors' Contribution:

A – Study Design
B – Data Collection
C – Statistical Analysis
D – Data Interpretation
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ABSTRACT:

Introduction: Sudden sensorineural hearing loss (SSNHL) is a distressing medical emergency characterized by a rapid onset of hearing loss in one or both ears over a period of 72 hours or less. With the multitude of etiologies, patients are often left with significant auditory deficits that change quality of life, affecting communication, engagement with social activities, and performance of daily tasks. This case report explores a potential link between sildenafil, a commonly prescribed medication for erectile dysfunction, and SSNHL, shedding light on the ototoxic risks associated with its use.

Case report: We report the case of a 29-year-old male who presented with tinnitus and a sudden, severe hearing loss in his right ear. Initial symptoms were managed in an emergency room in Spain, where he was prescribed Dexona (30 mg). Prior to this onset, he had taken sildenafil and dapoxetine. Audiometric evaluations revealed profound hearing loss in the right ear with a threshold of 90 dB at 500 Hz and 100 dB at 1000–2000 Hz, while the left ear maintained normal hearing levels. Electrophysiological tests confirmed the severity of the hearing impairment in the right ear. Despite comprehensive audiological and vestibular assessments and treatment at our clinic, including steroids and hyperbaric oxygen therapy, there was no significant improvement.

Conclusions: This case highlights the potential ototoxic effects of sildenafil, suggesting it may contribute to sudden and profound SSNHL. The absence of vestibular involvement points towards a cochleotoxic mechanism. Considering the widespread use of sildenafil, further research is imperative to fully understand its impact on auditory function and to establish guidelines for safer use. This report highlights the need for clinicians (not only audiologists/laryngologists) to be vigilant about the potential auditory side effects of sildenafil, especially in patients presenting with sudden hearing loss.

KEYWORDS:

audiology, ototoxicity, sildenafil, sudden sensorineural hearing loss

STRESZCZENIE:

Wprowadzenie: Nagły niedostuch odbiorczy (ang. *sudden sensorineural hearing loss*; SSNHL) jest niepokojącym, nagłym stanem, który charakteryzuje się szybkim wystąpieniem utraty słuchu w jednym lub obu uszach w odstępie 72 godzin lub krótszym. Przy mnogości możliwych etiologii, u pacjentów często utrzymują się znaczne deficyty słuchowe, które zmieniają jakość życia, co wpływa na możliwość komunikacji, zaangażowanie w działania społeczne i wykonywanie codziennych zadań. W niniejszym opisie przypadku zbadano potencjalny związek między sildenafiliem, powszechnie przepisywanym lekiem na zaburzenia erekcji, a SSNHL i rzucono światło na ryzyko ototoksycznego działania leku.

Opis przypadku: W pracy przedstawiono przypadek 29-letniego mężczyzny, u którego wystąpiły szumy uszne i nagła, poważna utrata słuchu w prawym uchu. Początkowe objawy zostały opanowane na oddziale ratunkowym w Hiszpanii, gdzie pacjentowi przepisano lek Dexona (30 mg). Uprzednio pacjent przyjmował sildenafil i dapoksetynę. Oceny audiometryczne wykazały głęboki ubytek słuchu w prawym uchu, z progami 90 dB przy 500 Hz i 100 dB przy 1000–2000 Hz, z zachowaniem normalnego poziomu słuchu w uchu lewym. Nasilenie upośledzenia słuchu w uchu prawym zostało potwierdzone w badaniach elektrofizjologicznych. Pomimo kompleksowej oceny audiologicznej i oceny czynności przedsionkowej oraz zastosowanego leczenia w klinice autorów, w tym sterydoterapii i hiperbarycznej terapii tlenowej, nie uzyskano znaczącej poprawy.

Wnioski: Opisany przypadek zwraca uwagę na potencjalne ototoksyczne działanie sildenafilu, co sugeruje, że lek ten może przyczyniać się do wystąpienia nagłego i głębokiego SSNHL. Brak wpływu na czynność przedsionkową wskazuje na mechanizm kochleotoksyczny. Z uwagi na powszechne stosowanie sildenafilu konieczne jest przeprowadzenie dalszych badań, które pozwolą w pełni zrozumieć jego wpływ na funkcje słuchowe i ustalić wytyczne w zakresie bezpieczniejszego stosowania tego leku. Przedstawiony opis przypadku podkreśla potrzebę zachowywania przez klinicystów (nie tylko audiologów/laryngologów) czujności w odniesieniu do potencjalnych słuchowych działań niepożądanych sildenafilu, zwłaszcza u pacjentów zgłaszających się z nagłą utratą słuchu.

SŁOWA KLUCZOWE: audiologia, nagły niedosłuch odbiorczy, ototoksyczność, sildenafil

ABBREVIATIONS

BERA – Brain Evoked Response Auditory
cGMP – cyclic guanosine monophosphate
CP – canal paresis
CRP – C-reactive protein
CT – computed tomography
DP – directional preponderance
DP-OAE – acoustic otoemission
ECG – electrocardiography
FDA – Food and Drug Administration
HBOT – Hyperbaric Oxygen Therapy
ION – ischemic optic neuropathy
JADER – Japanese Adverse Drug Event Report
MAP – mitogen-activated protein
NF- κ B – nuclear factors-kappa beta
NO – nitric oxide
PDE – phosphodiesterase
PDE11 – phosphodiesterase type 11
PDE5 – phosphodiesterase type 5
PDE5I – phosphodiesterase type 5 inhibitors
PDE6 – phosphodiesterase type 6
PT – preferred terms
RVO – retinal vascular occlusion
SRD – serous retinal detachment
SSNHL – sudden sensorineural hearing loss
SSRI – selective serotonin reuptake inhibitor

INTRODUCTION

Sudden deafness, characterized by sudden-onset sensorineural hearing loss, can manifest with varying degrees of severity, more than 30 dB or at least 3 contiguous audiometric frequencies occurring within 72 hours, and may involve inner ear and/or central auditory pathways [1]. The etiology of the majority of cases of sudden sensorineural hearing loss (SSNHL) is unknown. However, studies reported a variety of causes (e.g., infectious, autoimmune, neoplastic, neurologic, otologic, metabolic, or vascular diseases; ototoxic drugs; trauma) [2]. Ototoxic drugs possess the ability to disrupt hearing and balance functions by affecting the cochlear and vestibular systems. Among the well-known culprits in this category are aminoglycoside antibiotics, platinum-based antineoplastic agents (like cisplatin and carboplatin), and specific loop diuretics such as furosemide, salicylic acid, and quinine [3, 4]. This adverse effect aligns with reports from the Japanese Adverse Drug Event Report (JADER) database, where significant signals were also detected for drugs used in erectile dysfunction, including sildenafil [5]. On the other hand, phosphodiesterase type 5 (PDE5) inhibitors – such as sildenafil, vardenafil, and tadalafil – are used therapeutically, predominantly to treat erectile dysfunction, with sildenafil also serving as a treatment option for pulmonary hypertension. According to Maddox et al, PDE5 inhibitors have become widely prescribed since their introduction in 1998, with an estimated 40 million new prescriptions globally [6]. Sildenafil

in particular surpassed common drugs like Tylenol-codeine and albuterol in prescription numbers, and around 1 in 5 men over 40 in the United States have tried sildenafil alone. Maddox also mentions that considering the large user base of PDE5 inhibitors, it is certainly possible that observed SSNHL cases reflect the general incidence rate. Commonly associated side effects of sildenafil include headache, facial flushing, nasal congestion, dyspepsia, and nausea. There are, however, additional concerns regarding PDE5 inhibitors, with particular focus on the sense organs. There are increasing reports of visual side effects to sildenafil as well as hearing impairment, including sudden hearing loss [2, 3, 7–9]. Original reports completed in 2007 by Mukherjee et al. [3] highlighted the problem of SSNHL with sildenafil use, subsequently leading to changes in FDA policies. The aim of this study is to highlight the relationship between sildenafil, a commonly prescribed erectile dysfunction drug, and hearing impairment – SSNHL, and to highlight the necessary precautions and recommendations during use to avoid the risk of ototoxicity associated with its use.

CASE REPORT

Patient Information

A 29-year-old male presented to the Department and Clinic of Phoniatics and Audiology with tinnitus in the right ear and sudden hearing deterioration, which had developed 1 week prior to admission. He reported severe hearing loss in the right ear. Initial evaluation and treatment were performed at an emergency department in Spain, where he was prescribed Dexona 30 (dexamethasone 4 mg). A detailed medical history revealed that the patient had been taking high doses of sildenafil in the weeks preceding the onset of hearing loss. He also reported a severe headache and transient tinnitus episodes during the week before symptom onset. Although the exact cumulative dose of sildenafil is not known, the patient stated that he had taken 10–20 mg daily or every other day for approximately 3 weeks before developing hearing loss. In addition, he reported the use of dapoxetine 30 mg.

Medications

At the time of hospital presentation, the patient was taking Dexona tablets (dexamethasone): initially 4 mg orally every 12 hours for 4 days, followed by a taper to 1 mg twice daily at the time of admission. He was also taking vitamin C (1000 mg once daily). During the steroid treatment period prior to hospitalization, the patient continued to use sildenafil (10–20 mg as needed prior to sexual activity) and dapoxetine (60 mg prior to sexual activity).

Diagnostic Tests and Results

Comprehensive audiological and vestibular testing was performed. Some of the audiological tests are shown in Fig. 1.–3. Audiometry revealed that the left ear had a normal hearing threshold (Fig. 1.). The

right ear demonstrated residual hearing at 90 dB at 500 Hz and 100 dB at 1000 Hz–2000 Hz, with no response above these frequencies. Electrophysiological hearing tests (Brain Evoked Response Auditory [BERA]) indicated that the hearing threshold based on wave V was 100 dB HL in the right ear and 20 dB HL in the left ear (Fig. 2.). Acoustic otoemission (DP-OAE) tests showed no responses in the right ear, while the left ear had all responses recorded (8 x Pass) (Fig. 3.). Impedance audiometry revealed a type A tympanogram in both ears. No stapedius reflexes were recorded in the right ear for frequencies 0.5, 1, 2, and 4 kHz, while the left ear had stapedius reflexes recorded for 0.5, 1, and 2 kHz. Speech audiometry for the right ear showed no speech understanding at 110 dB. For the left ear, the speech detection threshold was 5 dB, and the speech discrimination score was 40 dB at 100%. Nystagmus was not observed. Vestibular function was assessed using the Hallpike caloric test – 30°C and 44°C water were used, postcaloric responses were normal, canal paresis (CP) was 2%, directional preponderance (DP) was 3%. Biochemical tests showed no abnormalities and were as follows: glucose – 93 mg/dl, HBA1c – 5.6%, cholesterol – 206 mg/dl, TGA – 69 mg/dl, LDL – 133 mg/dl, HDL – 56 mg/dl, K – 4.51 mmol/l, Na – 142 mmol/l, CRP – 0.8 mg/l, HGB – 14.6 g/dl, HCT – 43.6%, WBC – $9.79 \times 10^3/\text{ul}$, RBC – $4.57 \times 10^6/\text{ul}$, PLT – $307 \times 10^3/\text{ul}$. Blood pressure was normal, e.g., 119/73 mmHg, 116/69 mmHg, heart rate – 69–75/min. Neurological consultation also showed no pathology. Further diagnostic evaluation, including an angio-CT, was performed, with normal results.

Hospital Course

During his hospitalization in the Clinical Department of Phoniatrics and Audiology, the patient underwent comprehensive audiological and vestibular testing. He received standard treatment consisting of intravenous steroids (hydrocortisone [Corhydon] 100 mg twice daily), an osmotic diuretic (15% mannitol 100 mg twice daily), a proton-pump inhibitor (omeprazole 40 mg once daily, intravenous), and oral vitamin B supplementation (B1 100 mg, B6 200 mg, and B12 0.2 mg, 3 times daily). Despite this therapy, no significant improvement in hearing was observed.

Discharge Condition

The patient was discharged after 8 days in good general condition with recommendations for follow-up and further treatment. He was advised to continue treatment at the Hyperbaric Oxygen Center, and a referral for additional tests, including impedance audiometry, chest X-ray, and ECG, was issued. A follow-up visit at the Audiology Outpatient Clinic was scheduled for 2 weeks after completing treatment at the Hyperbaric Oxygen Center. A follow-up visit to the Audiology Clinic was scheduled in a fortnight's time i.e. after completion of treatment at the Hyperbaric Oxygen Centre. The patient was prescribed Metypred (32 mg for 3 days, 24 mg for 3 days, 16 mg for 3 days) and Controloc (20 mg, 1 tablet daily). The patient attended a follow-up appointment on the scheduled date, but there was no improvement in hearing in the right ear, the tinnitus subsided. Based on the clinical presentation and investigative findings, we arrived at a diagnosis of profound and unremitting SSNHL, likely attributable to the ototoxic effects of sildenafil. Notably, there was no evidence of concomitant vestibular

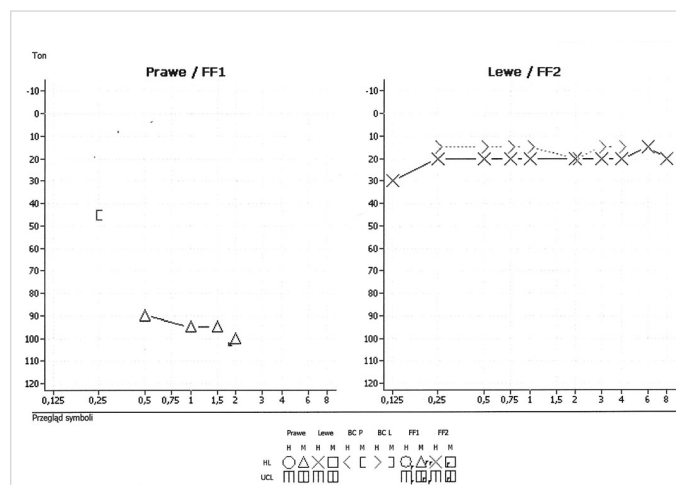


Fig. 1. Pure tone audiometry of the patient with sudden sensorineural hearing loss in the left ear.

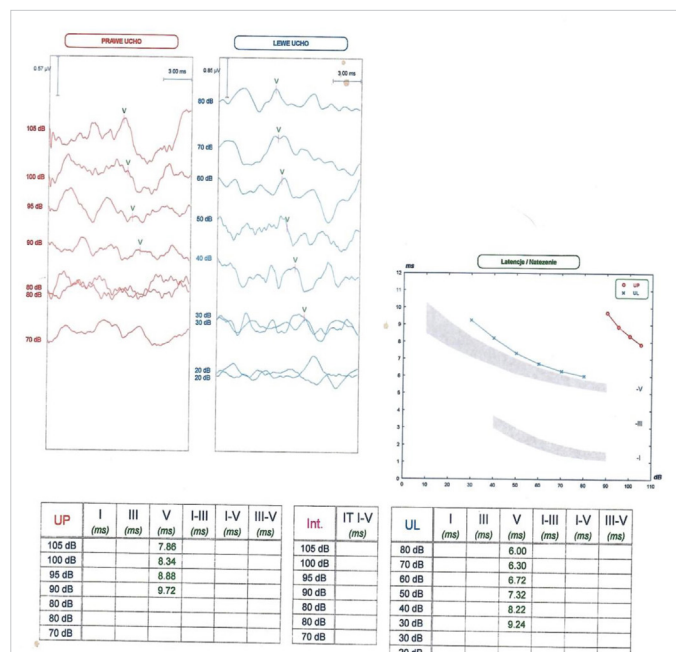


Fig. 2. Auditory brainstem evoked response to click stimuli – results of the case.

involvement, suggesting a cochleotoxic mechanism of action.

DISCUSSION

Recent studies have highlighted hearing impairment associated with the use of phosphodiesterase type 5 inhibitors (PDE5I). Zhang et al. concluded that hearing impairment related to PDE5 inhibitors is more prevalent among middle-aged and elderly men, with an average age exceeding 55 years. Generally, PDE5Is have shown an increase in reported cases since 2008, especially with concomitant medications such as anticoagulants, diuretics, proton pump inhibitors, NSAIDs, antihypertensives, and lipid-lowering drugs [10]. The reported doses of PDE5Is used in Zhang's et al. study fell within the recommended ranges for erectile dysfunction and pulmonary arterial hypertension. However, a prospective observational study highlighted that higher doses of tadalafil,

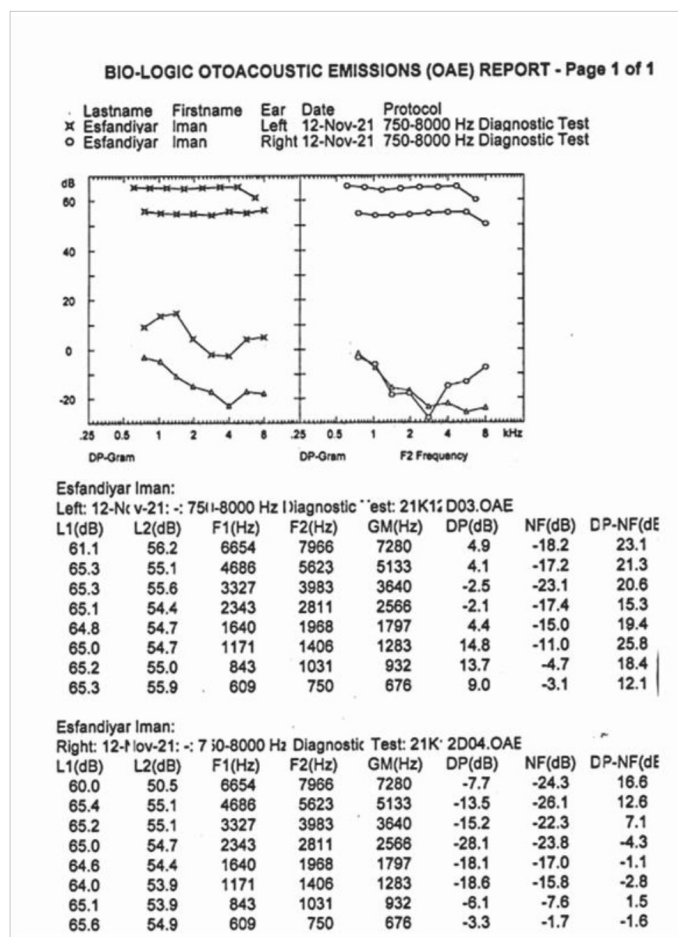


Fig. 3. DPOAE measurement from 1 to 8 kHz in the described patient.

exposure to ototoxic agents, renal failure, presence of infected congestion, hypoxia, and possibly old age [13].

In the case described here, there was certainly prolonged exposure to the drug. The drug was used daily for several days, but we cannot be completely sure of the entire dose taken due to the patient's uncertain medical history. It is difficult to assess the magnitude of the impact of previous fatigue, excessive exertion at work (night work, shift work), but it cannot be ruled out as a risk factor that this all weakened the patient's defence mechanisms.

Sildenafil Mechanism

The primary medical application for Sildenafil is to enhance penile erection. During sexual arousal, nerve impulses trigger neurotransmitter release in the corpora cavernosa. This then stimulates endothelial cells to produce nitric oxide, promoting the production of cyclic guanosine monophosphate (cGMP). Sildenafil, also known as a PDE5 inhibitor, blocks the phosphodiesterase-5 (PDE5) enzyme in the smooth muscle cells of blood vessels [14]. This prevents the breakdown of cGMP, allowing it to accumulate within the smooth muscle cells. Elevated cGMP activates protein kinase G, facilitating the relaxation of vascular smooth muscle. This cascade induces dilation of blood vessels, thereby enhancing blood flow. Within the penile arteries, there is a sustained erection [15]. Similarly, PDE5 inhibitors operate in the pulmonary arteries. The enzyme induces vasodilation, alleviating symptoms of pulmonary hypertension and preventing vascular remodeling. Studies show that sildenafil minimally reduces systemic and pulmonary artery pressure without serious cardiovascular effects and has minor impacts on carotid artery blood flow [14]. In heart failure patients, these inhibitors help prevent myocardial remodeling, and while most PDE5 inhibitors share similar mechanisms of action, they may exhibit selectivity for different PDE isozymes. Sildenafil, for example, affects PDE6 while tadalafil affects PDE11, leading to differing side effects [15]. PDE5 inhibitors reach a peak plasma level within an hour and have a half-life of 3–5 hours. PDE5 itself is found in various body tissues, including the brain; however, several studies found that there is no significant effect on cerebral blood flow or artery diameters. There are, however, increases in diameter, area, and flow volume of vertebral arteries [16]. In animal models, sildenafil also promoted angiogenesis in ischemic stroke, suggesting potential benefits for cerebrovascular diseases. This suggests that sildenafil could be beneficial in treating vertebrobasilar insufficiency [16].

Proposed Mechanisms of Sildenafil-Induced Hearing Loss

In more recent years, researchers devoted greater attention to the pathophysiology of PDE5-induced hearing loss. This increased awareness stems from evidence suggesting a link between the nitric oxide (NO)/cGMP pathway in cochlear pathophysiology [17, 18]. Blasitis' experiments focus on the disruptive impact of NO and cGMP analogs on cochlear structures, such as Deiters' cells, which coordinate sensory function within the cochlea [19]. Importantly, this mechanism is also seen in ototoxicity induced by antibiotics like gentamicin [18]. Thus, by enhancing the NO/cGMP pathway, PDE5 inhibitors may potentially induce ototoxicity through similar mechanisms. In the cochlea, the accumulation of intracellular cGMP

triggers the activation of various protein kinases and nuclear factors, (e.g., nuclear factors-kappa beta [NF- κ B], mitogen-activated protein [MAP] kinase), acting as secondary messengers. Particularly, the MAP kinases respond to extracellular stimuli and control gene expression, cell division, differentiation, and apoptosis. Ylikoski provides evidence that during cellular stress, MAP kinases activate within hair cells of the cochlea. When inhibited by the antibiotic medications mentioned above, cochlear hair cells are damaged [18, 20].

Manna et al. summarized clinical patient descriptions of PDE5Is cases. They noted that 75% reported unilateral hearing deficits, while 22% reported tinnitus. PDE5I-related hearing impairment has a poor prognosis. According to the data, only 32% of patients who reported PDE5Is-related hearing impairment documented hearing improvement from the initial visit [21]. No special and specific treatment is available for PDE5Is-related hearing impairment. As in other cases, systemic steroids are most often used [22]. However, Hyperbaric Oxygen Therapy (HBOT) can be treated as a part of a combination treatment for sudden hearing loss. It is proven that HBOT was significantly associated with improved hearing outcomes in patients with SSNHL [23]. Both treatment methods were used in the described case.

Other factors

Dapoxetine is not intended for continuous daily use. It should only be taken before a planned sexual intercourse. It should not be taken more than once every 24 hours. Dapoxetine is a novel selective serotonin reuptake inhibitor (SSRI) developed as an on-demand oral treatment for premature ejaculation [24]. Scientific results show that dapoxetine is associated with vasovagal (neurocardiogenic) syncope. No other associated significant cardiovascular adverse events were observed [24, 25]. No cases of deafness related to taking this drug have been reported in the literature. For this reason, the greatest contribution to hearing loss should be attributed to sildenafil.

CONCLUSIONS

This case report highlights the potential ototoxic effects of sildenafil, illustrating a profound instance of SSNHL potentially induced by

this commonly prescribed medication. The clinical presentation, involving severe and unremitting hearing loss without vestibular involvement, suggests a cochleotoxic mechanism, aligning with the established pharmacological effects of sildenafil on the NO/cGMP pathway within cochlear structures. Otologic side effects of drugs are more common than people imagine. There are more drugs that cause adverse effects on hearing than you might think. Aminoglycoside antibiotics, for example, are increasingly used in medicine. Meanwhile, the number of drugs from other groups with potentially negative effects on hearing is growing – including the commonly used ones, such as antihypertensives/antiarrhythmics and psychotropics [26].

The widespread use of PDE5 inhibitors such as sildenafil necessitates heightened awareness among clinicians regarding their possible adverse effects on auditory function. While the drug's efficacy in treating erectile dysfunction and pulmonary hypertension is well-documented, this report focuses on the importance of monitoring patients for auditory symptoms, particularly those at risk for SSNHL. The effect and severity of ototoxicity can vary immensely depending on pharmacological and individual patient risk factors. The lack of significant vestibular involvement and the specific nature of the hearing loss emphasize the need for further investigation into the exact mechanisms by which sildenafil may cause cochlear damage.

The sheer number of prescriptions dispensed each month in the world (e.g., in the U.S., about 20 million) means that a significant number of people could be impacted [27]. Given the large number of individuals using sildenafil globally, further research is imperative to fully explain its impact on auditory health and to develop comprehensive guidelines to ensure safer usage. Findings of the cohort study of Etminan et al. suggest that regular users of PDE5Is might have an increased risk for eye problems such as serous retinal detachment (SRD), retinal vascular occlusion (RVO), and ischemic optic neuropathy (ION). There have been an increasing number of reports, including this case report, of adverse effects of the drug on hearing loss. So, regular users of PDE5Is need to be cognizant of different adverse events associated with these drugs and alert their physicians if they experience any visual deficits. Future studies should focus on exploring the dose-dependent effects of sildenafil on hearing, potential interactions with other medications, and identifying patient populations most susceptible to its ototoxic effects.

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